
EXTRACAPSULAR CATARACT EXTRACTION IN DIABETICS WITH RUBEOSIS IRIDIS

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SUMMARY

We reviewed thirteen operated eyes (twelve diabetic patients) with rubeosis iridis who underwent extracapsular cataract extraction and intraocular lens implantation. Prior to surgery five had active proliferative retinopathy (APR), and eight had non-proliferative retinopathy (NPR), either quiescent proliferative retinopathy (QPR) or background retinopathy (BR). No case with APR was visually improved by surgery. Three cases with NPR achieved a visual acuity of 6/12. After surgery, vitreous haemorrhage or progression of proliferative retinopathy occurred in three cases with APR. Early postoperative fibrinous uveitis was severe in eyes with APR, resulting in permanent fibrin membrane formation in four. We suggest a significant prognostic indicator in diabetic cataract extraction with rubeosis iridis is the status of the underlying retinopathy. With NPR, postoperative visual acuity may be good and early postoperative complications less severe. In the presence of APR the visual outcome is poor, progression of retinopathy likely and early postoperative fibrinous uveitis may be severe enough to prevent postoperative panretinal photocoagulation. Maximum preoperative panretinal ablation is essential in these cases.

The final visual acuity after extracapsular cataract extraction in diabetics with background retinopathy ($85\% \geq 6/12$) is worse than in diabetics without retinopathy and in non-diabetics ($90\% \geq 6/12$).^{1,2,3,4,5} The prognosis is worse still in the presence of proliferative retinopathy ($40\% \geq 6/12$)⁴ and particularly so if rubeosis iridis is present. Operative complications such as persistent hyphaema and postoperative complications such as fibrinous uveitis are more common with rubeosis iridis, which may be further worsened by the surgery itself.^{2,6} Neovascular glaucoma is a frequent and devastating complication^{2,6,7,8} and an intraocular lens is probably contraindicated.^{2,9,10} Surgery in a diabetic patient with rubeosis iridis is indicated to improve

vision, especially if the fellow eye has advanced diabetic eye disease, and panretinal photocoagulation should be performed after surgery to prevent worsening rubeosis iridis. If possible, it is imperative to perform panretinal photocoagulation prior to surgery.^{11,12} If lens opacities preclude this, preoperative panretinal cryotherapy is indicated,¹³ although this is not recommended if there is no view of the retina. Failing these measures, active proliferative retinopathy or rubeosis iridis should be treated with early postoperative panretinal photocoagulation.^{6,14} Immediate postoperative uveitis, cyclitic membrane formation, fibrin deposition on the implant optic or posterior capsule may prevent this and result in worsening retinopathy.^{15,16} We present the first report dealing specifically with cataract extraction in diabetics with pre-existing rubeosis iridis.

METHODS

We retrospectively reviewed all diabetics with rubeosis iridis who underwent extracapsular cataract extraction and lens implantation between June 1986 and December 1990. Rubeosis iridis was defined as abnormal vessels coursing irregularly over the anterior surface of the iris for at least two clock hours. Patient age, sex, type and duration of diabetes, diabetic medication and coexisting systemic diseases were noted. Patients were excluded if:

- (1) there was a history of an unrelated ocular condition, e.g. age related macular degeneration or myopia,
- (2) diabetic related neovascular glaucoma or vitreous haemorrhage was present,
- (3) a previous vitrectomy had been performed.

Diabetic retinopathy was classified as active proliferative retinopathy (APR), i.e. active neovascularisation likely to result in vitreous haemorrhage, or non-proliferative retinopathy (NPR) which was either quiescent proliferative retinopathy (QPR), i.e. regressed retinal neovascularisation with no new neovascularisation for six months, or background retinopathy (BR), i.e. no neovascularisation. Time prior to surgery at which retinopathy was last adequately visualised was recorded.

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Table I. Preoperative diabetic and ocular status of each patient. BR: background retinopathy, M: maculopathy, APR: active proliferative retinopathy, QPR: quiescent proliferative retinopathy, IHD ischaemic heart disease. 1=focal laser therapy, 2= panretinal laser photocoagulation, 3=retinal cryotherapy.

	Age	Sex	Duration of diabetes (yrs)	Diabetic treatment	Pre-existing medic. disorders	Operated eye	Retinopathy right eye	Retinopathy left eye	Treatment right eye	Treatment left eye
1	63	F	10	Diet	Essential hypertension	R	M, APR	APR	0	2
2	76	M	6	Oral agent	-	L	BR, M	BR, M	1	1
3	71	M	30	Oral agent	Essential hypertension	R	QPR	APR	2	2
4	57	M	17	Insulin	-	R	APR	QPR	0	2
5	56	M	10	Oral agent	Essential hypertension	R	M, APR	QPR	2	2
6	58	M	30	Insulin	-	L	-	APR	2	2
7	73	F	2	Oral agent	-	L	QPR	QPR	2	2
8	71	M	3	Oral agent	-	R	QPR	M	2	2
9	76	F	15	Insulin	Essential hypertension IHD	R	BR, M	BR, M	1	1
10	73	F	12	Oral agent	-	R	QPR	QPR	2	2
11	73	M	28	Oral agent	Chronic obst. airways disease	R	M, QPR	M, QPR	2	2
12	73	M	27	Oral agent	as above	L	M, QPR	M, QPR	2	2
13	76	F	7	Oral agent	-	R	APR	QPR	2	2

Each patient underwent extracapsular cataract extraction with posterior chamber lens implantation, employing a limbal or corneal section. Posterior synechiae were divided where necessary, but iris sphincterotomies were avoided, and persistent hyphaema was treated by continuous anterior chamber irrigation. A circular anterior capsulotomy was fashioned with a 25 gauge needle. The nucleus was expressed using two point pressure at six and 12 o'clock. An intraocular lens was inserted and dialled under hydroxypropyl methylcellulose which was then removed. The section was closed with interrupted 10.0 monofilament nylon sutures. Peripheral iridectomy was not performed.

Patients were assessed on the first postoperative day and

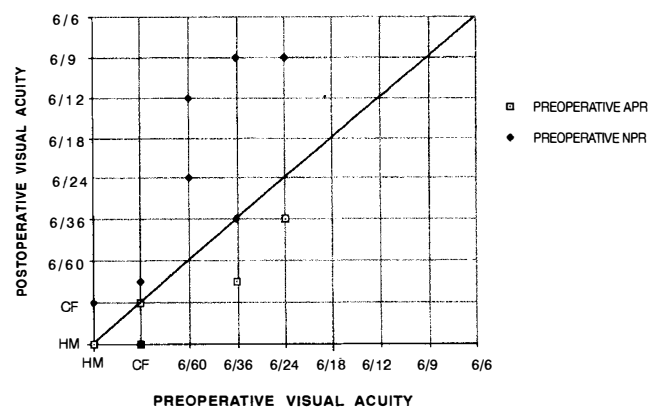


Fig. 1. Preoperative and postoperative visual acuity at one year.

panretinal photocoagulation performed if active proliferative retinopathy was present or previous panretinal photocoagulation was considered insufficient to control rubeosis iridis. Postoperative hypertensive uveitis and glaucoma were treated with topical agents and acetazolamide. No patients received oral steroid therapy for postoperative uveitis.

RESULTS

Thirteen operated eyes of twelve diabetics were identified, of whom seven were men and five were women. The average age was 63 years (range 56 to 76 years). Six patients were non-insulin dependent and six were insulin dependent diabetics. The average duration of diabetes was 15.2 years. Four patients were being treated for systemic hypertension, one had chronic obstructive airways disease, and one had ischaemic heart disease, (Table I).

Prior to surgery, best corrected visual acuity was 6/24 in two cases, 6/36 in four and $\leq 6/60$ in the remainder. In no patient was the visual acuity $> 6/24$ in the fellow eye and one patient had lost the fellow eye secondary to neovascular glaucoma. Classification of retinopathy was possible on average 7.6 months prior to surgery (range: two to 17 months). Five operated eyes had APR, six had QPR, and two had BR. At one year, postoperative visual acuity was $\geq 6/12$ in three cases with NPR, two further cases with NPR were $\geq 6/36$, but visual acuity was not improved in the three remaining cases and was not improved in any case with APR (Fig. 1).

After surgery, APR markedly deteriorated in two cases,

Table II. Postoperative complications in each case following extracapsular cataract extraction with lens implantation.

PATIENT NO.	PREOP. V.A.	PREOP. RETINOPATHY	POSTOP. V.A.	PROGRESSION RETINOPATHY	IMMEDIATE	PERSISTENT	WORSENING	NEOVASCULAR	VITREOUS
					FIBRINOUS UVEITIS	FIBRINOUS UVEITIS	RUBEOSIS IRIDIS	GLAUCOMA	HAEMORRHAGE
1	CF	APR, M	CF	1	1	1	0	0	0
2	6/36	BR, M	6/9	0	0	0	0	0	0
3	6/24	QPR	6/9	0	0	0	0	0	0
4	CF	APR	HM	1	1	1	1	0	0
5	6/36	APR, M	3/60	0	1	1	0	0	0
6	6/24	APR	6/36	0	1	1	0	0	0
7	CF	QPR	HM	1	1	1	1	1	0
8	6/60	QPR	6/12	0	1	0	1	1	0
9	CF	BR, M	3/60	0	0	0	1	1	0
10	HM	QPR	CF	0	0	0	1	1	0
11	6/60	QPR, M	6/24	0	1	0	0	0	0
11	6/36	QPR, M	6/36	0	1	0	0	0	0
12	HM	APR	HM	0	1	0	0	0	1

and developed in one case with QPR (Table II). Vitreous haemorrhage occurred in one case with APR. Five cases experienced worsening of rubeosis iridis, only one with APR. Four subsequently developed neovascular glaucoma one within three months and three after two years. Two cases were managed by Molteno tube implant surgery, one by repeated episodes of cycloablation with the Yag laser and one received no treatment. Nine operated eyes developed immediate postoperative fibrinous uveitis. This was severe in four cases with APR resulting in fibrin membrane formation on either the implant optic or the posterior capsule, despite intensive topical steroid therapy.

Postoperative panretinal photocoagulation was attempted in three of five cases with APR. In each case uptake by the retinal pigment epithelium was limited by intraocular inflammation and fibrin membrane formation, and worsening proliferative retinopathy was not prevented in any case. Vitreous haemorrhage and fibrin deposition prevented it being performed in two instances. Two cases with NPR received postoperative panretinal photocoagulation because preoperative photocoagulation was considered insufficient to cause regression of rubeosis iridis. Fibrinous uveitis prevented it being performed in one case in which APR had developed after surgery. It was not performed in the early postoperative period in the remaining cases.

DISCUSSION

Three patients out of eight with NPR achieved a final visual acuity $\geq 6/12$ at one year. In one of these cases, neovascular glaucoma developed a year later and this level of visual improvement was not sustained. Visual acuity in two further cases was $\geq 6/36$ after surgery but the remainder achieved navigational vision only. These results are in accordance with the previously held view that extracapsular cataract extraction in diabetics with rubeosis iridis has a limited prognosis for vision. Cases with Nrp are likely to do better than those with APR, and a few cases with NPR obtain good visual acuity, although whether this improvement is maintained longterm remains uncertain.

Active proliferative retinopathy worsened in two of the five cases and a further case was complicated by vitreous haemorrhage. Whilst it is known that cataract extraction in diabetics with APR is likely to worsen the condition, the operation may be performed so that adequate panretinal photocoagulation can be administered in the early postoperative period to control the situation. This was not possible in the cases reported here due to intensive postoperative fibrinous uveitis and fibrin membrane formation in four cases and vitreous haemorrhage in the fifth. This resulted in advancing proliferative retinopathy, no prospect of conventional panretinal photocoagulation and a poor prognosis.

Fibrinous uveitis occurred in five out of eight cases with NPR but was less severe than in APR eyes and was controlled by topical steroid therapy, in all but one case. The fibrinous uveitis was characterised by an intense anterior chamber flare but few cells, and presumably resulted from protein exudation from incompetent rubeotic vessels. It has been suggested that active rubeosis iridis which accompanies APR can be distinguished from inactive rubeosis,¹⁰ the greater blood flow and relative immaturity of active rubeotic vessels explaining the more severe fibrinous uveitis that accompanies APR compared to NPR. Worsening diabetic retinopathy did not occur in any fellow eye suggesting cataract extraction was responsible for the cases in which deterioration occurred. This has been reported previously.^{4,17} The exact pathogenesis remains unclear, although it has been suggested that surgery alters the concentration of angiogenic factor in the posterior segment to stimulate neovascularisation.¹⁸

After operation, rubeosis iridis worsened in five cases and neovascular glaucoma developed in four with preoperative QPR. Worsening of rubeosis iridis has been explained by forward diffusion of angiogenic factor through the posterior capsule or zonular fibres into the anterior segment. There is evidence that this occurs in animals where rubeosis has been induced by giant retinal tear formation in aphakic but not phakic rat eyes.¹⁹ In addition, in human diabetic eyes with proliferative retinopathy that undergo vitrectomy, rubeosis iridis is more common if

combined lensectomy is performed, suggesting that the lens acts as a barrier to diffusion of angiogenic factor or produces an angiogenic inhibitory factor.²⁰ The fact that rubeosis worsened after surgery in cases with QPR is surprising and suggests that mechanical manipulation of the iris may be sufficient to cause this in certain situations.

Postoperative panretinal photocoagulation proved difficult to administer in cases of APR due to fibrinous uveitis, fibrin membrane formation and vitreous haemorrhage. In no case did it control active proliferative retinopathy. Although these cases received intraocular implants it may be advantageous to leave them aphakic to avoid fibrin deposition on the intraocular implant and to increase the possibility of successful postoperative panretinal photocoagulation to prevent worsening retinopathy.

Although definite conclusions cannot be drawn from such a small series, we suggest:

(1) The overall visual prognosis in extracapsular cataract extraction in the presence of diabetic rubeosis iridis is poor and major complications are likely to occur. Two groups of diabetics patients with rubeosis iridis may exist, those with underlying active proliferative retinopathy prior to surgery and those with quiescent proliferative or background retinopathy.

(2) The visual outcome with quiescent proliferative or background retinopathy may be markedly improved, although in some cases the improvement may not be permanent due to the late development of neovascular glaucoma. Immediate postoperative fibrinous uveitis can usually be controlled with topical steroid therapy without fibrin membrane formation allowing postoperative panretinal photocoagulation to be performed if indicated.

(3) The visual acuity in cases with active proliferative retinopathy is rarely improved and progression of retinopathy is likely. Immediate postoperative fibrinous uveitis and fibrin membrane formation may be severe and may prevent postoperative panretinal photocoagulation. To proceed to cataract extraction with lens implantation in diabetics with rubeosis iridis and APR in order to perform postoperative panretinal photocoagulation may be unwise, and preoperative methods of panretinal ablation should be employed whenever possible or other methods of management considered.

Key words: Cataract extraction, diabetic cataract, iris, neovascular glaucoma, proliferative retinopathy, rubeosis iridis.

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