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Evaluation Of Changes In The Morphology Of Macula After Phacoemulsification Using Spectral Domain Optical Coherence Tomography.

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Purpose: To study the incidence of cystoid macular edema (CME) and changes in macular morphology after uneventful phacoemulsification using spectral domain optical coherence tomography (SD- OCT)

Method:
In this prospective study, 150 patients who had undergone uneventful phacoemulsification were included. The visual acuity and foveal thickness were measured before and at 01 week, 4 weeks, and 12 weeks after surgery. The changes in foveal thickness and morphology were analyzed and correlated with the visual acuity.

Results:
86(57.33%) were males and 64(42.67%) were females, with mean age being 65.33±6.82 years. The average central macular thickness (CMT) measured preoperatively was 243.35± 19.508. Out of 150 patients, 5 (3.33%) patients were detected to have post operative CME. In patients who developed CME, the mean CMT was 406.20µ, 476.60µ, and 386.20µ at 1 week, 4 weeks, and 12 weeks respectively. In those patients who did not develop CME, the changes in CMT noted was 248.42µ, 250.05µ, 247.32 at 1, 4 and 12 weeks respectively. Maximum decline in visual acuity and maximum peak increase in CMT was noticed at 04 weeks postoperative period. In those patients who developed post operative macular edema, and improvement was noted by the end of 12 weeks. Whereas those who did not develop CME, there was a steady improvement in the vision throughout the period of observation and mild increase in macular thickness (±35µ) was noticed in the OCT reading at the end of 04 weeks which got normalized by the end of 12 weeks.

Conclusion: CME can develop even in patients who had undergone uncomplicated cataract surgery. SD OCT is a very reliable and non invasive method to detect these changes.

I. INTRODUCTION:

Cataract is the leading cause of blindness in the world and the most prevalent ocular disease[1]. With the advancement in surgical methods and instrumentation, the visual outcome following cataract surgery has become much better[2]. Phacoemulsification and implantation of a foldable intraocular lens (IOL) is currently the preferred technique of surgery among cataract surgeons[2]. The visual outcome of cataract surgery depends upon various factors like condition of the cornea, type of cataract, manipulation of iris, presence of preexisting ocular conditions like chronic uveitis, any associated systemic disease, and occurrence of intra operative complications and also experience of the surgeon.[3,4,5] Postoperative cystoid macular edema (CME) represents a well-known entity associated with a variety of intraocular operations and the commonest cause for suboptimal vision after cataract surgery[6,7]. CME is more common after operations such as intra capsular cataract extraction, scleral buckling, pneumatic retinopexy, combined penetrating keratoplasty and trans-scleral sutured posterior chamber IOL implantation.[8,9,10,11]Macular edema is also seen in patients with various retinal disorders such as retinal vein occlusion, uveitis or diabetes.

In 1953 Irvine et al reported occurrence of CME following cataract surgery and Gass and Norton in 1966 demonstrated it angiographically, hence it is known as the Irvine-Gass syndrome[6,7]. Cystoid macular edema is defined as an abnormal thickening of the macula associated with accumulation of fluid in the extracellular space of the outer plexiform layer (OPL) and inner nuclear layer (INL), and occasionally in the intracellular space[12].

Several diagnostic modalities provide important information about macular status and can assist ophthalmologists in diagnosing CME. The main techniques used in everyday practice include slit lamp

biomicroscopy, fundus fluorescein angiography (FFA) and fundus stereo photography. Optical Coherence Tomography (OCT) offers a non-invasive imaging technique that provides high resolution cross sectional images of the macula[13]. CME in OCT appears as a collection of hyporeflective spaces within the retina, with an overall increase in macular thickening and loss of the foveal depression. OCT is as effective as FFA at detecting macular edema, while it produces highly reproducible measurements so that serial examination may be used for follow up.

The Mean macular thickness measured by OCT in Asian population is 262.8±13.34 µm with males having a slightly thicker macula than the females[15], and the thickness differs from machine to machine. The chances of CME increase if large-incision technique is required, instead of small-incision phacoemulsification with implantation of a foldable intraocular lens (IOL). Incidence of CME increases if we have a complicated surgery with iris trauma, or capsule rupture with vitreous loss, or in patients with high risks (such as those with uveitis or diabetes). Intra operative release of inflammatory mediators, use of adrenergic drugs intra operatively, local action of inflammatory mediators on macula and vitreo-macular traction are the factors attributed to development of CME.[16] A macular thickness change that is equal or more than 50µm from baseline thickness has been described as an index of significant macular edema.[14] Ching and associates, reported the incidence of postoperative CME as 3.05% after phacoemulsification, where as it was 9.8% by Subramanian and associates.[7,9]

So this prospective, analytical study was planned to evaluate the incidence of CME after an uneventful phacoemulsification, in patients with no other prior predisposing factors. Further this study proposes to ascertain morphological changes in foveal thickness following uneventful phaco-emulsification.

II. MATERIAL AND METHODS:

One hundred and fifty patients with uncomplicated cataract, undergoing uncomplicated phacoemulsification were considered for this study. All patients with sufficient media clarity to permit preoperative OCT examination were eligible for inclusion in the study. Single eye from each patient (both sexes within the age group of 50-75yrs) were included. Patients on prostaglandin analogues, topical or systemic steroids or non steroidal anti- inflammatory drugs, history of uveitis, prior intraocular injections or surgery, past or pre-existing retinal and choroidal diseases that could affect retinal thickness were excluded from the study. Diabetic retinopathy or maculopathy, retinal vein occlusion, age-related macular degeneration, radiation retinopathy and previous laser treatment and subjects with abnormal macula based on clinical examination and preoperative macular OCT scans were also excluded from this study. A detailed informed consent was taken from every patient.

A comprehensive ocular examination included measurement of visual acuity by Snellen's chart, refraction, slit lamp biomicroscopy, and detailed fundus exam was performed to rule out any chorioretinal pathology. Macular thickness was measured using SD-OCT (Cirrus - Carl Zeiss Meditech, Inc, Jena, Germany). All of them had sufficient media clarity (Signal strength more than 6) to permit preoperative OCT for measuring the central macular thickness.

Phacoemulsification with posterior chamber intra ocular lens implantation was performed under topical anaesthesia. To minimize the surgeon oriented bias all the cases included in this study were done by the same surgeon. None of the patients were given topical non steroidal or steroidal eye drops pre operatively. Post operatively all patients were given Eye Drop Moxifloxacin 0.5% and Dexamethasone on tapering dose for 04 weeks. They were followed up at 1st postoperative day, 1st, 4th and 12th week post operatively. Visual acuity and OCT was repeated during each visit. The changes in foveal thickness and morphology were analyzed using SD-OCT (Cirrus - Carl Zeiss Meditech, Inc, Jena, Germany). Scanning was done using the macular cube scan (512 ×128 scan pattern) centered on the fovea. Scans with signal strength 6 or greater were considered for the study. The changes in foveal thickness and morphology were studied using optical coherence tomography (OCT). Eyes with post operative mean macular thickness >300µ or an increase of >50µ from preoperative baseline value, with or without cystoid changes, sub foveal nerosensory detachment (based on OCT) were considered as CME. The data was analyzed using statistical software (SPSS, version 19.0, SPSS Inc, Chicago, Illinois, USA).

III. RESULTS:

We evaluated a total of one hundred and fifty patients without any predisposing ocular or systemic illnesses, other than cataract. Out of 150 patients, 86 were males and 64 were females, with a mean age of 65.33 ±6.82 years (Fig 2). Preoperative visual acuity ranged from 3/60 to 6/9. They underwent uneventful phacoemulsification and in-the-bag intraocular lens implantation. Out of 150 patients, five patients developed CME during follow up. Three patients developed CME at the end of 1st week, whereas other two were detected to have CME at the end of 4 weeks. In four (4/5) of these patients CME persisted till 10 weeks, and normalized by 12 weeks. The mean central macular thickness changes noticed in the cases which developed CME was 246µ

preoperatively and 406µ, 475µ and 386µ at 1st, 4th and 12th week respectively(Fig 3). Mean central macular thickness in patients without CME was 242µ preoperatively, which was almost same as those who developed postoperative CME, while it was noted to be 248µ, 250µ and 247µ at 1st , 4th and 12th weeks during postoperatively follow up period. Among the five patients who developed CME, cystoid spaces were noted in 3 patients, 2 had diffuse spongiform edema(Fig 1). The incidence of CME in our study was 2%, 3.3% and 2.7% at 1 week, 4weeks and 12 weeks postoperatively respectively which was comparable with other studies. In those patients who developed post operative macular edema, there was a decline in visual acuity which was peak at 04 weeks and it was corresponding to maximum CMT which was also noticed at the same time postoperatively. Visual improvement was noted by the end of 12 weeks in all 5 patients(Fig 4). Whereas those who did not develop CME, there was a steady improvement in the vision throughout the period of observation and mild increase in macular thickness (±35µ) was noticed in the OCT reading.

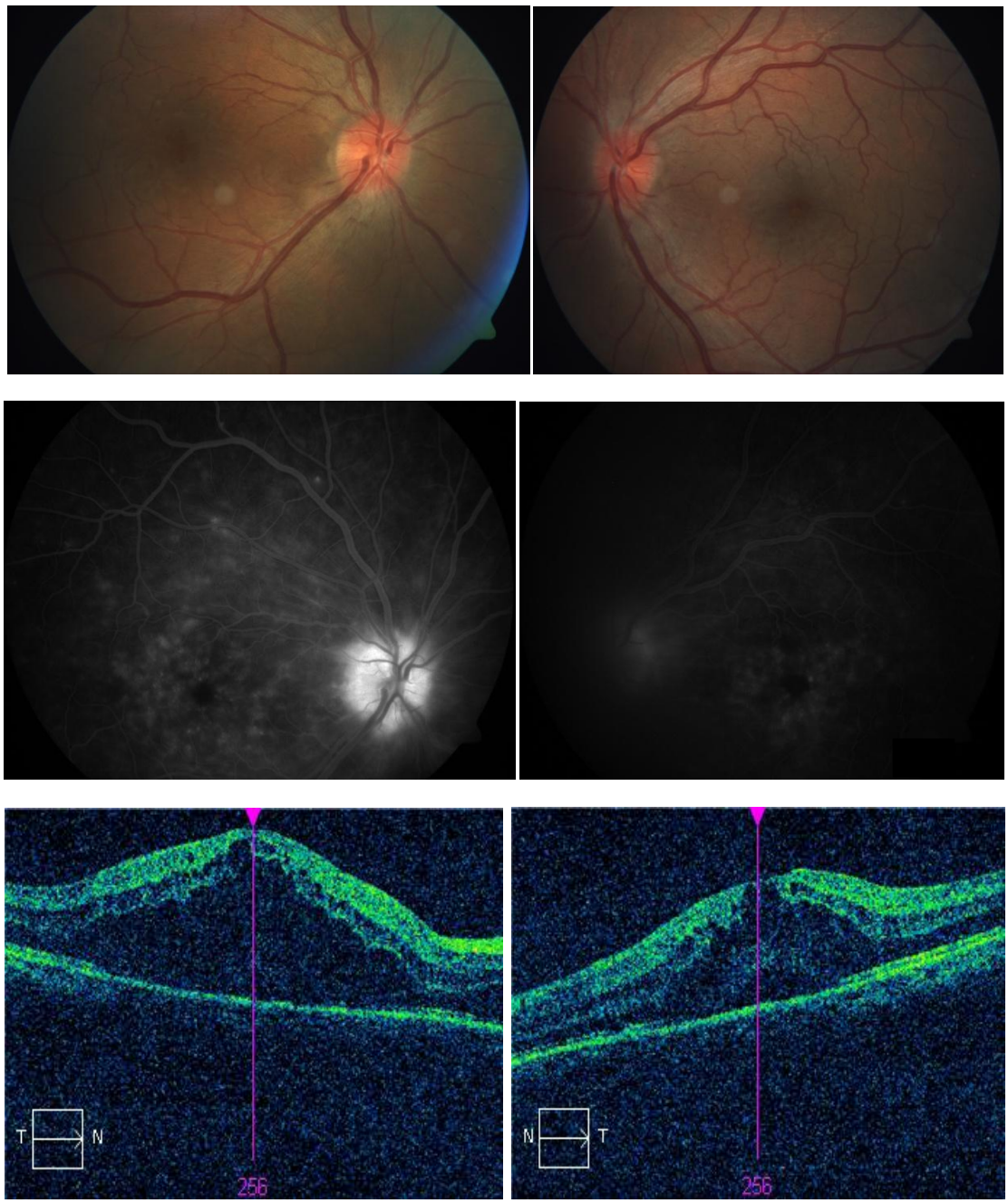


Fig 1: Fundus pictures showing cystoid macular oedema, FFA in late phase shows a flower petal pattern of hyperfluorescence in both eyes, OCT shows hypo reflective spaces with in retina, macular thickening and loss of foveal depression.

IV. DISCUSSION:

One of the first reports of macular edema was published by Edward Jaeger in 1856[16], where he described diabetic macular edema using a direct ophthalmoscope. Irvine in his paper described about CME after intra and extra capsular cataract extraction associated with a complicated cataract surgery.[6,7] Because of its unique anatomy, macula is most susceptible for CME. It has a central avascular zone, large number of cells with increased metabolic activity. Because of the water shed area between choroid and retina the absorption of fluid is slow in this area. This causes thickening of the retina and it can manifest as focal, diffuse or cystic edema clinically. The causes of macular edema can be attributed to surgery, diabetes, vascular occlusions, uveitis, epiretinal membrane, retinitis pigmentosa, intra ocular tumors, choroidal neovascualarisation and radiation retinopathy.[17]

Postoperative CME is a well-known entity and can develop after any operations such as cataract surgery, scleral buckling, pneumatic retinopexy, combined penetrating keratoplasty and trans-scleral sutured posterior chamber IOL implantation can be complicated by post-operative CME.[18]In general, intraocular surgery seems to trigger the accumulation of macrophages and neutrophils that are further activated by circulating inflammatory agents, including cycloxygenase and lipoxygenase metabolites and proteolytic agents, leading to the appearance of clinical signs of inflammation such as perilimbal injection and anterior chamber flare. Cytokines such as interferon-γ, interleukin-2 and tumor necrosis factor-α also participate in the process inducing the production of cycloxygenase.[19,20,21] Experimental studies of lens implantation in animal models have confirmed that trauma of the lens epithelial cells leads to the secretion of inflammatory mediators.[22] Other factors such as nitric oxide, complement and platelet-activating factor secreted by different cell types are believed to play important role in triggering inflammation postoperatively.[23]There is also alteration in the active transport mechanism for removal prostaglandins across anterior uvea by the release of inflammatory mediators leading to accumulation of prostaglandins.[24] Furthermore, the procedure of cataract surgery itself has been suggested recently to induce pro-inflammatory gene expression and protein secretion.[25] Inflammation produced due to surgery can also result in vitreo-macular contraction and traction on macula or formation of epiretinal membrane. These can be accelerated if there is trauma to iris, retained lens matter, malpositioning of IOL or vitreous loss during surgery. After a cataract operation, posterior diffusion of inflammatory factors leads to break down of blood retinal barrier (BRB). The BRB is responsible for restricting movement of plasma constituents into the retina and in maintaining retinal homeostasis. A disruption in BRB will lead to increased capillary permeability of the perifoveal network, and results in cyst formation and intraretinal fluid accumulation both intra and extracellularly.[26] Fluid accumulation disturbs cell function and retinal configuration. Müller cells are thought to act as metabolic pumps, which keep the macula dry. The accumulation of fluid in the outer plexiform layer is considered to be a late phenomenon following breakdown of the Müller cells. This produces the characteristic petalloid pattern of CME on fluorescein angiography. These patients present with the clinical features of reduced visual acuity, reduced contrast sensitivity, relative scotoma, metamorphopsia and altered color vision. Clinically macular edema can be diagnosed by using slit lamp biomicroscopy with a contact or non contact lens, characterized by an altered light reflex, with or without cystic spaces in a honeycomb pattern around fovea. It is important to distinguish the different varieties of CME that can range from subclinical CME, which can be picked up by fluorescein angiography findings only (angiographic CME) to symptomatic CME. In chronic cases clinically significant CME can persist for more than six months. [27] Cystoid macular edema (CME) is one of the most common causes of unexpected poor postoperative visual recovery after an otherwise uneventful surgery. The incidence of angiographic CME is estimated to be around 10-20%.[10]

Optical Coherence Tomography is a noninvasive new modality which gives a cross sectional image of the retina. It is an important diagnostic modality for various retinal disorders. It can give a quantitative measure of cystic spaces within the macula. Fluorescein angiography is the gold standard for diagnosing CME but it is an invasive procedure with complications ranging from urticaria, vomiting and pain at injection site to anaphylactic reactions and rarely death. OCT is a quick, noninvasive and quantitative procedure with no side effects. In a comparative study by Mitne et al, they found 88% correlation between the two methods.[28] Antcliff et al reported in their study that sensitivity and specificity of OCT was 96% and 100%.[29] To study the effects of phacoemulsification on macula Subramanian and associates conducted a prospective trial in which 81 eyes of 61 subjects were studied. Postoperatively they found that eight eyes (9.87%) demonstrated angiographic CME.[9] Although no exclusion was made with respect to existing systemic illness such as diabetes, hypertension or coronary artery disease a high incidence warrants investigation in uncomplicated cases.

Ursell and associates conducted a prospective study at British Teaching Hospital in 1996 to ascertain the incidence of cystoid macular edema (CME) after phacoemulsification. The rate of angiographic CME on day 60 was 19%. Visual acuity at each visit was significantly worse in the CME group. The previous studies indicate that incidence of pseudophakic CME also depends on the modality used for its detection.

We noticed in our study that there was a moderate increase in CMT in all patients even in those patients who did not develop CME in the 1st month though it did not cause a drop in visual acuity and the mean central macular thickness reverted back to pre-operative values at 12 weeks postoperatively. In cases which showed CME, an initial reduction in visual acuity was noticed with increased macular thickness. Mean macular thickness gradually reduced along with disappearance of cystic spaces over 12 weeks period. Incidence of CME was 2%, 3.3% and 2.7% during 1 week, 4weeks and 12 weeks postoperative visits respectively which is comparable to the results given by Ching et al and Perenté et al.

V. CONCLUSION:

It was noticed that the mean macular thickness increased in all patients during the first four weeks which returned back to the preoperative levels at three months. Visual acuity remained normal with respect to the mean macular thickness in cases that did not develop CME. Incidence of cystoid macular edema in our study was 2%, 3.3% and 2.7% at 1 week, 4weeks and 12 weeks postoperatively respectively, which was comparable with other studies. We believe that OCT is playing a considerable clinical role in evaluation of macular thickness and analyzing the morphology, especially in cases with post operative CME. Our results support the conclusion that OCT is more useful as compared fluorescein angiography in the diagnosis and management of the cases of CME since it is fast and non invasive method. OCT examination should be a part of preoperative evaluation, before any intraocular surgery.

Central Macular Thickness(CMT): - Pattern of variation noted in central macular thickness pre operatively, 1 week, 4 weeks, and 12 weeks postoperatively.

Table 1: Mean and standard deviation (SD) for central macular thickness (μm).

	CME PRESENT		CME ABSENT		TOTAL	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Pre Op	265.80	7.190	242.58	19.345	243.35	19.508
Post Op 1 week	**406.20**	246.181	248.42	17.729	253.68	52.329
Post Op 4 weeks	**475.60**	139.851	250.05	17.918	257.57	49.856
Post Op 12 weeks	**386.20**	58.947	247.52	17.523	252.14	31.842

Figure 2: Age distribution

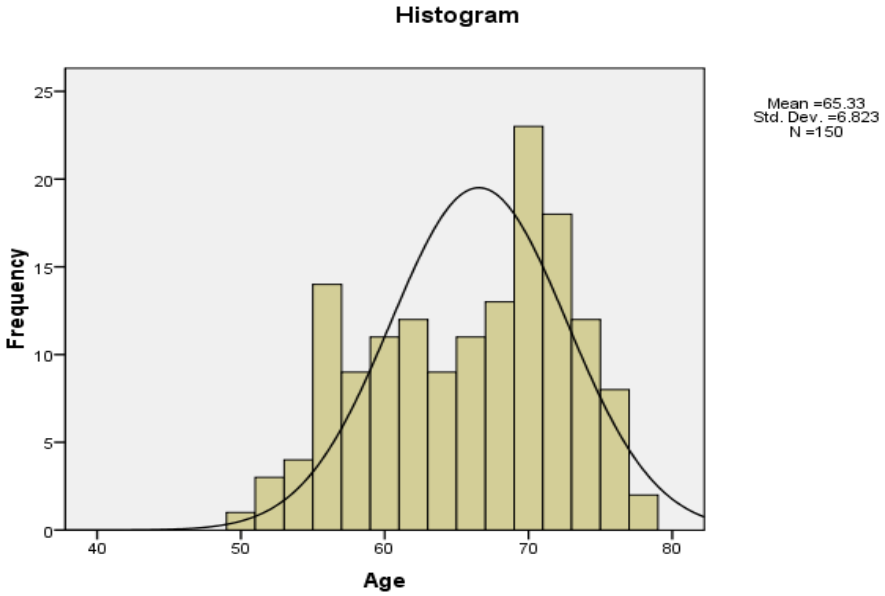
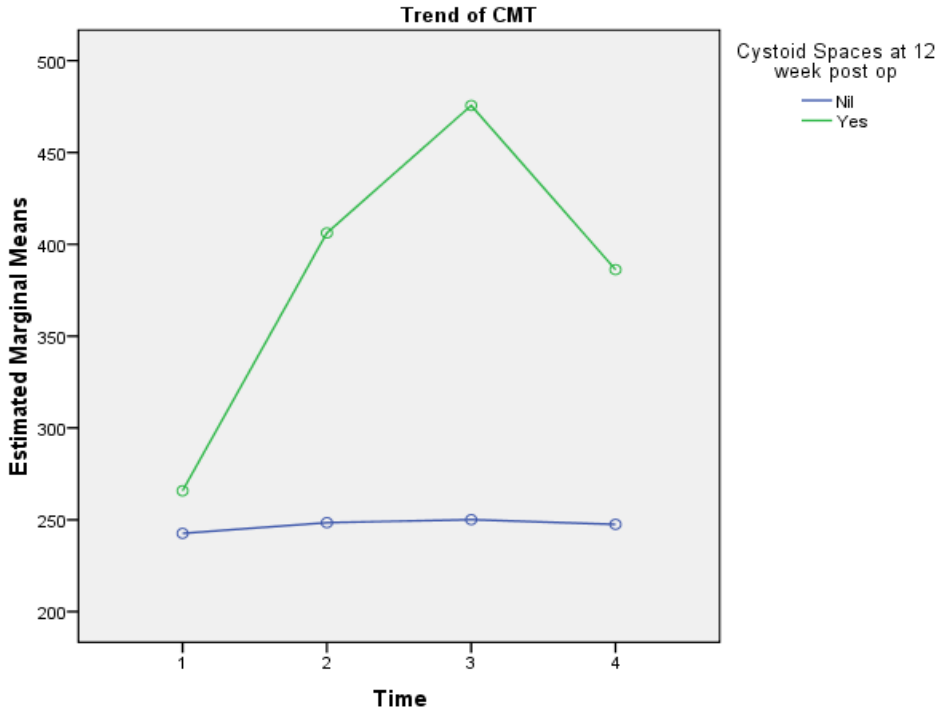


Figure 3: Central Macular Thickness(CMT): - Pattern of variation noted in central macular thickness pre operatively, 1 week, 4 weeks, and 12 weeks postoperatively



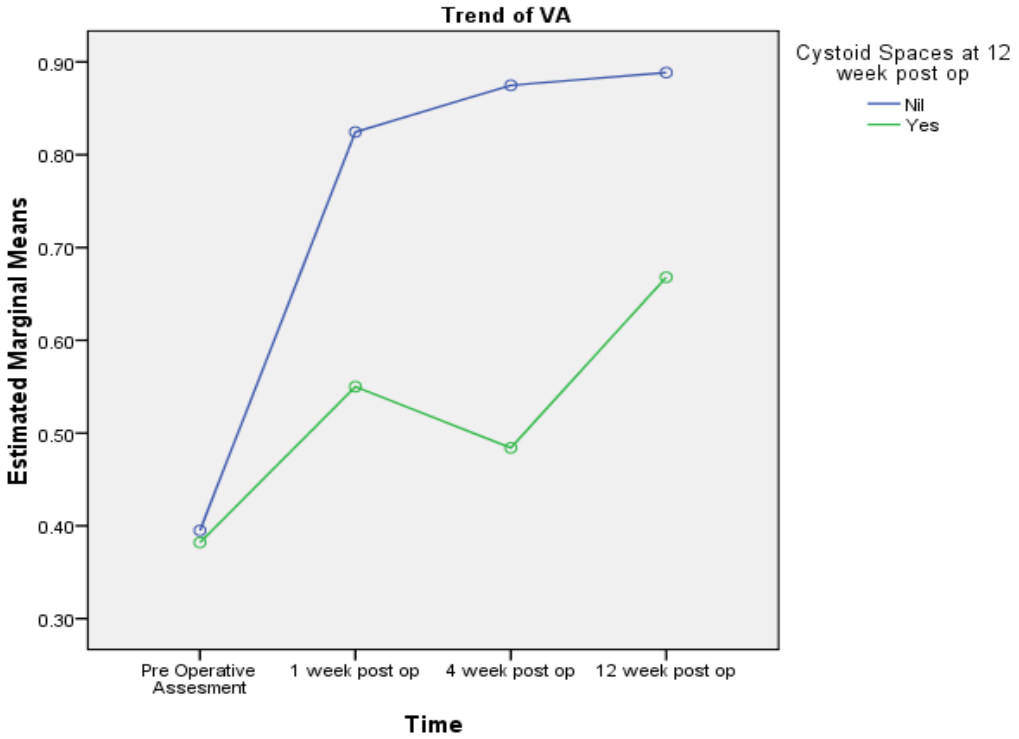


Figure : 4 Pattern in variation in Visual acuity

Table 2: Visual Acuity(VA): - Pattern of variation noted in visual acuity pre operatively, 1 week, 4 weeks, and 12 weeks postoperatively

	CME PRESENT		CME ABSENT		TOTAL	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Pre Op	0.3820	0.11256	0.3950	0.12856	0.3945	0.12774
Post Op 1 week	**0.5500**	0.29908	0.8245	0.17896	0.8153	0.18920
Post Op 4 weeks	**0.4840**	0.34100	0.8748	0.16068	0.8618	0.18173
Post Op 12 weeks	**0.6680**	0.23690	0.8885	0.15663	0.8811	0.16369

Table 3. Incidence of CME observed at 1 week, 4 weeks and 12 weeks postoperatively

	NO OF CASES WITH CME	**INCIDENCE**
Pre-op	Nil	Nil
1 week post op	3	2
4 weeks post op	5	**3.3***
12 weeks po	4	2.7

REFERENCES

[1]. Foster A, Johnson GJ. Magnitude and causes of blindness in the developing world. Int Ophthalmol 1990; 14(3): 135-40.

[2]. Datiles MB, Kinoshita JH. Pathogenesis of cataracts. In: Tasman W, Jaeger EA, eds. Duane's clinical ophthalmology, vol 1. Philadelphia: JB Lippincott; 1991. P.1-14.

[3]. Bruce GE. The role of nutrition in cataract. In: Tasman W, Jaeger EA, eds. Duane's clinical ophthalmology. Vol 1. Philadelphia: JB Lippincott; 1991. P.1-9

[4]. Linebarger EJ, Hardten DR, Shah GK. Phacoemulsification and modern cataract surgery. Surv-Ophthalmol 1999;44:123–47

[5]. Subramanian ML, Devaiah AK, Warren KA: Incidence of Postoperative Cystoid Macular Edema by a Single Surgeon. DJO 2009;4:235-6.

[6]. Gass JD, Norton EW. Cystoid Macular edema and papilledema following cataract extraction: a fluorescein fundoscopic and angiographic study. Arch Ophthalmol 1966;76:646–61.

[7]. Irvine AR. Cystoid maculopathy. Surv Ophthalmol 1976;21:1–17.

[8]. 8.Ching HY, Wong AC, Wong CC, Woo DC, Chan CW Cystoid macular oedema and changes in retinal thickness after phacoemulsification with optical coherence tomography. Eye. 2006;14:297–303.

[9]. 9.Gass J.D., Norton E.W. (1969). Follow-up study of cystoid macular edema following cataract extraction. Trans Am Acad Ophthalmol Otolaryngol, Vol. 73, No. 4, pp. (665-682). Gass J.D., Norton E.W. (1969). Follow-up study of cystoid macular edema following cataract extraction. Trans Am Acad Ophthalmol Otolaryngol, Vol. 73, No. 4, pp. (665-682).

[10]. Van der Schaft T.L., van Rij G., Renardel de Lavalette J.G. & Beekhuis W.H. (1989). Results of penetrating keratoplasty for pseudophakic bullous keratopathy with the exchange of an intraocular lens. Br J Ophthalmol, Vol. 73. No. 9, pp. (704-708)

[11]. Tunc M., Lahey J.M., Kearney J.J., Lewis J.M. & Francis R. (2007). Cystoid macular oedema following pneumatic retinopexy vs scleral buckling. Eye, Vol. 21, No. 6, pp. (831-834

[12]. Flach A.J. (1998). The incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery. Trans Am Ophthalmol Soc, Vol. 96, pp. (557-634) Yanoff M, Fine BS, Brucker AJ, et al. Pathology of human cystoid macular edema. Surv Ophthalmol. 1984;28(Suppl):505–11

[13]. Trichonas G, Kaiser PK. Optical coherence tomography imaging of macular edema. Br J Ophthalmol 2014;98:ii24-ii29.

[14]. Wittpenn J.R., Silverstein S., Heier J., Kenyon K.R., Hunkeler J.D. & Earl M., on behalf of the Acular LS for Cystoid Macular Edema (ACME) Study Group. (2008). A randomized, masked comparison of topical ketorolac 0.4 plus steroid vs steroid alone in low-risk cataract surgery patients. Am J Ophthalmol, Vol.146, pp. (554 –560)

[15]. Adhi M, Aziz S, Mohammad K, Mohammad I. Macular Thickness by age and gender in healthy eyes usind SD-OCT. DOI 2012;1371-72.

[16]. Datiles MB, Kinoshita JH. Pathogenesis of cataracts. In: Tasman W, Jaeger EA, eds. Duane's clinical ophthalmology, vol 1. Philadelphia: JB Lippincott; 1991. P.1-14.

[17]. In: Tasman W, Jaeger EA, eds. Duane's clinical ophthalmology, vol 1. Philadelphia: JB Lippincott; 1991. P.1-14.
[18]. Henderson BA, Kim JY, Ament CS, Ferrufino-Ponce ZK, Grabowska A, Cremers SL. Clinical pseudophakic cystoid macular edema: risk factors for development and duration after treatment. J Cataract Refract Surg. 2007;14:1550–8.
[19]. Tunc M., Lahey J.M., Kearney J.J., Lewis J.M. & Francis R. (2007). Cystoid macular oedema following pneumatic retinopexy vs scleral buckling. Eye, Vol. 21, No. 6, pp. (831-834
[20]. Flach A.J. (1998). The incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery. Trans Am Ophthalmol Soc, Vol. 96, pp. (557-634)
[21]. Wakefield D, Lloyd A. The role of cytokines in the pathogenesis of inflammatory eye disease. Cytokine. 1992;4:1–5
[22]. Miyake Y, Miyake K, Shiroyama N, et al. Classification of aphakic cystoid macular edema with focal macular electroretinograms. Am J Ophthalmol. 1993;116:576–83
[23]. Lightman S, Chan CC. Immune mechanisms in choroido-retinal inflammation in man. Eye. 1990;4:345–53
[24]. El-Harazi S.M., Feldman R.M. (2001). Control of intra-ocular inflammation associated with cataract surgery. Curr Opin Ophthalmol, Vol. 12, pp. (4–8)
[25]. Xu H., Chen M., Forrester J.V. & Lois N. (2011). Cataract Surgery Induces Retinal Pro- inflammatory Gene Expression and Protein Secretion. Invest Ophthalmol Vis Sci, Vol. 52, pp. (249 –255).
[26]. Yanoff M, Fine BS, Brucker AJ, et al. Pathology of human cystoid macular edema. Surv Ophthalmol. 1984;28(Suppl):505–11. Gass J.D., Norton E.W. (1969). Follow-up study of cystoid macular edema following cataract extraction. Trans Am Acad Ophthalmol Otolaryngol, Vol. 73, No. 4, pp. (665-682).
[27]. Trichonas G, Kaiser PK. Optical coherence tomography imaging of macular edema.Br J of Ophthalmol 2014;98:ii24-ii29
[28]. Grzybowski A, Sikorski BL, Huerva V. Pseudophakic CME: Update. Clinical Interv Aging 2016;11: 1221-1229.
[29]. Optical coherence tomography in patients undergoing cataract surgery Arq Bras Ophthalmol 2015; Vol 178.

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