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Research Article

A Clinical Profile of Cystoid Macular Edema following Cataract Surgery at Regional Eye Hospital

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ABSTRACT
Background: Despite advantages, significant complication that can lead to visual impairment is cystoid macular edema (CME). If steps are not taken properly to prevent, identify & treat cystoid macular edema, it can be considered as 2nd most common cause of preventable blindness next to cataract. The objective was to study the incidence, risk factors of CME following cataract surgery and to assess the visual prognosis to various treatment modalities.
Methods: Prospective, Observational study was conducted at Ophthalmology department at Regional Eye Hospital, Warangal for one year. All patients diagnosed as having CME after cataract surgery were included in this study. Socio demographic details, visual acuity, slit lamp examination, OCT and IOP were measured. 40 patients were divided in to 3 groups depending upon the visual improvement and 3 different treatment modalities were compared.
Results: This study included 40 patients with CME of which 38 patients (95%) were implanted with PCIOL and 2 patients (5%) with ACIOL. Out of 22 patients in group A, 14 patients showed resolution of CME. Out of 12 patients in group B, 9 patients showed resolution of CME.Out of 6 patients in group C, 4 patients showed resolution of CME. Postoperative examination revealed 18 patients (45%) had uveitis, 12 patients (30%) had vitritis, 2 patients (5%) had iris incarceration in wound. 2 patients (5%) had vitreous in anterior chamber.
Conclusions: Periocular steroids were found to be most effective form of treatment. Topical NSAIDS and topical steroids were also effective in treating Pseudophakic cystoid macular edema and intravitreal steroids can be given in resistant cases. But periocular and intravitreal steroids most commonly associated with raised IOP compared to topical steroids.

Keywords: Cataract Surgery, Cystoid Macular Edema, Treatment Modalities, Steroids, Nsaids.

INTRODUCTION

Cataract represents global public health challenge for all countries. It still remains as the major cause of preventable blindness. Cataract surgery is the most common surgery performed in the field of ophthalmology. Among different surgical techniques performed for cataract, extracapsular cataract extraction (ECCE) / small incision cataract (SICS) surgery / Phacoemulsification with posterior chamber intraocular lens implantation is the commonest one.[1]

Despite these advantages, significant complication that can lead to visual impairment is cystoid macular edema (CME). If steps are not taken properly to prevent, identify & treat cystoid macular edema, it can be considered as 2nd most common cause of preventable blindness next to cataract.[2]

Despite major advances in cataract surgery techniques and instrumentation, CME continues to be one of the leading causes of decreased visual acuity after cataract surgery. The prevalence of CME after cataract surgery depends upon the experience of the surgeon, type of surgery, design of study, definition of disease, method for diagnosis, patient population, prior ocular surgery, coexisting ocular diseases, accompanying systemic diseases, surgical complications and follow up criteria.[1,2]

Angiographic CME is very common and has been reported to occur in over 50% of patients after cataract surgery with or without IOL implantation. But reduced visual acuity due to clinical CME occurs in up to 8% of patients. Incidence of pseudophakic CME also depends upon the time lapse after surgery. It is highest at 44% in the first six weeks postoperatively, 24% at six months, and 12% at 12 months. It has been rarely reported after one year.[3]

The absolute risk factors of CME include Iris incarceration in wound[4], Iris IOL contact[5], Posterior capsular rupture [6], Postoperative uveitis [7], Phototoxicity and operative

microscope [8], Postoperative exposure to ultraviolet radiation [9]. The possible risk factors include age of the person [10], CME in the fellow eye [11], Diabetes mellitus [12], other Systemic diseases like hypertension, cardiac disease, vascular diathesis, rosacea, telangiectasia, chronic alcoholism etc.[12]

The preventive measures to be taken and treatment, which includes medical and surgical modes. Although it is impossible at present to completely avoid PCME in all cases, a few precautions may prove important in reducing incidence and severity of this important postoperative complication. They are Bag fixation of IOL, Use of PMMA IOL, Avoidance of primary posterior capsulotomy, minimize microscopic light exposure, Minimize operation time, Use of UV filters and UV IOL, Prophylactic steroids in high risk factors. Various models of treatment have been tried for CME. These include drugs like NSAIDS, both topical and systemic, corticosteroids, including oral, periocular and intravitreal [13,14], immunosuppressive, oral carbonic anhydrase inhibitors and vitrectomy, hyperbaric oxygen therapy and grid LASER photocoagulation. The challenge concerning the management of macular edema arises in chronic and persistent cases for which a stepwise approach is optimal.[15,16]

The complications of CME are development of permanent retinal damage due to prolonged edema, spontaneous rupture of the inner wall of a large central cystoid space to form a lamellar hole. Retinal pigment epithelium in the base of the hole is undisturbed. Cellophane maculopathy and macular pucker caused by epiretinal membrane may form either at the onset of CME or as a late complication of CME. When they occur, it is less likely that visual acuity will return to normal even after the resolution of CME. Additionally, these membranes may peel spontaneously from the surface of the retina and good visual acuity is restored and Prolonged CME may occasionally produce atrophy of the outer retinal layers. Macula may appear normal except for absence of foveal reflex.[15], With this background we aimed to study the incidence, risk factors of CME following cataract surgery and to assess the visual prognosis to various treatment modalities.

MATERIAL AND METHODS

The Prospective, Observational study had been conducted at Ophthalmology department at Regional Eye Hospital, Warangal between July 2021 to June 2022. All patients diagnosed as having CME after cataract surgery were included in this study. Diagnosis of CME was by clinically identifying cystoid macular edema in the macular area using slit lamp biomicroscopy with 78D lens. OCT was done in all cases at the time of diagnosis and at each follow up to correlate the course of CME before and after treatment. The study population include Patients undergoing Cataract extraction at REH, Warangal. All CME cases identified following Cataract surgery during study period were included in the study. Patients with any known case of preexisting CME secondary to causes other than cataract extraction, like uveitis with cystoid macular edema, diabetic macular edema., Prior intraocular surgery like trabeculectomy, retinal detachment and vitrectomy surgeries, drug-induced cystoid macular edema like latanoprost and patients with any coexisting anterior segment or fundus pathology were excluded.

After informed consent, patient's age, presenting complaints, duration of symptoms and mode of onset were taken. Review of medical records was done to identify preop, intraop and postop risk factors. Detailed history of diabetes mellitus and any other systemic illness was noted. Detailed ocular examination including best corrected visual acuity, anterior segment and posterior segment examination by slit lamp biomicroscopy to intraop and postop risk factors for CME.

a. **Visual Acuity:** BCVA was measured by subjective and objective refraction with Snellen chart.
b. **Slit Lamp Examination:** was carried out to look for any iris or vitreous incarceration at the wound site, Anterior chamber- cells and flare noted, Vitreous in anterior chamber, Posterior capsular rupture, PCIOL (in bag /sulcus), Cells in the anterior vitreous
c. **Fundus illumination by slit lamp biomicroscopy with 78D lens:** All patients diagnosed as having CME were examined with slit lamp biomicroscopy using a noncontact lens after dilatation of pupil. Using thin angled slit beam, macula appears thickened with translucent intraretinal cystoid spaces which were best appreciated using red free light. Detailed fundus examination to rule out other causes of CME was also done.
d. **OCT:** OCT was done in patients wherever needed, at the time of diagnosis and after treatment during subsequent follow-up.

Macular thickness was measured at the time of diagnosis and after resolution of CME.

e. **Intraocular Pressure**: IOP was recorded by Goldmann applanation tonometer in patients who received periocular and intravitreal steroids at the time of diagnosis of CME and during subsequent follow up.

Treatment Groups

Patients were divided into 3 groups depending upon the visual improvement as follows

GROUP A: Those patients whose visual acuity ranged from 6/6 partial – 6/18 were considered as group A. They were treated with topical steroids (prednisolone acetate 1% four times daily for 1 month) and topical NSAIDS (ketorolac tromethamine 0.5% four times daily for 1 month) & were followed up after 4 weeks. On follow-up, IOP was measured in all patients and resolution of CME was noted. Those patients in whom CME did not resolve were treated as group B.

GROUP B: Those patients whose visual acuity ranged from 6/24 – 6/36 were considered as group B and they were treated with periocular steroid injections.

Procedure of Posterior Subtenon Injection: 4% lignocaine eyedrops applied every 5 minutes for 3 times with cotton tipped applicator, superotemporal quadrant was anaesthetized. Upper eyelid was retracted and patient was instructed to look down and nasally. With tuberculin syringe 30mg of triamcinolone acetonide was given. These patients were followed up after 4 weeks and IOP was measured. Patients who had raised IOP were treated with topical antiglaucoma medications. Resolution of CME was noted and those in whom CME was not resolved, repeat injections were given.

GROUP C: Those patients whose visual acuity was </= 6/60 were considered as group C and treated with intravitreal steroids.

Procedure of Intravitreal Steroids: This procedure was performed under sterile conditions in OT using an operating microscope and topical anaesthesia. Lids and adnexa were cleaned with povidone iodine. Lid speculum applied and 4mg of injection triamcinolone acetonide was withdrawn in 1ml tuberculin syringe and was injected transconjunctivally at a distance of 3-3.5 mm from the limbus. Eye was examined immediately after injection for the presence of central retinal artery pulsations and those with impending obstruction underwent paracentesis. All eyes were reexamined at 15 minutes to half an hour after the injection to measure IOP and any immediate post injection complications. Patients were prescribed antibiotic eyedrops for at least 3 weeks.

Patients were followed up after 2 weeks and IOP was measured and those patients whose IOP was high were treated with topical antiglaucoma drugs. They were followed up after 4 weeks and resolution of CME was noted. All the data was entered in Microsoft excel 2019. The categorical variables were presented as frequency and percentages.

RESULTS

This study included 40 patients with CME following 860 cataract surgeries with IOL forming incidence of 4.65%. Out of 40 patients, 26 (65%) were men and 14 (35%) were women. (Table 1)

Table 1: Socio-Demographic Factors of Study Subjects

Variable		Frequency	Percentage
Age	<50 years	2	5
	51-60 years	18	45
	61-70 years	16	40
	>70 years	4	10
Gender	Male	26	65
	Female	14	35

Table 2: Surgery Related Factors, Systemic Illness and Risk Factors and BCVA of Study Subjects

Variable		Frequency	Percentage
Duration from cataract surgery	4-6 weeks	22	55
	7-10 weeks	10	25
	11-24 weeks	6	15
	>24 weeks	2	5

Type of IOL	PCIOL ACIOL	38 2	95 5
Systemic illness	DM HTN Both Nil	4 4 2 30	10 10 5 75
Risk factors	Iris incarceration in Wound Vitreous in AC PCR Uveitis Vitritis	2 2 6 18 12	5 5 15 45 30
Pre-treatment BCVA	6/6 Partial – 6/18 6/24 – 6/36 </= 6/60	22 12 6	55 30 15
FINAL BCVA	6/6 – 6/18 6/24 – 6/36 < 6/36	33 5 2	88.3 8.4 3.3

38 patients (95%) were implanted with PCIOL and 2 patients (5%) with ACIOL. 22 patients (55%) developed CME in 1st 4-6 weeks postoperative period and 10 patients (25%) in 6-10 weeks and 6 patients (15%) developed CME after 11-24 weeks and 2 patients (5%) developed CME after 24 weeks. All 40 patients with CME underwent SICS with IOL (100%).

Among systemic diseases, 4 patients had Diabetes mellitus, 4 patients had Hypertension, 2 patients had both diabetes and hypertension. Among 40 patients, none of the patients were treated with preop topical NSAIDS for preop uveitis. 4 patients were found to have CME in fellow eye. Among 40 patients, 6 patients (15%) had posterior capsular rupture associated with vitreous loss intraoperatively and had PCIOL implantation after anterior vitrectomy. Among 40 patients, 2 patients had ACIOL implantation due to > 180-degree zonular dialysis.

Postoperative examination revealed 18 patients (45%) had uveitis, 12 patients (30%) had vitritis, 2 patients (5%) had iris incarceration in wound. 2 patients (5%) had vitreous in anterior chamber. (Table 2)

Table 3: Treatment Modalities and Resolution Status of CME

Variable		Frequency	Percentage
Treatment modalities	Topical NSAIDS & Steroids Periocular steroids Intra vitreal steroids	22 12 6	55 30 15
Resolution of CME	Resolved Not Resolved	35 5	87.5 12.5
Increasing IOP on follow up	Yes No	7 33	17.5 82.5

Table 4: Topical Vs Periocular Vs Intravitreal Steroids for Treatment of Cme

Treatment given	Topical NSAIDS & Topical steroids (Group A)	Periocular Steroids (Group B)	Intravitreal Steroids (Group C)
Resolved	14 (64)	9 (75)	4 (67)
Not Resolved	8 (36)	3 (25)	2 (33)
Total	22	12	6

GROUP A: In this study 22 patients whose visual acuity range from 6/6partial-6/18 were considered as group A. Mean pretreatment visual acuity was 6/13. Mean pretreatment macular thickness was 340 microns. They were treated with topical steroids (prednisolone 1% eyedrops 4 times daily for 1 month) and topical NSAIDS (0.5% ketorolac eyedrops 4 times daily for 1 month) at initial diagnosis of CME. These patients were reviewed after 4 weeks and their IOP was measured by Goldmann applanation tonometer. Out of 22 patients, 14 patients showed resolution of CME. Their mean post treatment visual acuity was 6/9.2. Their mean

post treatment macular thickness was 250 microns.

In 8 patients, CME was not resolved with topical steroids and topical NSAIDS. They were treated with periocular steroids (triamcinolone acetonide 30mg by posterior subtenon route). These patients were reviewed after 4 weeks. CME was resolved in all 8 patients both clinically and by OCT wise. 6 patients showed visual acuity improvement by 1 to 2 lines and in 2 patients visual acuity remained the same. Macular degeneration is the cause for nonimprovement of visual acuity in these cases. In one patient, IOP was high level (26mmHg) and patient was treated with 0.5% timolol maleate eyedrops two times per day.

GROUP B: 12 patients whose visual acuity ranged from 6/24 – 6/36 were considered as group B. Mean pretreatment visual acuity was 6/30 and pretreatment macular thickness was 480 microns. They were treated with periocular steroids (posterior subtenon injection of triamcinolone acetonide 30mg). Before giving periocular steroids, IOP was measured.

These patients were reviewed after 4weeks. Out of 12 patients, 9 patients showed resolution of CME. Mean post treatment visual acuity was 6/26 and mean post treatment macular thickness was 300 microns. IOP was measured in the 1st follow up. In 3 patients, CME was not resolved and they were treated with repeat posterior subtenon injection. At the final follow up also, these 3 patients did not show improvement in visual acuity due to epiretinal membrane, macular hole and macular degeneration. They showed raised IOP in the range of 24-28mmHg and were treated with 0.5% timolol eyedrops two times per day.

GROUP C: 6 patients whose visual acuity was </= 6/60 were considered as group C. Their mean pretreatment visual acuity was 6/120 and pretreatment macular thickness was 780microns. Since they presented with significant CME, they were treated with intravitreal steroids (triamcinolone acetonide 4mg). Before giving injection, IOP was measured. They were reviewed after 2 weeks and complications like raised IOP noted and again reviewed after 4 weeks and resolution of CME was noted.

Out of 6 patients, 4 patients showed resolution of CME and in 2 patients CME was not resolved. Their mean post treatment visual acuity was 6/80 and mean post treatment macular thickness was 350microns. 2 patients visual acuity got worsened and the reason was due to chronic CME. 3 patients showed raised IOP in the range of 28-30mm Hg and were treated with timolol 0.5% eyedrops twice daily. (Table 3 and 4)

DISCUSSION

CME after cataract surgery is the most common cause of decreased vision in the postoperative period. This clinical condition was first recognized by IRVINE in the year 1953 and now this condition is known as IRVINE GASS SYNDROME. Most of the patients in this study developed CME in 4-6 weeks (55%). CME was most common in ECCE with IOL (45%) than SICS with IOL (35%) than PHACO with IOL (20%). The most important intraoperative risk factor includes posterior capsular rupture associated with vitreous loss which cause breakdown in blood aqueous barrier and chronic inflammatory reaction. In this study, 6 patients had posterior capsular rupture as intraoperative complication in which 3 eyes with CME was not resolved due to formation epiretinal membrane, macular whole and macular degeneration. In this study, major post operative risk factor includes uveitis (18%) and vitritis (12%). These factors indicate that inflammatory mediators like prostaglandins and leukotrienes play a key role to cause post operative CME. All patients were examined by slit lamp biomicroscopy with 78D lens and OCT was done in all patients at the time of initial diagnosis and subsequent follow up which showed resolution of CME after treatment. As OCT is a noninvasive device, which obtains high resolution images of the retina and detects the presence of retinal thickening and it causes minimal discomfort to the patient. Flanch Allan et al had done a double masked placebo controlled randomized study in 1987 which indicated that ketorolac 0.5% eyedrops in aphakic and pseudophakic patients with macular edema showed that statistically significant visual acuity improvement than those patients with placebo. [17] As per Jeffrey et al study, combination therapy of ketorolac and prednisolone eyedrops in acute CME, who had done a randomized double masked prospective trial of 28 patients who developed clinical CME after 21-90 days postoperatively. Patients experienced recovery of 2 or more lines of visual acuity after combination therapy. They concluded that combination therapy appears to offer more benefit than monotherapy. [18] More recently, it has been reported that treatment of acute, visually significant PCME with topical ketorolac and prednisolone combination therapy appears to offer benefits over

monotherapy with either drug alone as their synergistic effort results in rapid resolution of CME after treatment. In this study, 22 patients presented with CME between 4-6 weeks postop period whose visual acuity range from 6/6 partial- 6/18 was treated with administration of topical ketorolac tromethamine 0.5% eyedrops 4times per day for 1 month and topical steroids 1% eyedrops for 1 month. 14 patients showed resolution of CME at 1st follow up (i.e.) after 4 weeks. In remaining 8 patients CME was not resolved and they were treated with periocular steroids (i.e.) posterior subtenon injection of triamcinolone acetonide 30mg. CME was resolved in 8 cases and in 2 cases visual acuity did not improve due to macular degeneration.

2nd step in the treatment of postop CME is periocular steroids. According to McCartney et al [19], injection triamcinolone acetonide 40mg given as posterior subtenon injection is more likely to be effective due to closer location of drug to the macula. In this study, 12 patients who presented with CME between 4-6weeks postop period whose visual acuity range from 6/24-6/36 was treated with posterior subtenon injection triamcinolone 30mg. Out of 12 patients, 9 of them showed resolution of CME in 1st follow up (i.e.) after 4 weeks. In 3 patients, CME was not resolved and they were given repeat triamcinolone 30mg by posterior subtenon route. At the final follow up also, these 3 patients did not show improvement in visual acuity due to formation of epiretinal membrane, macular hole and macular degeneration. And they showed increased IOP in the range of 24-28mm Hg and were treated with timolol 0.5% eyedrops twice daily. 3rd step in treatment of CME is intravitreal triamcinolone acetonide. Conway mandi et al [20] had done randomized retrospective study about intravitreal triamcinolone acetonide for refractory chronic PCME. A study of 8 eyes of 8 patients with history of PCME recalcitrant to current standard treatment modalities. Patients received intravitreal injection of 1mg triamcinolone acetonide and were followed up for 8 months. Visual acuity was increased in all patients and there were temporary increase in IOP which were easily controlled with topical antiglaucoma drugs.

In this study, 6 patients whose visual acuity was <6/60 were treated with injection intravitreal steroids (i.e.) intravitreal triamcinolone acetonide 4mg. They were reviewed after 2 weeks and complication like raised IOP was noted in 3 patients. And they were reviewed after 4 weeks and resolution of CME was noted. Out of 6 patients, 4 of them showed resolution of CME and in 2 patients CME was not resolved due to chronic CME. 4th step in management of CME is pars plana vitrectomy. In our study, none of the patients underwent PPV. Walter KA et al [21] reviewed 24 consecutive cases who underwent PPV in 1 eye for PCME. All 24 patients failed to improve on medical therapy and had preop evidence of either vitreous adhesion to anterior segment structures or iris capture with IOL. 17 patients experience 3 or 4 lines of postop visual acuity improvement and all 24 patients had at least 1 line improvement. In this study, out of 40 patients 5 patients CME was not resolved even at the final follow up. Of which 3 patients who had posterior capsule rupture associated with vitreous loss as an intraoperative complication and they had uveitis postoperatively and they were treated with periocular steroids at the time of diagnosis of CME and they were reviewed after 4 weeks, since CME was not resolved, the injection was repeated. At the final follow up also, CME was not resolved due to formation of epiretinal membrane, macular hole and macular degeneration. 2 patients who had zonular dialysis as an intraoperative complication and they had uveitis and vitritis postoperatively and they were treated with intravitreal steroids. CME was not resolved due to chronic CME. Out of 40 patients, 7 patients showed raised IOP after 4 weeks of treatment. Out of 7 patients, 2 of them who received posterior subtenon injection once and 2 patients who received posterior subtenon injection twice showed raised IOP in the range of 24-28mm Hg and 3 patients who received intravitreal steroids showed raised IOP in the range of 28-30mm Hg. All these patients were treated with topical 0.5% timolol eyedrops twice daily and their IOP were under control.

CONCLUSION

In this study, CME was the most common vision threatening complication following cataract surgery who presented with defective vision in 1st 4-6 weeks postoperatively. Periocular steroids were found to be most effective form of treatment. Topical NSAIDS and topical steroids were also effective in treating Pseudophakic cystoid macular edema and intravitreal steroids can be given in resistant cases. But periocular and intravitreal steroids most commonly associated with raised IOP compared to topical steroids. Other modalities of treatment like pars plana vitrectomy and grid

laser photocoagulation should be considered in resistant cases.

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