Diabetic retinopathy before and after cataract surgery

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Abstract

***Aims/background*—Increased retinopathy progression has been reported after cataract surgery in patients with diabetes mellitus. To assess the influence of cataract surgery on visual acuity and retinopathy progression, all diabetic patients who were subjected to cataract surgery during 1991–3 have been followed up at the Department of Ophthalmology in Helsingborg. The average follow up time was 2 years.**

***Methods*—One eye of each of 70 patients was included in the study, 35 monocularly and 35 binocularly operated on. Sixteen of the 70 patients had proliferative diabetic retinopathy (PDR) at baseline. The Wisconsin scale was used for the grading of retinopathy. The degree of glycaemic control was assessed by measurements of HbA_{1c}.**

***Results*—Most patients obtained improved visual acuity; a postoperative visual acuity of 0.5 or better was achieved in 89% of diabetic surgical eyes. Progression of the retinopathy occurred in 30 out of the 70 eyes, and was associated with mean level of HbA_{1c} (p=0.04), duration of diabetes (p=0.02), insulin treatment (p=0.001), and presence of retinopathy at baseline (p=0.01). Patients who progressed had a significantly higher incidence of macular oedema (p=0.006) than those who did not progress. No significant differences were found when operated and non-operated eyes were compared in the 35 patients with monocular surgery. Two patients in this group, however, ended up with macular oedema and worse vision in the operated eye than in the eye which was not operated on. Both patients had background retinopathy before surgery.**

***Conclusions*—Patients in this study, also those with PDR, obtained good visual acuity, better than in most previous studies. Poor glycaemic control was a factor of importance for the progression of diabetic retinopathy after cataract surgery.**

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Diabetic patients have an increased risk of developing cataract. This risk is modified by age, severity of retinopathy, and duration of the disease.[1]

Some studies have reported that cataract surgery causes progression of retinopathy with new haemorrhages, exudates, and macular oedema, and that progression is associated with poor visual prognosis.[2–9] It has also been claimed that neovascular glaucoma is common after cataract surgery[9–11]; more often after intracapsular than after extracapsular surgery.[11] In another study, however, no increase in progression of retinopathy was observed after cataract surgery.[12] While progression of retinopathy has been shown to be related to the degree of glycaemic control,[13 14] the association between such control and progression of retinopathy after cataract surgery has not been evaluated.

At the Department of Ophthalmology in Helsingborg (population of 145 000) in southern Sweden, we have offered regular photographic eye examinations to all diabetic patients in the catchment area since 1990. Of the known diabetic population <75 years old, approximately 70% are estimated to have been included.[15] Patients requiring cataract surgery are taken care of within our own department. Out of the 2232 diabetic patients examined with fundus photography between January 1991 and December 1993 cataract surgery was performed on 77 by four surgeons using similar surgical techniques.

The aim of the present follow up study was to examine the visual acuity 2 years after cataract surgery, and to relate any progression of retinopathy to the degree of glycaemic control, duration of diabetes, and mode of treatment.

Patients and methods

Seventy seven diabetic patients underwent cataract surgery between January 1991 and December 1993. Among them, 70 were followed up 18–32 months postoperatively. Four of the seven who were not followed up died; two patients were unable to come back for examination because of other diseases; another patient moved from the region and could not be traced.

Forty one patients were on insulin treatment; 29 were treated with oral antihyperglycaemic agents or diet alone. During the study period five of the latter changed to insulin treatment. Nineteen (27%) were type 1 diabetic patients, and 51 (73%) type 2 diabetic patients, as decided by the patients' diabetologists. Clinical characteristics are given in Table 1.

In 57 patients the first eye was operated on during the study period. Thirteen patients had previously undergone surgery in one eye. At follow up, a total of 35 patients had been operated on bilaterally, and 35 had been subjected to monocular surgery. We included the first

Table 1 Clinical characteristics of diabetic patients operated for cataract (mean (SD))

	Cataract surgery group
Number (male/female)	70 (29/41)
Age at cataract surgery (years)	70.1 (8.2)
Duration of diabetes at cataract surgery (years)	18.4 (14.9)
Treatment	
Insulin treatment (%)	59
Oral treatment (%)	30
Diet alone (%)	11
Type of diabetes	
Type 1 (%)	27
Type 2 (%)	73
Mean HbA_{1c} (%) (n=61)	7.6 (1.5)
Distribution of retinopathy before surgery	
Median (range)	21 (10–70)
No DR (level 10) (%)	43
Mild DR (levels 21–31) (%)	27
Moderate to severe NPDR (levels 41–51) (%)	7
PDR (60+) (%)	23
Macular oedema before surgery (%)	14
Macular treatment before surgery (%)	21

DR=diabetic retinopathy; NPDR=non-proliferative diabetic retinopathy; PDR=proliferative diabetic retinopathy.

operated eye in patients with bilateral surgery during the study period.

Forty eight of the 70 patients included in the present study were participants in the screening programme before surgery, the remaining 22 were enrolled when they were scheduled for cataract surgery. The stage of retinopathy and the prevalence of macular oedema were thus known before surgery from previous photographic examinations in most patients, and were confirmed at a preoperative biomicroscopic examination in all but six patients, who had dense cataracts. In these six patients we used the immediate postoperative examination as our best estimate of pre-surgery retinopathy level. Results from the biomicroscopic examinations were translated into the alternative classification of the Wisconsin study.

During the 2 year study, surgeons at the department changed technique from extracapsular surgery to surgery with small incision and phacoemulsification. Of the 70 patients, 20 were operated on with the latter technique and 46 with planned extracapsular extraction and posterior chamber implants. Capsular breaks and dialysis caused the implantation of anterior chamber lenses in four patients; vitrectomy was performed in one of these.

EXAMINATION PROCEDURE

A full medical and ocular history was taken before cataract surgery. This included questions on diabetes treatment, use of antihypertensive medication, and duration of diabetes.

A complete ocular examination was also performed initially; this included determination of best corrected visual acuity (Monoyer), tonometry, slit-lamp examination, and retinal biomicroscopy. At follow up the retinopathy level was determined by fundus photography.

FUNDUS PHOTOGRAPHY

Colour fundus photographs were taken at an angle of 50° with a Topcon TRC-50 VT fundus camera (Tokyo Optical Co Ltd, Japan). The photographs covered fields 1–3 of the 7 standard fields, with stereo pairs of the macula (field 2). When retinopathy had been detected, photographs included at least two additional fields.[15] The alternative classification of the Wisconsin Epidemiologic Study of Diabetic Retinopathy was used to classify the retinopathy level.[16]

Levels of retinopathy were defined as follows[16]: level 10, no diabetic retinopathy; level 21, very mild non-proliferative diabetic retinopathy (NPDR); level 31, mild NPDR; level 41, moderate NPDR; level 51, severe NPDR; level 60, fibrous proliferations only; level 61, panretinal laser treated proliferative diabetic retinopathy (PDR) (no evidence of levels 60 and 65); level 65, PDR without high risk characteristics; level 70, PDR with high risk characteristics.[17] All levels of PDR were also pooled into one group, 60+. Furthermore, retinopathy levels were divided into four groups and progression was evaluated; no DR (level 10), mild NPDR (levels 21–31), moderate-severe NPDR (levels 41–51), and PDR (60+).

Macular oedema was defined as the presence of hard exudates and/or retinal thickening within 1 disc diameter of the centre of the macula and was assessed as existent, non-existent, or questionable, and was in this study considered present, even questionably so.

The photographs were graded in a two step procedure. Intergrader reproducibility was 82%, and intragrader reproducibility varied between 84% and 88% about the same level.

The photographic screening examinations were performed on an annual or biennial basis. In this screening programme we also recorded age at diagnosis, mode of treatment, and use of antihypertensive drugs. Best corrected visual acuity was tested with Monoyer charts.

Table 2 Clinical characteristics of patients with and without retinopathy progression following cataract surgery (eyes with PDR at baseline excluded) (mean (SD))

	No progression	Progression	Mean difference (95% confidence interval)	p Value
Number (male/female)	24 (10/14)	30 (13/17)		
Age at cataract surgery (years)	72.3 (6.3)	70.9 (8.0)	1.4 (–2.5; 5.3)	0.49
Duration of diabetes (years)	9.6 (12.4)	17.9 (13.1)	8.4 (1.3; 15.4)	0.02
Type 1 diabetes (%)	4	23	19 (2; 36)	0.05
Insulin treatment (%)	21	67	46 (24; 68)	0.001
Mean HbA_{1c} (%)	6.9 (1.6) (n=21)	7.9 (1.4) (n=25)	1.0 (0.1; 1.8)	0.04
Antihypertensive treatment (%)	38	30	8 (–17; 33)	0.56
Time at follow up (months)	22.4 (7.7)	25.2 (4.6)	3.0 (–0.4; 6.4)	0.08

Table 3 Retinopathy before surgery and visual acuity before and after surgery in patients with and without retinopathy progression (eyes with PDR at baseline excluded)

	No progression	*Progression*	*p Value*
Number (male/female)	24 (10/14)	30 (13/17)	
Retinopathy at baseline (median and range)	10 (10–51)	21 (10–51)	0.01
Preoperative visual acuity (median and range)	0.1 (<0.1;0.4)	0.1 (<0.1;0.4)	0.56
Visual acuity at follow up (median and range)	0.9 (<0.1;1.0)	0.8 (<0.1;1.0)	0.19
≤0.1	1 (4%)*	4 (13%)	
0.2–0.4	1 (4%)		
0.5–0.7	8 (33%)	11 (37%)	
0.8–1.0	14 (58%)	15 (50%)	
Macular oedema preoperatively, n (%)	2 (8%)	6 (20%)	0.23
Macular oedema postoperatively, n (%)	0	13 (43%)	0.0002

Data are n (%), mean (SD); *Total percentages do not add up to 100 because of rounding up or down.

GLYCAEMIC CONTROL

Haemoglobin A_{1c} (HbA_{1c}) was determined by ion exchange chromatography, Mono S-HPLC (non-diabetic range 3.5–5.5%).[18] For each patient we computed the mean of all measurements listed in their hospital records during the 3 years before the follow up examination. The median number of measurements per patient was 7 (range 1–22). There was no significant difference in the number of measurements between the patients whose retinopathy progressed and those who did not.

MAIN OUTCOME MEASURES

The influence of age at surgery, diabetes duration, type of diabetes, mode of treatment, and degree of glycaemic control on progression of retinopathy was assessed by a case-control approach within the cohort. To study if retinopathy progressed more in the eyes which had been operated on than in those which had not, 35 patients with monocular surgery were also examined separately to assess possible asymmetric retinopathy. Patients with PDR at baseline were excluded when progression was estimated.

STATISTICAL METHODS

The distribution of the different stages of DR and the distribution of visual acuities were compared in eyes with and without retinopathy progression using the Mann-Whitney U test. To compare age at surgery, duration of diabetes, and mean HbA_{1c} we used Student's t test; and for the frequencies of type of diabetes, insulin and antihypertensive treatment we used the χ^2 test.

Table 4 Macular oedema, retinopathy, and visual acuity at baseline and at follow up in patients with PDR at baseline

Number (male/female)	16 (6/10)
Age at cataract surgery (years)	64.8 (9.3)
Duration of diabetes at surgery (years)	32.6 (10.5)
Type 1 diabetes (%)	69
Panretinal laser treatment before surgery (%)	94
Preoperative retinopathy level (median and range)	61 (60–70)
Preoperative macular oedema (%)	13
Preoperative macular treatment (%)	63
Macular oedema at follow up (%)	19
Macular treatment at follow up (%)	75
Preoperative visual acuity (median and range)	0.15 (<0.1–0.7)
Visual acuity at follow up (median and range)	0.7 (<0.1–1.0)
≤0.1	2 (13%)
0.2–0.4	
0.5–0.7	8 (50%)
0.8–1.0	6 (37%)
Mean HbA_{1c} (%)	8.1 (1.4)

Data are n (%), mean (SD).

Results

At the baseline examination there were 30 eyes (43%) without retinopathy, 24 eyes (34%) with NPDR, and 16 eyes (23%) with PDR (Table 1). The degree of retinopathy was unchanged in 40 eyes (57%) at follow up, had progressed 1–2 levels in 27 eyes (39%), and 3 levels in three eyes (4%).

When progression of retinopathy was evaluated in cataract operated patients without PDR at baseline (n=54), we found that progression was related to a higher average level of HbA_{1c} (p=0.04), a longer duration of diabetes (p=0.02), a higher prevalence of insulin treatment (p=0.001), and more advanced retinopathy at baseline (p=0.01). The incidence of macular oedema during follow up was higher in patients with retinopathy progression than in those who had not progressed (p=0.006) (Tables 2 and 3). Different techniques of surgery or YAG laser treatment had no influence on the progression of retinopathy.

Patients with PDR at baseline (n=16) had on average >30 years' duration of diabetes and most of them were type 1 diabetic patients. All except one had previously had panretinal laser treatment and 10 had received focal laser treatment of the macula. The prevalence of macular oedema was low both at baseline and at follow up (Table 4).

Most patients obtained a significantly better visual acuity (VA) after surgery; 62 patients achieved a VA of 0.5 or better (median 0.8; range 0.5–1.0) (p<0.001). The remaining eight patients had a VA of 0.2 or less; two of them with moderate to severe NPDR (levels 41 and 51) developed macular oedema with poor vision after surgery. Four of them had been unsuccessfully treated for macular oedema before surgery and had poor vision before the development of cataract. A hemithrombosis occurred 1 year postoperatively in one patient, and another had age-related macular degeneration (Table 5).

The degree of retinopathy in the 35 patients who received monocular surgery was similar in the two eyes at baseline. There were no significant differences with regard to retinopathy progression in the operated and non-operated eyes (Tables 6 and 7).

Discussion

In our material most of the operated patients achieved good visual acuity and significantly improved vision. The visual acuity after surgery was 0.5 or better in 89% of eyes.

Our results contrast with those of some other studies, which have shown an increased risk of progression of retinopathy and worsening of vision after cataract surgery.[2–9] Progression in these studies included an exudative response. Patients with background retinopathy before surgery were at greater risk.[2 3] Continuing neo-

Table 5 Clinical characteristics of patients with poor vision at follow up examination

Patient	Age at surgery	Sex	Preop VA	Postop VA	Comments	Operative complications	Preop ret level	Diab type	Treatment	YAG treatment
A	85	F	0.1	0.1	MO + ARMD	—	21	2	Insulin	No
B	81	F	0.4	<0.1	Hemithrombosis postoperatively	Fibrin exudation and postop raised IOP	10	2	Insulin	Yes
C	80	F	HM	0.1	MO and low vision many years preop	—	70	2	Insulin	No
D	77	F	CF	0.2	MO, treated preop but worsened postop, better VA in the non-operated eye	Capsular dialysis and anterior chamber lens	41	2	Oral	No
E	76	F	<0.1	0.1	MO worsened postop, better VA in the non-operated eye	—	51	2	Oral	No
F	71	F	P	HM	Venous thrombosis several years earlier	—	51	2	Oral insulin	No
G	66	F	CF	CF	MO and low vision many years preop	—	31	1	Insulin	No
H	66	F	CF	CF	MO and PDR preop, MO worsened postop	—	70	2	Insulin	No

ARMD = age-related macular degeneration; P = light perception; HM = hand movements; CF = counting fingers; MO = macular oedema; YAG laser treatment for capsular opacification.

Table 6 Distribution of retinopathy in operated eye at baseline and at follow up in 35 monocularly operated patients

	Follow up				
	No DR (level 10)	Mild NPDR (levels 21–31)	Moderate/severe NPDR (levels 41–51)	PDR (level 60+)	Total
Baseline					
No DR	12 (34)	8 (23)			20 (57)
Mild NPDR		3 (9)	2 (6)	1 (3)	6 (17)
Moderate/severe NPDR			3 (9)	1 (3)	4 (11)
PDR				5 (14)	5 (14)
Total	12 (34)	11 (31)	5 (14)	7 (20)	35 (100)*

DR=diabetic retinopathy; NPDR=non-proliferative diabetic retinopathy; PDR=proliferative diabetic retinopathy.
Data are n (%); * total percentages do not add up to 100 because of rounding up or down.

vascularisation was a threat to vision.[7] Old age predicted low postoperative visual acuity in one previous study.[9] The degree of glycaemic control, a known risk factor for retinopathy progression,[13 14] was not reported in these studies.

In the current study neither patients without retinopathy nor patients with treated PDR experienced severe retinopathy progression after cataract surgery. Patients with PDR had a very long diabetes duration and all except one had been treated with panretinal photocoagulation and had quiescent PDR. Most of the patients were focally treated for macular oedema. Despite previous macular and panretinal treatment, the majority (87%) obtained a postoperative visual acuity of 0.5 or better (Table 4). This is in contrast with the poor visual prognosis reported in patients with PDR in a previous study.[8] The postoperative visual acuity in that study was better, however, in those with quiescent than in those with active PDR with visual acuity 20/40 or better in 52% of patients in the former group. In our study, low visual acuity at follow up in patients with PDR was related to the preoperative retinal state, although in one patient (H) the macular oedema worsened (Table 5).

The rate of retinopathy progression was similar in the two eyes of patients undergoing monocular surgery (Tables 6 and 7). In two patients with unilateral surgery and background retinopathy, however, there was deterioration of the macular oedema and these patients (D and E) ended up with worse vision in the operated eye than in the eye which had not been operated on (Table 5).

Four of the eight patients with low postoperative visual acuity achieved some visual improvement, and none experienced severe postoperative complications (Table 5). Thus, cataract surgery may have given useful improvement of the visual acuity also in this group of patients. One patient in this group (H) had cataract surgery in order to improve the possibility of completing the panretinal treatment. Cataract surgery has been a recommended therapy when lenticular opacity prevents treatment.[19] A long history of macular oedema or venous thrombosis, such as in cases F and G, was associated with bad postoperative visual function, a fact that should be kept in mind if cataract surgery is considered (Table 5).

Table 7 Distribution of retinopathy in non-operated eye at baseline and at follow up in 35 monocularly operated patients

	Follow up				
	No DR (level 10)	Mild NPDR (levels 21–31)	Moderate/severe NPDR (levels 41–51)	PDR (level 60+)	Total
Baseline					
No DR	11 (31)	8 (23)	1 (3)		20 (57)
Mild NPDR		5 (14)			5 (14)
Moderate/severe NPDR			4 (11)	1 (3)	5 (14)
PDR				5 (14)	5 (14)
Total	11 (31)	13 (37)	5 (14)	6 (17)	35 (100)*

DR=diabetic retinopathy; NPDR=non-proliferative diabetic retinopathy; PDR=proliferative diabetic retinopathy.
Data are n (%); *total percentages do not add up to 100 because of rounding up or down.

In our study, progression of retinopathy following cataract surgery was significantly related to degree of glycaemic control as assessed by the average level of HbA_{1c} before surgery and during the follow up period. Progression was also related to presence of retinopathy at baseline, duration of diabetes, and insulin treatment. These risk factors for retinopathy progression are in accordance with those found in other studies.[14 16 20] The findings of our study should be interpreted cautiously because of the small number of patients and the short follow up time. It is plausible, however, that the glycaemic level and the presence of retinopathy at baseline also influenced the progression of retinopathy in this group of patients subjected to cataract surgery, but other pathogenic mechanisms may have been operational as well. It is well known that cystoid macular oedema (CMO) may occur postoperatively in cataract operated eyes. Inflammatory mediators may play a role, as they cause a breakdown of the blood–retinal barrier.[21] A higher postoperative frequency of CMO has been reported in eyes of diabetic patients, even in those without retinopathy.[22] Breakdown of the blood–retinal barrier is also an early sign of diabetes induced changes in the retina.[23] The factors contributing to the breakdown of the blood–retinal barrier and causing macular oedema after cataract surgery in non-diabetic eyes could possibly act synergistically with factors that cause breakdown of the blood–retinal barrier in background diabetic retinopathy. This might cause retinopathy to progress after cataract surgery.

Some studies have reported an increased incidence of neovascular glaucoma and continuing retinal neovascularisation following cataract surgery.[9–11] This was not observed in any of our patients.

It should be emphasised that the present study, like most similar studies, was based on a small number of patients. While our results are encouraging, a larger study with a longer follow up time could provide more conclusive data. Results from the preoperative examination might have been less reliable because of the obscuring cataract. In the two eyes of patients with monocular surgery, however, the retinopathy levels were very similar. Patients were randomly allocated to different examiners at follow up, and hence we have no reason to believe that there was a systematic error in the evaluation of the postoperative visual acuity or in the grading of photographs.

One might conclude that in the current study visual prognosis after cataract surgery was good. Those patients whose retinopathy progressed most had background retinopathy and poorer glycaemic control. It is possible that these factors in conjunction with the trauma of surgery in some patients caused the increased progression of retinopathy after cataract surgery.

Our screening programme covered most patients, many of them elderly. Thus, most patients with vision threatening retinopathy and a need for laser treatment, whether focal or panretinal, had received this at the time when cataract surgery was performed. This may be the reason why few diabetic patients in the present study had poor visual outcome after cataract surgery.

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