SCIENTIFIC REPORT

Cystoid macular oedema in paediatric aphakia and pseudophakia

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Aim: To assess the incidence of cystoid macular oedema (CMO), following lensectomy, anterior vitrectomy (limbal and pars plicata), and primary posterior capsulorhexis following surgery for congenital and developmental cataract.

Methods: A prospective study was carried out involving 30 eyes (20 children) with cataract. Lensectomy, primary posterior capsulorhexis, and anterior vitrectomy were performed on all eyes. Vitrectomy was performed using either a limbal or pars plicata approach. Fluorescein angiography was carried out 4–7 weeks after surgery. Fluorescein was administered intravenously and visualised using the Retcam.

Results: Anterior vitrectomy was carried out via the limbal approach in 12 eyes and, of these, six were implanted with an intraocular lens. The pars plicata approach was carried out in 18 eyes and 10 received an implant. CMO was not detected in any eye.

Conclusion: CMO was not detected in the early postoperative period irrespective of approach to anterior vitrectomy or presence of an intraocular lens. Intravenous fluorescein angiography was performed without complication

Cystoid macular oedema (CMO) is a recognised complication of anterior vitrectomy following cataract surgery in adults complicated by rupture of the posterior capsule.[1] In contrast, posterior capsulorhexis and anterior vitrectomy are performed routinely in children under the age of 6 years.[2] The aim of this study was primarily to investigate the incidence of CMO following lensectomy, primary posterior capsulorhexis, and anterior vitrectomy in congenital and developmental cataract surgery. The efficacy of intravenous fluorescein angiography with fundus visualisation using the Retcam was also investigated.

PATIENTS AND METHODS

A prospective study was carried out on children less than 6 years of age who underwent surgery at the Children's University Hospital, Temple Street, Dublin, over a 15 month period from January 2004 to March 2005 for congenital and developmental cataract. Cataract extraction involving lensectomy, primary posterior capsulorhexis, and anterior vitrectomy was carried out on all eyes. Vitrectomy was carried out using a choice of two approaches: (a) limbal and (b) pars plicata.

Surgical procedure

A combination of cyclopentolate 0.5% (1% if patient more than 1 year of age) and phenylephrine 2.5% were instilled in the eyes three times at 30 minute intervals before surgery. All surgery was carried out under general anaesthesia. A 2.8 mm clear corneal incision was performed at 12 o'clock and sodium hyaluronate (Healon GV) was injected into the anterior chamber. Anterior capsulorhexis was performed using the Oertli universal diathermy unit. Lens material was aspirated using the bimanual technique. The remaining surgery was carried out using a choice of two techniques.

(1) A posterior capsulorhexis 5.0 mm in diameter was made using the Oerlti system. Anterior vitrectomy was performed using a vitrector passed through the posterior capsule opening. The corneal wound was extended to 3.2 mm and the capsular bag was inflated with Healon GV. A foldable Acrysof lens was inserted and the corneal incision sutured using 10/0 Vicryl.

(2) In this technique, the implant was inserted in the capsular bag and the corneal wound was closed using 10/0 Vicryl. A scleral incision was made 2–2.5 mm posterior to the limbus. The vitrector was passed through the incision and a posterior capsulorhexis and anterior vitrectomy were performed. The scleral wound was closed using a 7/0 Vicryl suture.

All patients received intracameral dexamethasone (Decadron) 400 μg into the anterior chamber of the eye. A subconjunctival injection of dexamethasone (2 mg) and tobramycin (10 mg) were also given. All children less than 1 year of age whose eyes were implanted with an intraocular lens (IOL) were commenced on a course of systemic steroids postoperatively. The regimen involved intravenous methylprednisolone at a dose of 1 mg/kg body weight for 3 days followed by oral steroids for 1 week.

In the initial 72 hour postoperative period, all eyes received 2 hourly topical steroids, which were then gradually tapered over a 2 month period. In addition, cyclopentolate and chloramphenicol were both instilled in the eyes six times daily.

Fluorescein angiography

Ethics committee approval for the intravenous administration of sodium fluorescein to young infants was obtained form the National Maternity Hospital, Holles Street, Dublin. All patients underwent fluorescein angiography 4–7 weeks postoperatively at routine follow up examination under anaesthesia. Sodium fluorescein (10%) at a dose of 7.5 mg/kg body weight was administered intravenously via a cannula inserted in the forearm for the purposes of anaesthesia. Before angiography, colour fundal photographs were taken using a Retcam. Sodium fluorescein was then injected and, through use of a cobalt blue filter, the Retcam was used to visualise the various stages of the angiogram while observing evidence of CMO.

RESULTS

In all, 30 eyes of 20 children (12 male, eight female) were included in the study (table 1). Ten children had bilateral and 10 had unilateral cataracts and all surgery was conducted

Abbreviations: CMO, cystoid macular oedema; IOL, intraocular lens

Table 1 Patient details

Patient	Cataract	Eye	Age at surgery (months)	Approach to vitrectomy	IOL	FFA duration postop (weeks)
1	Congenital	Right	5	Pars plicata	N	4
	Congenital	Left	5	Pars plicata	N	5
2	Congenital	Right	0.75	Pars plicata	N	5
	Congenital	Left	1	Pars plicata	N	4
3	Congenital	Right	0.75	Pars plicata	N	5
	Congenital	Left	1	Pars plicata	N	4
4	Congenital	Right	5	Pars plicata	Y	4
5	Congenital	Right	7	Limbal	N	5
	Congenital	Left	7	Limbal	N	6
6	Congenital	Right	9	Pars plicata	Y	4
7	Congenital	Right	2	Pars plicata	N	5
	Congenital	Left	3	Limbal	N	4
8	Congenital	Left	2.5	Pars plicata	Y	6
9	Congenital	Right	1	Pars plicata	Y	5
10	Congenital	Left	1.75	Pars plicata	Y	4
11	Congenital	Right	1	Pars plicata	N	5
	Congenital	Left	1.25	Limbal	N	4
12	Congenital	Right	1	Limbal	Y	7
	Congenital	Left	1.25	Limbal	Y	7
13	Congenital	Right	1.75	Limbal	N	5
	Congenital	Left	2	Limbal	N	4
14	Congenital	Left	11	Limbal	Y	6
15	Developmental	Left	20	Pars plicata	Y	5
16	Developmental	Right	29	Limbal	Y	7
	Developmental	Left	30	Pars plicata	Y	5
17	Developmental	Right	77	Pars plicata	Y	7
18	Developmental	Right	58	Pars plicata	Y	5
	Developmental	Left	58	Pars plicata	Y	4
19	Developmental	Left	51	Limbal	Y	4
20	Developmental	Right	70	Limbal	Y	4

over a 14 month period from January 2004 to February 2005; 21 cataracts were congenital and eight were developmental. The mean age at time of surgery was 3.1 months in the group of children with congenital cataracts and 55 months in the developmental group.

Primary posterior capsulorhexis and anterior vitrectomy were carried out in all eyes, 12 via a limbal approach and 18 via the pars plicata. Of those eyes in which the limbal approach was used, four had an intraocular lens inserted; 10 eyes in which the pars plicata approach was used were implanted. In total, 14 eyes remained aphakic. All surgery was uncomplicated (table 2).

At postoperative fluorescein angiography, CMO was not detected in any eye.

DISCUSSION

In 1950 Hruby first described the development of macular oedema following cataract surgery.[3] Irvine later described the occurrence of CMO following cataract surgery complicated by the presence of vitreous in the anterior chamber.[3] Histological analysis has revealed that CMO arises from the build up of fluid in the outer plexiform and inner nuclear layers of the retina. It is thought to be the result of leakage from small capillaries in the macular region secondary to breakdown in the blood-retinal barrier.[4] Extensive research has been conducted on the role of prostaglandins in the development of CMO.[5] Surgical trauma to the iris, ciliary body, and lens epithelial cells result in prostaglandin synthesis in the aqueous humour. This leads to breakdown of the blood-aqueous barrier and in turn the accumulation of additional inflammatory mediators in the aqueous humour. Prostaglandins diffuse from the aqueous humour into the vitreous cavity where their action on the retinal capillaries leads to disruption of the blood-retinal barrier and fluid leakage into the surrounding retinal tissue and CMO.[5] The interaction between prostaglandins and other inflammatory mediators in the pathogenesis of CMO has not yet been fully elucidated, nor has their interaction with other causative factors. Cataract surgery complicated by capsule rupture and vitreous loss is now a well recognised risk factor for the development of CMO.[1 6] Disturbance of the vitreous resulting in traction at the vitreoretinal interface is thought to be responsible.

Not all CMO is clinically identifiable at slit lamp examination. Gass and Norton first described the fluorescein angiographic appearance of CMO[7] and identification using

Table 2 Number of eyes in which limbal or pars plicata approach was used, number of pseudophakic/aphakic eyes in each group, and incidence of cystoid macular oedema (CMO) on fluorescein angiography.

	Posterior capsulotomy/anterior vitrectomy	
	Limbal (no of eyes)	Pars plicata (no of eyes)
Total	12	18
Pseudophakic	6	10
Aphakic	6	8
CMO	0	0

this technique has resulted in a reported incidence of CMO as high as 70% following cataract surgery in adults. However, the incidence of clinically identifiable CMO is much lower and is estimated at 1–2% using current surgical techniques.[1] Angiographic determination of CMO therefore allows detection of very mild cases, whereas CMO detectable clinically is more advanced and usually associated with loss of visual acuity.

Posterior capsule opacification is a major factor complicating cataract surgery in infants and young children with a reported incidence approaching 100%.[8–10] In view of this, primary posterior capsulorhexis and anterior vitrectomy are recommended in children less than 6 years of age in order to maintain a clear visual axis.[2 11] CMO occurring in this age group is a potentially sight threatening complication owing to its capacity to induce amblyopia. However, the incidence of CMO in children is thought to be low. This has been attributed to a number of factors including superior structural integrity of blood vessel walls compared to that of adult vasculature, differences in prostaglandins, and operative techniques.[12] A small number of studies to date have investigated the incidence of CMO in children following cataract surgery. Early studies performed investigated the incidence of CMO in aphakic children. Varying results have been reported. Hoyt *et al*[13] reported CMO in 10 eyes of 27 infants and children following transpupillary lensectomy and anterior vitrectomy for developmental cataract. Gilbard *et al*[14] carried out fluorescein angiography and angioscopy on 25 eyes after pars plicata lensectomy and vitrectomy for congenital cataract. With the exception of one questionable result, no CMO was detected. Morgan *et al*[15] assessed 11 eyes after congenital cataract surgery using orally administered fluorescein. No CMO was detected. Pinchoff *et al*[16] found no evidence of CMO in 12 eyes after posterior capsulectomy (limbal approach) and anterior vitrectomy.

Ahmadieh *et al*[17] conducted a study involving 38 eyes of children with developmental and traumatic cataracts. The mean age at time of surgery was 6.3 (SD 2) years. All surgery involved lensectomy and anterior vitrectomy and primary intraocular lens implantation. Sodium fluorescein was injected intravenously and indirect ophthalmoscopy was performed. No CMO was detected. Similarly, Rao *et al*[18] found no evidence of CMO in 28 pseudophakic eyes following lensectomy and anterior vitrectomy using orally administered fluorescein.

In this study we investigated the incidence of CMO in children following primary posterior capsulorhexis and anterior vitrectomy carried out by one of two approaches, limbal and pars plicata. Both approaches are widely used by surgeons with the choice being determined largely by individual preference and experience. A number of eyes were implanted with an intraocular lens. Fluorescein angiography was carried out 4–7 weeks postoperatively to investigate for the presence of CMO as this is considered to be the peak time for its development.[18] CMO was not detected in any eye irrespective of approach used or presence (or absence) of an IOL.

The vast majority of studies to date involving angiographic detection of CMO have involved oral administration of fluorescein. In contrast, fluorescein was administered intravenously in this study. The safety and efficacy of intravenous fluorescein in young infants has been demonstrated in a previous study carried in our unit (O'Keefe *et al*, in press). Sodium fluorescein was injected intravenously to a series of premature babies with retinopathy of prematurity and angiography was performed using the Retcam. No adverse side effects were observed in these babies and we continue to do this without complication. Utilising the same technique to screen for CMO proved to be safe but also efficacious. It was quick and easy to perform and permitted careful examination while the child was under general anaesthetic. It allowed for greater flexibility in the timing of angiography compared with orally administered fluorescein, the latter requiring 45–60 minutes to elapse following ingestion in order to reach maximal intensity of dye leakage at the macula in the presence of CMO.

The Retcam proved useful in the assessment of CMO. In contrast with indirect ophthalmoscopy, the Retcam has the advantage of allowing simultaneous viewing by a number of assessors. It is easy to use and has image storage capability.

In conclusion, no evidence of CMO was detected in aphakic or pseudophakic eyes in the early postoperative period following lensectomy, primary posterior capsulotomy and anterior vitrectomy (limbal or pars plicata) for congenital and developmental cataract. Utilising the Retcam, intravenous fluorescein angiography was carried out without complication in this young age group.

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