Pseudoexfoliation syndrome and secondary cataract

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Abstract

***Aim/background*—The pseudoexfoliation (PEX) syndrome is frequently associated with impairment of the blood-aqueous barrier. This study analysed if this might stimulate secondary cataract following cataract extraction.**

***Methods*—This historical cohort study included 197 eyes of 197 patients (99 with and 98 without PEX) that underwent extracapsular cataract extraction with posterior chamber lens implantation (PMMA optic) between 1985 and 1991. Secondary cataract was defined as opacification of the axial posterior capsule and decrease of visual acuity by two or more lines. Mean follow up was 23.8 months. For statistical analysis, the Kaplan–Meier method and multivariate Cox regression analysis were used.**

***Results*—Secondary cataract was observed within 24 months in 35% (SD 7%) of all eyes, and was significantly more frequent in eyes with PEX (45 (11)%) than in eyes without PEX (24 (9)%, p<0.03). Eyes with diabetes mellitus (n=32) showed a significantly lower frequency of secondary cataract (11 (11)%) than eyes without diabetes mellitus (39 (8)%, p<0.01). The influences of sex, open angle glaucoma, type of cataract, surgeon, positioning of IOL, and phacoemulsification versus nuclear expression on secondary cataract did not reach statistical significance.**

***Conclusion*—The higher frequency of secondary cataract could be considered as another potential complication of cataract surgery in eyes with PEX.**

(*Br J Ophthalmol* 1997;**81**:862–866)

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Accepted for publication
10 April 1997

Pseudoexfoliation (PEX) syndrome is diagnosed by slit-lamp detection of deposits of a fibrillogranular material on the anterior lens capsule and at the pupillary margin.[1 2] It is frequently associated with open angle glaucoma,[3] melanin dispersion,[4] poor pupillary dilatation,[5] and may present with a specific type of PEX keratopathy.[6] These features in combination with zonular instability[7 8] predispose eyes with PEX to complications during cataract extraction.[9–13]

A number of studies have presented evidence of dysfunction of the blood-aqueous barrier in PEX[14–18] with consecutive increase of aqueous flare[19] and aqueous protein concentration.[20] There is also an increasing number of reports on inflammatory reactions and fibrin formation in eyes with PEX following cataract extraction[21–24] or trabeculectomy.[25] Based on these observations, we hypothesised that increased and prolonged breakdown of the blood-aqueous barrier may also lead to a higher rate of secondary cataract in eyes with PEX. We performed a historical cohort study to analyse the frequency of secondary cataract in eyes with and without PEX following extracapsular cataract extraction and posterior chamber lens implantation.

Methods

From the clinical documentation data base of all patients who underwent extracapsular cataract extraction (ECCE) with implantation of a posterior chamber intraocular lens (PC-IOL) at our institution between 1985 and 1991, a sample of 99 patients with PEX and a sample of 98 patients without PEX was extracted. The control group of patients without PEX was matched for age, sex, and presence of diabetes mellitus (group matching). For those patients in whom PEX was manifest in both eyes and cataract extraction had been performed in both eyes, one eye was chosen at random. In the control group, only patients without PEX in either eye were included and one eye was chosen at random if both eyes had undergone cataract surgery. Exclusion criteria were traumatic cataract following penetrating ocular injury, history of uveitis, history of previous intraocular surgery, and intraoperative rupture of the posterior capsule and/or vitreous loss. Surgery was performed by a total of 10 surgeons, with three very experienced surgeons operating on 80% of all patients. Either nuclear expression or phacoemulsification were used. Plano convex non-heparin coated IOLs with 6.5 to 7.0 mm PMMA optics and propylene C-shaped haptics were implanted in 84% of eyes, and one piece biconvex all PMMA IOLs with 6.5 to 7.0 mm optics in the remainder of the eyes. All data were collected by reviewing the standardised patient charts and the standardised computerised surgical report. Factors analysed included presence of PEX, preoperative visual acuity, presence of open angle glaucoma, surgeon, nuclear expression or phacoemulsification, intended localisation of IOL (ciliary sulcus or capsular bag), intraoperative use of viscoelastics, intraoperative complications (rupture of posterior capsule, vitreous loss), postoperative visual acuity, development of secondary cataract, and Nd:YAG capsulotomy. The exact type of

Table 1 Population data of 197 patients

Group	No	Sex		Age (years) mean (SD) (range)	Follow up (months) mean (SD) (range) patients without secondary cataract	No with diabetes mellitus
		F	M			
All eyes	197	64	133	76.5 (6.6) (57–90)	27.0 (15.9) (3–84)	32
PEX	99	32	67	76.5 (6.7) (59–89)	28.4 (18.6) (3–84)	17
No PEX	98	32	66	76.5 (6.7) (57–90)	25.9 (13.5) (3–82)	15
Diabetes mellitus	32	7	25	77.4 (5.4) (66–88)	25.8 (17.1) (8–84)	—
No diabetes mellitus	165	57	108	76.3 (6.9) (57–90)	27.3 (15.6) (3–82)	—

PEX = pseudoexfoliation.

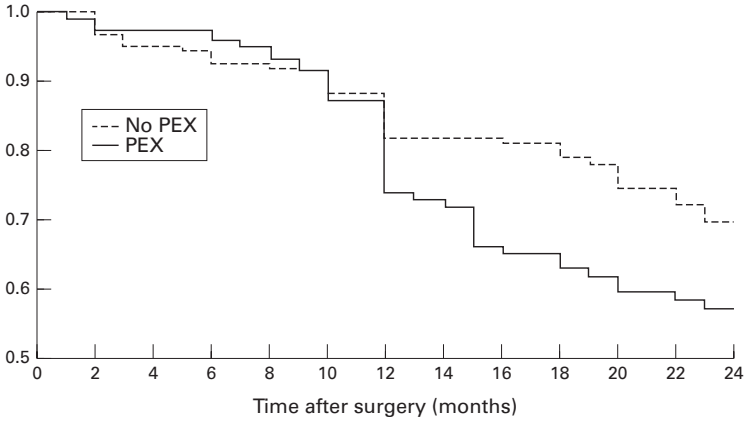

Figure 1 Kaplan–Meier survival curve of secondary cataract following extracapsular cataract extraction and posterior chamber lens implantation in eyes with or without pseudoexfoliation (PEX).

opening of the anterior lens capsule (can opener technique, letter box technique, or continuous circular capsulorhexis) was not recorded. Secondary cataract was defined as opacification of the axial posterior capsule visible at slit-lamp examination associated with a decrease of best corrected visual acuity by two or more lines. It was not differentiated between proliferative pearl-type and fibrotic secondary cataract. For patients who were not followed in our hospital, a standardised questionnaire was sent to their ophthalmologists to gain information on postoperative visual acuity and development of secondary cataract, using the above mentioned criteria.

Influence of patient age, sex, and diabetes mellitus was analysed within the PEX and non-PEX group.

For statistical analysis of secondary cataract, the Kaplan–Meier estimate for the probability of secondary cataract after 24 months was determined in the different patient groups. For all patients and separately for patients with and without PEX, additional probabilities for secondary cataract after 6, 12, and 18 months are presented. All other rates of secondary cataract are 24 month Kaplan–Meier estimates. For the main results 95% confidence intervals (rate plus or minus 2 SE) are given.

Significance testing was performed using the Cox regression model with inclusion of the matching criteria.

Table 2 Secondary cataract according to pseudoexfoliation (PEX) and diabetes mellitus (DM)

Group	No	Secondary cataract rate after 24 months* (SD)	p Value†
All eyes	197	35 (7)%	
Eyes with PEX	99	45 (11)%	
Eyes without PEX	98	24 (9)%	<0.03
Eyes with DM	32	11 (11)%	
Eyes without DM	165	39 (8)%	<0.01
Eyes with PEX and with DM	17	13%	
Eyes with PEX without DM	82	52%	
Eyes without PEX with DM	15	8%	
Eyes without PEX without DM	83	26%	

*Kaplan–Meier estimate.
†Cox regression analysis with diabetes mellitus, PEX, patient age (p>0.9) and sex. (p>0.3) included.

Results

Mean patient age at surgery was 76.5 (SD 6.6) years (range 57–90 years), and there were 64 males and 133 females. Mean follow up was 23.8 (15.4) months (range 3–84 months). Diabetes mellitus was present in 32 of the 197 patients. The demographic distribution was very similar in eyes with and without PEX (Table 1).

For all eyes the secondary cataract rate was 4% (SD 3%) after 6 months, 19 (6)% after 12 months, 26 (6)% after 18 months and 35 (7)% after 24 months.

For patients without PEX the rates were 6 (5)%, 14 (7)%, 16 (7)% and 24 (9)%, whereas 2 (3)%, 25 (9)%, 36 (10)% and 45 (11)% of the patients with PEX developed secondary cataract after 6, 12, 18, and 24 months ($p<0.03$, Cox regression analysis) (Table 2), with the Kaplan–Meier curve indicating that the frequency of secondary cataract started to differ between eyes with and without PEX at about 1 year after surgery (Fig 1). Eyes of patients with diabetes mellitus developed secondary cataract far less frequently (11 (11)%) than eyes of patients without diabetes mellitus (39 (8)%, $p<0.01$, Cox regression analysis) (Table 2), with the Kaplan–Meier curve indicating that the difference between eyes with and without diabetes mellitus started early in the postoperative period (Fig 2). Considering both PEX and diabetes, eyes with PEX without diabetes had the highest occurrence of secondary cataract (52%), followed by eyes without diabetes without PEX (26%), and by eyes with diabetes with PEX (13%) (Table 2). The lowest occurrence of secondary cataract was observed in the group of eyes with diabetes without PEX (8%).

The proportion of secondary cataract was higher for eyes with open angle glaucoma (33 (10)% versus 27 (12)%) in eyes without open angle glaucoma, nuclear cataract versus cortical cataract (41 (12)% in eyes with nuclear cataract versus 30 (9)% in eyes with cortical cataract), IOL placement into the ciliary sulcus (38 (12)%) versus placement into the capsular bag (33 (10)%), and phacoemulsification (46 (16)%) versus nuclear expression (31 (8)%).

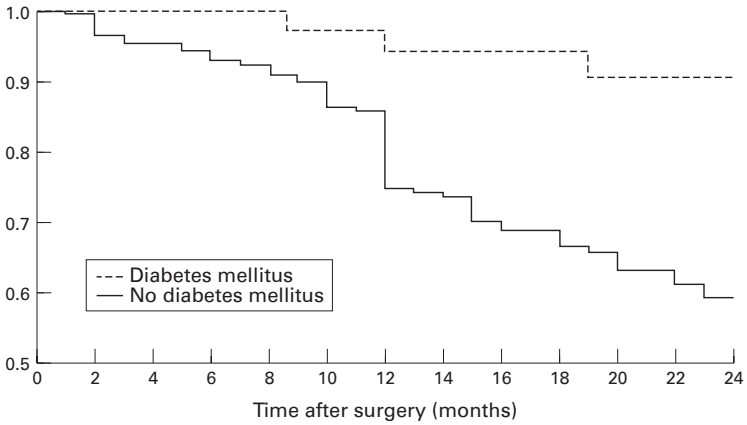

Figure 2 Kaplan–Meier survival curve of secondary cataract following extracapsular cataract extraction and posterior chamber lens implantation in eyes with or without diabetes mellitus.

Table 3 Secondary cataract according to ocular and surgical factors

Factor	*No of eyes*	*No of eyes with secondary cataract (SD)*	*p Value**
Nuclear cataract†	78	41 (12)%	>0.4
Cortical cataract†	89	30 (9)%	
Presence of glaucoma	81	33 (10)%	>0.2
Absence of glaucoma	116	27 (12)%	
Most experienced surgeon	104	38 (10)%	>0.3
Less experienced surgeons	93	31 (10)%	
Phacoemulsification†	49	46 (16)%	>0.1
Nuclear expression†	146	31 (8)%	
IOL implantation 'in the sulcus'†	73	38 (12)%	>0.2
IOL implantation 'in the bag'†	124	33 (10)%	
Intraoperative use of hyaluronic acid†	95	39 (11)%	>0.9
No intraoperative use of hyaluronic acid	75	27 (10)%	

*Univariate Cox regression analysis, similar results after inclusion of PEX, sex, diabetes mellitus, and age.
†Factors not present or not documented in all eyes.

Table 4 Secondary cataract according to age and pseudoexfoliation (PEX)

	All eyes		*PEX*		*No PEX*	
Age group	*No of eyes*	*Secondary cataract (SD)*	*No of eyes*	*Secondary cataract (SD)*	*No of eyes*	*Secondary cataract (SD)*
57–70	32	32%	16	55%	16	7%
71–80	102	36%	51	49%	51	22%
81–90	63	34%	32	34%	31	36%
All ages	197	35 (7)%	99	45 (11)%	98	24 (9)%

However, none of these differences reached statistical significance (Table 3).

The influence of patient age at surgery on development of secondary cataract seemed to be different for eyes with and without PEX. Whereas the frequence of secondary cataract decreased with increasing patient age in PEX (from 55% in patients younger than 71 years to 34% in patients older than 80 years), it apparently increased with increasing patient age in patients without PEX (from 7% in patients younger than 71 years to 36% in patients older than 80 years) (Table 4).

Discussion

Opacification of the posterior lens capsule after cataract surgery is a major medical and socioeconomic problem. Formation of secondary cataract may result from two mechanisms with many pathogenetic factors still poorly understood.[26–28] Migration of lens epithelial cells onto the posterior lens capsule leads to the proliferative variant with formation of Elschnig pearls, or metaplasia of lens epithelial cells into myofibroblasts is followed by fibrosis and contracture of the posterior capsule. The reported incidence of secondary cataract after extracapsular cataract surgery varies widely, ranging between 14% and 53% without PC-IOL implantation and between 7% and 50% with PC-IOL implantation.[29–33] It is difficult to compare the rates of secondary cataract formation in various studies because of high variations in follow up time, definition of secondary cataract, patient age, surgical techniques, and type of intraocular lenses.

Many factors have been reported to influence development of secondary cataract, most notably patient age,[34] type of cataract,[35] intraocular lens design including presence of laser ridges,[36–40] intraocular lens placement,[41] phacoemulsification,[42] and intraocular lens material.[43 44]

Clinical reports on inflammatory reactions and fibrin formation in eyes with PEX following cataract extraction[21–23] appear to be related to ultrastructural changes of the morphological correlates of the bood-aqueous barrier. Accordingly, electron microscopy and tracer studies have demonstrated alterations of the endothelial cells and basement membrane of the iris vessels[45–48] and the non-pigmented ciliary body epithelium.[47 48] In addition, non-invasive laser flaremetry has demonstrated postoperative breakdown of the blood-aqueous barrier, which has been shown to be significantly increased in eyes with PEX after cataract extraction with PC-IOL implantation,[24] following trabeculectomy,[25] and following argon laser trabeculoplasty.[49]

We were able to confirm our hypothesis that eyes with PEX have a higher frequency of secondary cataract. Several possible mechanisms may account for this observation. Firstly, intraoperative factors may be responsible for the higher rate of secondary cataract. Poor intraoperative pupillary dilatation, weak zonular support, posterior synechiae,[50] and corneal decompensation[6] may force the surgeon to minimise removal of equatorial lens cortex and lens epithelium and to reduce polishing of the posterior capsule, thus predisposing eyes to proliferation of lens epithelium and formation of secondary cataract. However, the observation that secondary cataract increased only 1 year after cataract surgery (Fig 1) in eyes with PEX indicates that intraoperative factors may be of lesser importance. Secondly, impairment of the blood-aqueous barrier and prolonged postoperative barrier breakdown in PEX may provide a protein rich 'culture medium' with plasma factors and growth factors,[51] thus supporting the proliferation of residual lens epithelium cells. However, the fact that eyes with diabetes mellitus showed a reduced number of secondary cataracts in our study rather speaks against a direct correlation between defects of the blood-aqueous barrier and formation of secondary cataract, as blood-ocular barrier breakdown is frequently seen in diabetic patients.[52 53] Thirdly, weakened zonular support may lead to focal areas of zonulolysis with increased capsular folds

Table 5 Pseudoexfoliation as a potential risk factor in cataract surgery

1	Zonular instability[8]
2	Poor pupillary dilatation[5]
3	Postoperative inflammation and fibrin formation[21–24]
4	Corneal endotheliopathy[6]
5	Melanin dispersion[4]
6	Increased rate of secondary cataract

and subsequent facilitated migration of lens epithelium or fibrotic changes. Finally, it has been demonstrated that intraocular hypoxia exerts a strong stimulatory effect on proliferation of intraocular cells.[54] As anterior chamber hypoxia has been demonstrated in eyes with PEX,[55] this may induce proliferation of lens epithelium and cause secondary cataract.

An unexpected and surprising finding of the present study was the reduced incidence of secondary cataract in eyes of patients with diabetes mellitus, as we had rather expected secondary cataract to be more frequent in diabetic eyes as a result of the impairment of the blood-ocular barriers. However, our data have to be cautiously interpreted with regard to diabetes mellitus and secondary cataract, as this study was not designed to evaluate the effect of diabetes mellitus on the frequency of secondary cataract; the number of eyes with diabetes was small (n=32); and we did not analyse the diabetic population in our study in detail with regard to stage of diabetic retinopathy and other factors. Therefore, we cannot exclude the possibility that our sampling scheme may have induced an overestimation of the role of diabetes mellitus. Knorz *et al* [56] and Tetz *et al* [57] observed similar findings of decreased incidence of secondary cataract in diabetics independently, whereas the contrary has also been reported.[58]

The influence of other factors—intended location of IOL, phacoemulsification, surgeon, and type of cataract—did not reach statistical significance in our study with regard to the occurrence of secondary cataract. However, these results must also be seen in light of the fact that this study was not designed specifically to evaluate the effect of these factors on secondary cataract.

PEX may be associated with potential serious ocular complications including rapidly progressive open angle glaucoma,[3] corneal decompensation secondary to PEX keratopathy,[6] spontaneous subluxation or luxation of the lens,[59] cilio-lenticular angle closure glaucoma,[60] and complications during cataract surgery. From the results of our study, it appears that secondary cataract should be added to the already long list of complications of cataract surgery in PEX (Table 5). Thus, in eyes with PEX, surgeons should probably try to take intraoperative measures to reduce remaining lens epithelium and minimise the chance of secondary cataract formation, and patients with PEX should probably be informed of their increased risk of developing secondary cataract.

Presented in part at the Annual Meeting of the American Academy of Ophthalmology, Atlanta, Georgia, 29 October to 2 November 1995.

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Br J Ophthalmol 1997 81: 862-866
doi: 10.1136/bjo.81.10.862