

Outcomes of acute postoperative inflammation after cataract surgery

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PURPOSE. *To determine characteristics and final visual and surgical outcomes of patients who experienced early onset postoperative inflammation after cataract surgery and their early and late complications.*

METHODS. *This is a prospective case series of 126 patients out of 1500 cases who underwent cataract surgery and experienced early onset postoperative inflammation during the first 2 weeks after cataract surgery. All the patients received complete ocular examinations at onset of signs and symptoms of inflammation. A total of 110 patients with follow-up examinations between 3 and 30 months after recovery of early onset postoperative inflammation (mean follow-up 11.6 months) were enrolled in the next part of the study to evaluate the final visual and surgical outcomes. Background systemic and ocular diseases, type of surgery, type of intraocular lenses and viscoelastic agent, early and late complications, and final best-corrected visual acuity were studied.*

RESULTS. *Among 1500 patients, 126 cases (8.4%) had early onset postoperative inflammation, 64 cases (50.7%) were male, and 62 cases (49.3%) were female. Early complications were posterior synechia in 9 cases (7.1%), pupillary block in 2 cases (1.5%), and acute rise of intraocular pressure in 6 cases (4.7%). Late complications consisted of posterior capsular opacity in 38 cases (34.5%) and cystoid macular edema in 4 cases (3.2%). Peak of signs and symptoms of inflammation was during 2 weeks after surgery and all patients responded well to topical steroids. Final best-corrected visual acuity (BCVA) was 20/30 and better in 76 cases (69%), between 20/40 and 20/80 in 24 cases (22%), and less than 20/80 in 10 cases (9%). All cases with BCVA less than 20/80 had ocular diseases like choroidal neovascularization scar or age-related macular degeneration, advanced glaucoma, or corneal opacity.*

CONCLUSIONS. *Early onset postoperative inflammation causes pain, decreased vision, and patient anxiety in the acute phase but neither decreases final visual acuity nor has any significant or permanent ocular sequels. (Eur J Ophthalmol 2007; 17: 20-8)*

KEY WORDS. *Early onset postoperative inflammation, Cataract surgery*

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INTRODUCTION

Postoperative inflammation can result from various factors including exacerbation of underlying uveitis, retained intraocular foreign material after surgery, re-

tained lens material, intraocular lens (IOL) chafing, phacoanaphylactic endophthalmitis, sympathetic ophthalmia, and infection. Prolonged surgical time, toxic reactions to irrigating solutions, viscoelastic products, or polishing agents on IOLs may cause blood-

aqueous barrier breakdown and leakage, leading to more pronounced postoperative inflammation (1).

Noninfectious postoperative uveitis has been reported in up to 30% of patients following cataract surgery (2). It may cause significant anxiety for surgeon and patients and sometimes leads to increased intraocular pressure (IOP), corneal edema, endothelial injury, fibrin formation on IOL surface, posterior synechia (PS), posterior capsular opacity (PCO), cystoid macular edema (CME), and chronic anterior uveitis (2-4).

There is no information about the final outcomes of these serious inflammatory processes that are on the borderline with infection many times in terms of diagnosis.

The aim of this study is to find characteristics and final visual and surgical outcomes of patients who had experienced early onset postoperative inflammation following cataract surgery in Labbafi Medical Center (LMC) over a 3-year period (2000–2002).

To our knowledge, this is the largest series of patients with postoperative uveitis reported in the literature with a long follow-up period to evaluate late outcomes.

MATERIALS AND METHODS

This prospective case series was done by examining 126 of 1500 cases who underwent cataract surgery (phacoemulsification [PE] versus extracapsular cataract extraction [ECCE] + posterior chamber IOL [PC IOL]) and experienced early onset postoperative inflammation after cataract surgery in an academic hospital (LMC) after the project was approved by the institutional review board (IRB) of Ophthalmic Research Center.

Acute postoperative inflammation was defined as anterior chamber (AC) reaction equivalent to (or more than) 2+ cells and flare or fibrin formation in anterior chamber without vitreous reaction on slit lamp examination in a 0.2 mm² light spot with x40 magnification during the first 2 weeks after cataract extraction, with concomitant symptoms such as decreased visual acuity, pain, or photophobia.

Table I shows the grading system of AC reaction in our patients.

Exclusion criteria

Postoperative endophthalmitis had been clinically ruled out by the clinical status and vitreous tap and culture (in severe cases, i.e., grade 3–5 AC reaction).

Patients with grade 1 AC reaction were also excluded from the study. In order to stand on the safe margin and not to miss the infectious cases, patients with vitreous reaction, corneal haziness or relative afferent pupillary defect, recurrent and recalcitrant cases to steroids, or starting of symptoms 2 weeks after surgery were excluded from the study. We were not absolutely sure about the sterile nature of the inflammation in all of our patients, as we did not perform paracentesis of aqueous or vitreous for all of them due to high false positive rate of the former and relative risks of the latter, and self limiting and nonrecurring nature of the inflammation. However, we performed vitreous tap and culture in cases with severe postoperative inflammation (grade 3–5 AC reaction) to exclude the positive culture proven cases, if any.

Follow-up program

At the first step, 126 patients were completely examined by the authors in the same settings with adjusted and uniform criteria for diagnosis and management. Follow-up examinations were done on a regular basis exclusively by the authors in identical conditions and with the same instruments on days 1, 2, 3; weeks 1 and 2; and then at least 3 months after beginning of symptoms of acute inflammation in our clinic.

The treatment consisted of topical betamethasone 0.1% every 1 hour that was tapered according to the rate of subsiding of the inflammatory process judged by grade of AC reaction (Tab. I). Topical mydriatics were prescribed every 8 hours for cases with fibrin formation or posterior synechia formation. Systemic steroids (oral prednisolone 1 mg/kg/day) was also pre-

TABLE I - GRADING OF ANTERIOR CHAMBER REACTION

Grade	Cells in anterior chamber	Aqueous flare
0	0	No
1	1–5	Rare
2	6–15	Mild without plastic aqueous
3	16–30	Mild with plastic aqueous
4	>30	Severe with fibrin formation
5	Hypopyon	—

scribed for young patients (age <40 years) for 7 days.

Characteristics such as concomitant systemic diseases (diabetes mellitus [DM], systemic hypertension [HTN]) and ocular problems (pseudoexfoliation syndrome [PXF], glaucoma, history of uveitis), history of previous ocular surgery on fellow eye, technique of surgery, type of IOL, type of viscoelastic used during surgery, time of onset of symptoms after surgery, early complications such as pupillary block, and acute rise of IOP were recorded.

At the last examination, a thorough ocular examination including a dilated fundus examination was performed. Final best-corrected visual acuity (BCVA) and late complications such as persistent posterior synechia, captured PC IOL, clinical CME, sustained rise of IOP, and PCO were studied.

RESULTS

The main outcome measures of the current study are to find early and late complications and final BCVA of patients who had early onset postoperative inflammation after cataract surgery.

Among 1500 patients who were examined, 1374 cases (91.6%) with grade 1 AC reaction were excluded from the study (not considered as acute onset postoperative inflammation); 126 cases (8.4%) had acute onset postoperative inflammation after cataract surgery. Sixty-four patients were male and 62 patients were female. Mean age was 58±14.9 years (range 15–85 years). One hundred cases (79.3%) had cataract surgery

for the first time and 26 cases (26.6%) had history of previous cataract surgery in the fellow eye.

Diabetes mellitus was the most common background systemic disease (20 cases; 15.9%) and glaucoma and chronic uveitis were the most common background ophthalmic diseases (each of them 6 cases; 4.8%).

Technique of surgery was PE + PC IOL in 72 cases (57.2%) and ECCE+PC IOL in 54 cases (42.8%) (Tab. II).

In all patients methylcellulose was used as intraoperative viscoelastic and Coatel® (Chauvin, Opsia, France) was the most common brand mark used (86.5%) and the implants were polymethylmethacrylate (PPMA) IOLs with different brand marks (Agena, Saphir, PSM3, Centra, Sixflex-1, Azurite) from different companies.

Intraoperative complications

Intraoperative trauma and complications were sphincterotomy or sector iridectomy in 7 cases (5.5%), rupture of posterior capsule in 3 cases (2.3%), and vitreous loss in 2 cases (1.6%).

The mean postoperative time of onset of symptoms was 10.4 days with peak on the 12th day. Patients with history of cataract surgery in their fellow eyes revealed signs and symptoms of acute inflammation in the earlier phase of the postoperative course (Tab. III).

Anterior chamber reaction was 2+ cells (grade 2) in 67 cases (53%) (mild form) and 3+–4+ cells (grade 3–4) in 59 cases (47%) (severe form). Eight patients (6.3%) had macroscopic hypopyon (grade 5) and fib-

TABLE II - DISTRIBUTION OF BACKGROUND DISEASE IN 126 STUDIED PATIENTS

Background disease	Number	Percent
Systemic disease		
DM	20	15.9
HTN	13	10.3
Atopy	3	2.4
Ocular disease		
Glaucoma	6	4.8
Chronic uveitis	6	4.8
PXF	5	4

DM = Diabetes mellitus; HTN = Systemic hypertension; PXF = Pseudoexfoliation



Fig. 1 - Acute early onset postoperative inflammation after cataract surgery with hypopyon formation (postoperative day 7).



Fig. 2 - Same patient 1 week after topical steroid therapy.

rin formation was seen in 48 patients (38%) (grade 4). There was no statistically significant difference between mild and severe forms regarding clinical therapeutic response and later complications in the follow-up period ($p > 0.05$) (Figs. 1–3).

Although we performed vitreous tap and culture only for severe cases (grade 3–5), the cultures were negative in all patients.

Patients with DM had significantly more anterior chamber reaction and hypopyon formation (4 patients out of 20 cases with DM) ($p < 0.05$).

Sixteen cases missed follow-up after 1 month. All of these missed cases had mild forms of inflammation. Among 110 patients who had follow-up examinations for at least 3 months (range 3–30 months) after recovery from postsurgical uveitis, mean BCVA was 0.2 logMAR (20/30) that was gained during 20 ± 5 days after resolution of the inflammatory process.

Final BCVA was 20/30 or better in 76 cases (69%), 20/40 to 20/80 in 24 cases (22%), and less than 20/80



Fig. 3 - Fibrin formation in early postoperative period on intraocular lens.

in 10 cases (9%) (Fig. 4). All cases with poor visual acuity (less than 20/80) were due to background diseases such as advanced glaucoma, age-related macular degeneration, or corneal opacity (Tab. IV).

TABLE III - DISTRIBUTION OF THE PATIENTS REGARDING THE HISTORY OF CATARACT SURGERY IN THEIR FELLOW EYES AND ONSET OF SIGNS AND SYMPTOMS OF INFLAMMATION

Time of onset	Less than 10 days, n (%)	More than 10 days, n (%)	Total, n (%)
First eye	31 (31)	69 (69)	100 (100)
Second eye	16 (61.5)	10 (38.5)	26 (100)
Total	47 (37.6)	79 (62.4)	126

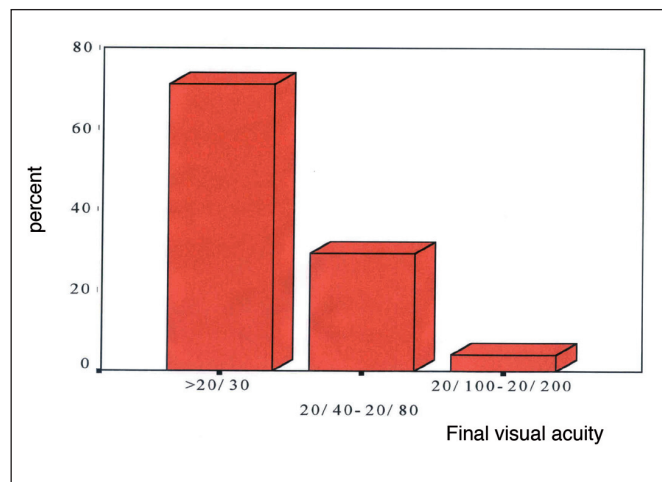


Fig. 4 - Final visual acuity at the last follow-up examination.

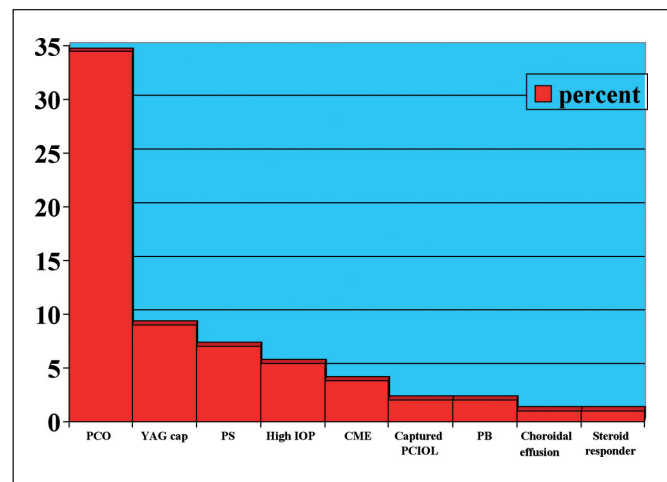


Fig. 5 - Incidence of postoperative complications after cataract surgery in our patients.

TABLE IV - DISTRIBUTION OF CAUSE OF BEST-CORRECTED VISUAL ACUITY LESS THAN 20/80 IN 10 EYES

Causes	Number	Percent
Macular scar of ARMD	3	30
Advanced glaucoma	4	40
Myopic macular degeneration	1	10
Corneal opacity	2	20
Total	10	100

ARMD = Age-related macular degeneration

Late complications examined at least 3 months after recovery of early onset postoperative inflammation were PCO in 38 cases (34.5%) (10 cases [9%] needed Nd:YAG capsulotomy), clinical CME in 4 cases (3.6%) (all improved with medical treatment), captured PC IOL in 2 cases, and rise of IOP secondary to prolonged steroid dependence in one case (Fig. 5).

There was no case of hyphema, choroidal hemorrhage, retinal detachment, or pseudophakic bullous keratopathy (Tab. V).

TABLE V - COMPARISON OF THE RATE OF COMPLICATIONS BETWEEN POWE SYNTHESIS OF THE LITERATURE (26) AND OUR STUDY

Type of complication	Present study, percent	Mean of complications (26) (range)	Number of studied eyes (26)	Number of studied reports (26)
PCO	34.5	19.7 (0.7-47.6)	14,677	41
Iris trauma	5.6	1.3 (0-9.1)	5147	8
Clinical CME	3.6	1.4 (0-7.6)	20,671	43
Posterior capsule rupture	2.4	3.1 (0-9.9)	19,052	38
Malpositioned IOL	1.8	1.1 (0-7.8)	17,944	40
Vitreous loss	1.6	0.8 (0-4)	14,622	26
≠ IOP (closed angle)	1.6	0.2 (0-1.6)	4391	11
≠ IOP (open angle)	5.6	1.2 (0-19.7)	11,376	34
Bullous keratopathy	0	0.3 (0-6)	15,971	27
Retinal detachment	0	0.7 (0-2)	33,603	42
Wound dehiscence	0	0.6 (0-3)	7499	17
Hyphema	0	0.5 (0-4)	7765	19
Vitreous hemorrhage	0	0.3 (0-8)	4386	5
Choroidal hemorrhage	0	0.3 (0-2)	3638	3

PCO = Posterior capsule opacification; CME = Cystoid macular edema; IOL = Intraocular lenses; IOP = Intraocular pressure

DISCUSSION

Noninfectious postoperative uveitis includes postoperative lens-induced uveitis (phacoanaphylactic uveitis and phacogenic uveitis), IOL-related inflammation, and sympathetic ophthalmia (1-7).

Phacoanaphylactic endophthalmitis is a rare autoimmune inflammatory response to lens protein resulting from disruption of tolerance (5-7).

Ocular inflammation may vary from mild to severe, with a large number of anterior chamber and vitreous cells. A hypopyon may be observed and granulomatous keratic precipitates and posterior synechia are generally present (1).

As none of our cases had keratic precipitates or vitreoretinal involvement, phacoantigenic uveitis may not be the causative diagnosis in our series.

Phacoantigenic uveitis is a nongranulomatous, nonspecific chronic inflammatory reaction to lens protein (8, 9) and it is believed to be a nongranulomatous variant of phacoanaphylactic endophthalmitis (10). Patients can present with inflammation weeks to months following surgery, and synechia or hypopyon may be present (10, 11). A history of retained lens fragments may be obtained but inflammation may also be seen with small amounts of retained cortex (1).

In contrast to patients with phacoanaphylactic endophthalmitis, keratic precipitates are usually absent and if they are present they are typically nongranulomatous (10) and histologic classic zonal inflammatory reaction noted in phacoanaphylactic endophthalmitis is absent. This entity responds well to removal of retained cortical materials and corticosteroid treatment but intraocular antibiotics are often given if infection cannot be initially ruled out (11, 12).

Perhaps a significant number of our patients are compatible with this diagnosis; however, we excluded suspected cases of infectious endophthalmitis who underwent vitreous tap and received intravitreal antibiotic from our study.

Inflammation secondary to IOL placement is much less common now that it was when IOL were first introduced (1). Early IOL polishing and sterilization techniques resulted in a toxic inflammatory response (1); however, it may still be seen even with new posterior chamber IOLs.

Liberation of iris pigment granules (especially with anterior chamber and sulcus placed IOLs, which caus-

es chafing of the iris) may simulate uveitis but true inflammation may also occur because of mechanical alteration of the blood aqueous barrier and liberation of mediators of inflammation (13-15).

We do not exactly know the postoperative site of IOLs (bag, sulcus, or bag-sulcus) in our patients but as in all patients rigid PMMA IOLs were used, it is possible that some amount of inflammation is due to IOLs.

Sympathetic ophthalmia is a rare cause of bilateral, granulomatous panuveitis, which is particularly devastating because of its potential to blind both eyes. It may occur following either traumatic or surgical manipulation of the eye but is more frequently encountered following ocular trauma (16-19).

None of our cases was compatible with this diagnosis because we had neither bilateral simultaneous ocular inflammation nor severe granulomatous pattern in our series.

In this study 126 cases experienced early onset postoperative inflammation, all of which clinically responded to steroids alone. However, we were not absolutely sure about the sterile nature of inflammation because we did not perform paracentesis of aqueous or vitreous for all our patients (only patients with grade 3-5 of AC reaction underwent vitreous tap and culture) due to high false positive of the former and relative risks of the latter and self limiting and nonrecurring nature of inflammation. Nevertheless, it is probable that bacterial endotoxins may have some role in inducing the inflammation. It is neither wise nor ethical to impose a high risk intervention (vitreous tap) for a patient with visual acuity of 20/20 and only 2+ AC reaction. Interestingly, the vitreous cultures were negative even in patients with severe forms of inflammation.

A total of 110 cases had later follow-up examinations after remission of early onset postoperative inflammation (range: 3-30 months, mean 11.7 months). The number of patients and follow-up period both are significant in comparison to other studies (2, 4). Mean age of patients was 58 ± 14 years and no significant difference was found between the number of male and female patients.

As background systemic and ocular diseases are associated with increased postoperative uveitis (1, 2, 20-23), these diseases were studied among our patients and there was only a statistically significant as-

sociation between diabetes and postoperative hypopyon formation ($p < 0.05$).

No significant difference was noted among our patients with different techniques of cataract surgery (PE versus conventional ECCE or lensectomy) ($p > 0.05$) as found in Martin et al's study (24), but Alio et al (25) found that PE + PC IOL causes less postoperative inflammation. Powe et al (26) reported no difference between these techniques. Kraff and Sanders (27) reported that PE causes less postoperative inflammation than ECCE.

However, Corbett et al (28) stated that there is no difference in postoperative inflammation between ECCE or PE with a 7 mm incision (small incision PE was not included). Oshika et al (29) reported that both aqueous flare and cell count measured with the laser flare cell meter were highest postoperatively in the 11 mm incision with less inflammation noted in decreasing order by the 7 and 4 mm incision groups.

In our study, hydroxypropyl methylcellulose was used intraoperatively for all patients as viscoelastic material.

Jaffe (30) has introduced cellulose and its derivatives as one cause for ocular inflammation; however, Holmberg and Philipson (31) found that Healon[®] does not significantly increase postoperative inflammation. Lanzl and Merte[®] (32) reported that silicone bubbles that are found in the end of the syringe of methylcellulose 2% cause endothelial injury and rise of IOP after surgery. Methylcellulose is a viscodispersive material (32) and it fragments to multiple fractions on aspiration at the conclusion of surgery and part of it may remain in the anterior chamber and cause severe inflammation and rise of IOP. We strongly recommend copious irrigation of this type of viscoelastic material after IOL implantation with at least 10 cc of balanced salt solution (BSS).

The IOL material was PMMA in all patients. PMMA and its electrical charge and air dust over it was found as a factor for postoperative inflammation (2). Shimuzi and Sakai (33) noted that ethylene oxide (used for sterilization of IOLs) should be less than 25 ppm for IOLs. Philipson and Fagelholm (34) reported that heparin modification of IOL surface decreases inflammation. Scharesberger et al (35) found that there was no difference among silicone, acrylic, and HEMA IOLs in causing inflammation. Hollick et al (36) found that postoperative inflammation is more with PMMA

IOLs than silicone and both cause more inflammation than acrylic IOLs. In the Samuelson et al (37) study giant cell formation over IOL surface was less in second generation of silicone IOL than acrylic IOLs. Acrylic IOLs caused less inflammation than first generation silicone IOLs. Abela et al showed that uveal and capsular biocompatibility are inversely related to the level of ocular inflammation and there were no significant differences in inflammation after implantation of hydrophilic acrylic, hydrophobic acrylic, or silicone IOLs in eyes with cataract and uveitis and blood aqueous barrier (BAB) recovery was similar between the uveitis and non-uveitic eyes (38, 39).

Most patients had early onset postoperative inflammation in the first 2 weeks after cataract surgery (mean 10.4 days, mode 12 days) and patients who had previous cataract surgery in their fellow eyes were affected earlier than patients whose first eyes were operated and had no history of ocular surgery ($p < 0.05$).

Mean BCVA after mean follow-up of 11.7 months was 20/40 or better in 80% of patients, 20/50–20/200 in 18%, and only 2% had BCVA less than 20/200. All patients with BCVA less than 20/80 had background ocular diseases such as age-related macular degeneration, advanced glaucoma, or corneal opacity.

In their literature review, Powe et al (26) noted that in 17,390 eyes, mean BCVA was 20/40 or better in eyes with no background disease and 89% in all patients with and without background disease who had history of cataract surgery. The final BCVA in overall patients in this study is not significantly better than in our patients with postoperative uveitis (Tab. V).

Four patients had clinical CME; all resolved with steroid therapy. Acute rise of IOP was seen in six cases, which was transient. One patient was a steroid responder with increased IOP that decreased with tapering of steroids.

Thirty-eight cases (34.5%) had some degree of PCO on slit lamp examination but only in 10 cases (9%) was the PCO clinically significant and needed Nd:YAG capsulotomy.

Shaumberg et al (40) showed that PCO was found in 25% of eyes after cataract surgery, throughout their life. Ursell et al (41) found that PCO was formed after 2 years follow-up in 44% of patients with PMMA, 34% in silicone and 12% in acrylic IOLs.

The rate of complications in patients with early onset postoperative inflammation was somewhat high-

er than overall cataract surgery results in the literature (26) but significant vision-threatening or permanent visual loss did not occur in our patients (Tab. V).

In conclusion, adequately treated acute postoperative inflammation after cataract surgery does not result in a significant visual loss in the long term.

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