"A ONE YEAR RANDOMISED CLINICAL TRAIL TO COMPARE THE EFFICACY AND SAFETY OF TOPICAL DIFLUPREDNATE OPHTHALMIC EMULSION 0.05% WITH TOPICAL PREDNISOLONE ACETATE 1% OPHTHALMIC SUSPENSION IN THE CONTROL OF POST-OPERATIVE INFLAMMATION FOLLOWING CATARACT SURGERY IN KLES DR.PRABHAKAR KORE HOSPITAL BELGAUM.

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Endorsement by the HOD, Principal/Head of the Institution

This is to certify that the dissertation entitled **"A one year randomized clinical trail to compare the safety and efficacy of Topical Difluprednate Ophthalmic Emulsion 0.05% with Prednisolone Acetate 1% Ophthalmic Suspension in control of Post-operative Inflammation following Cataract Surgery at KLES. Dr. Phabhakar Kore Hospital Belgaum "** is a bonafide research work done by REGISTRATION NO: BK0110003.

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LIST OF ABBREVIATIONS USED

1.	AC	=	Anterior Chamber
2.	ACTH	=	Adrenocorticotropin
3.	BSS	=	Balanced salt solution
4.	CME	=	Cystoid Macular Edema
5.	ECCE	=	Extracapsular Cataract Extraction
6.	FDA	=	Food and Drug Administration
7.	IOL	=	Intraocular Lens
8.	IOP	=	Intraocular Pressure
9.	KP’s	=	Keratic Precipitates
10.	NSAID’S	=	Non Steroidal Anti-Inflammatory Drugs.
11.	PCIOL	=	Posterior Chamber Intraocular Lens
12.	PMMA	=	PolyMethylMethAcrylate
13.	SICS	=	Small Incision Cataract Surgery

ABSTRACT

TITLE:

"A ONE YEAR RANDOMISED CLINICAL TRAIL TO STUDY THE EFFICACY ANS SAFETY OF TOPICAL DIFLUPREDNATE 0.05% OPHTHALMIC EMULSION WITH TOPICAL PREDNISOLONE ACETATE 1% OPHTHALMIC SUSPENSION IN CONTROL OF INFLAMMATION FOLLOWING CATARACT SURGERY IN KLES DR.PRABHAKAR KORE HOSPITAL BELGAUM."

BACKGROUND:

Cataract is responsible for 50% of blindness in the world; the overall prevalence rate varies from 1% to 4% of the population and it should be performed with equal emphasis on quality and quantity of surgery. Cataract surgery is the most common ophthalmic surgery performed. A mild postoperative inflammation may be considered a normal accompaniment of cataract surgery rather than its complication Surgical techniques in all fields of ophthalmology has evolved considerably over the years from transition to clear corneal incisions by anterior segment surgeons to adoption of small-gauge minimally invasive pars plana vitrectomies by vitreo-retinal specialists

Just as ophthalmologists have enjoyed advances in surgical technique and technology, patient expectations of their results have grown proportionately. Currently, the most widely prescribed strong topical corticosteroid in India is prednisolone acetate1%.While, it controls inflammation effectively, it has not been shown to consistently address post-operative pain and discomfort in a large clinical trail. Thus, Difluprednate is the first ophthalmic steroid developed in the past 35 years

with high potency, a favorable safety profile, and the ability to reduce postoperative pain.

OBJECTIVES:

1. To study the efficacy and safety of topical Difluprednate 0.05% ophthalmic emulsion with topical Prednisolone Acetate 1% ophthalmic suspension in control of post-operative inflammation following cataract surgery.
2. Measurement of IOP in study population.

METHODOLOGY:

A total of hundred patients diagnosed senile cataracts coming to KLES Dr. Phabhakar Kore Hospital Belgaum over a one year period who fulfilled the inclusion criteria were enrolled in the study. The patients were divided into two groups. The post-operative grades of inflammation following cataract surgery was assessed by slit-lamp examination.

Statistical analysis was done using Student's 't' test, chi square test and paired t test.

RESULTS:

In the present study, the average age group of patients in Difluprednate group was 60.5 years while in Prednisolone group was 58.8 years. Maximum of the total patients were in age group of 60 to 69 years, male: female ratio in Dfluprednate group was 29:21 and in Prednisolone group was 35:15. Majority of the cases were senile immature cortical cataract (50%) out of which 22 patients (44%) were in Difluprednate group and 25 patients (50%) in Prednisolone group. Maximum number of patients were in the range of 14.6 to 15.9 mmHg in terms of IOP.

In the present study, only12% in Difluprednate group and 12% patients in Prednisolone group had lid edema on Day 1. By Day 7 the lid edema subsided in both

the groups. Conjunctival congestion persisted in 6% patients in Prednisolone group as compared with none of the patients in Difluprednate group on day 15.Ciliary congestion was seen in 3% patients in the Prednisolone group as compared with none of the patients in Difluprednate group by Day 15. Corneal edema was reduced equally by both the drugs at all observation times.

Anterior chamber flare and cells –Mild flare was seen in 22% of patients in Prednisolone group and in only 8% of patients in the Difluprednate group by day 15, which regressed completely in both the groups by day 30. When the total scoring of all these parameters was compared, in Prednisolone group 34% of the patients persisted with mild (Grade1-3) inflammation on Day 15 as compared with 26% of the patients in Difluprednate group.

CONCLUSION:

From, the present study it was noted that both Difluprednate Ophthalmic Emulsion 0.05% eye drops and Prednisolone Acetate.1% eye drops were equally effective in reducing the inflammation following uncomplicated cataract surgery. With proven efficacy of Difluprednate, we now have a new standard for potency in a topical corticosteroid, with excellent anti-inflammatory properties and an ideal formulation for our patients.

KEYWORDS: Difluprednate(Durezol), Prednisolone Acetate, Ocular Inflammation, Dose uniformity.

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INTRODUCTION

Cataract is responsible for 50% of blindness in the world; the overall prevalence rate varies from 1% to 4% of the population and it should be performed with equal emphasis on quality and quantity of surgery.[1]

Cataract surgery is the most common ophthalmic surgery performed. A mild postoperative inflammation may be considered a normal accompaniment of cataract surgery rather than its complication.[1]

Surgical techniques in all fields of ophthalmology has evolved considerably over the years from transition to clear corneal incisions by anterior segment surgeons to adoption of small-gauge minimally invasive pars plana vitrectomies by vitreo-retinal specialists[2].

This inflammation is self-limiting and invariably subsides within two or three days. Persistent and more severe uveitis can have a detremental effect on the patients final vision after surgery, and the control of inflammation is a chance to make a difference in the final visual outcome. Ocular inflammation following cataract surgery can be due to surgical trauma it self and due to various physical, chemical and biological agents introduced during the surgery. The host response to these injurious agents in the form of inflammation is a complex interaction of immuno reactive cells, their products and other chemical mediators of inflammation. More is known today about chemical mediators of inflammation-the prostaglandins, the kinins the complement system etc and their role in inflammation. It is becoming more apparent that prostaglandins help to mediate the response of eye to acute trauma. This irritative response is characterized by hyperemia of the conjunctiva, miosis, disruption of the

blood aqueous barrier and a transient increase in intraocular pressure followed by relative hypotension.[2]

In the recent years, operative technique in cataract surgery has improved and the operation is becoming less traumatic to the eye. As a result there is less postoperative inflammatory reaction and less break down of the blood aqueous barrier. The synthesis of cortisone in 1950 was a major break through in the treatment of inflammatory diseases in general and this applies to ocular inflammation also[3].

In the immediate post-operative period, topical corticosteroids are employed to suppress the production of inflammatory mediators, offering local treatment without the risk of systemic adverse effects. By inhibiting the release of arachidonic acid from the cell membrane phospholipids, corticosteroids prevent the formation of both leukotreines and prostaglandins, disrupting the inflammatory cascade[4]. These agents are continued until the anterior chamber (AC) reaction has resolved and the blood-aqueous barrier has been reestablished[4.]

Topical corticosteroids are commonly used as a routine treatment during several weeks postoperatively in order to reduce the inflammatory reaction. However, adverse effects of steroids are well known and include intraocular hypertension in susceptible patients (steroid responders) impairment of cicatrisation (inhibition of wound healing) and increased risk of infections particularly viral ones.[5]

Patients expectations of surgical results and post-operative comfort have advanced with evolving surgical techniques and instrumentation. Despite this, no strong steroids have been approved by the FDA for the treatment of ocular inflammation since 1973. Technological improvements in pharmaceutical development now permit the creation of potent topical steroids with better

bioavailability and rapid local metabolism, both of which minimize systemic exposure. Although weaker steroids and nonsteroidal anti-inflammatory drugs may have a better safety profile, many patients will require the strongest available steroid to control inflammation.[6]

Just as ophthalmologists have enjoyed advances in surgical technique and technology, patient expectations of their results have grown proportionately. Currently, the most widely prescribed strong topical corticosteroid in India is prednisolone acetate1%.While, it controls inflammation effectively, it has not been shown to consistently address post-operative pain and discomfort in a large clinical trail[7].

Difluprednate Ophthalmic Emulsion 0.05% was approved by US Food and Drug Administration (FDA) for treatment of inflammation and pain associated with ocular surgery in June 2008. It is the first ophthalmic steroid approved by FDA since 1973[7].

A study was to done to assess the efficacy of Difluprednate 0.05% ophthalmic emulsion to a placebo in the treatment of inflammation associated with ocular surgery. Difluprednate 0.05% Ophthalmic Emulsion safely clears post-operative inflammation with no serious adverse effects, fewer adverse affects were noted in Difluprednate treated groups than the placebo group. Thus, Difluprednate is the first ophthalmic steroid developed in the past 35 years with high potency, a favorable safety profile, and the ability to reduce postoperative pain[7].

AIMS AND OBJECTIVES

1) To study the efficacy of topical Difluprednate Ophthalmic Emulsion 0.05% versus Topical Prednisolone Acetate 1% Ophthalmic suspension.

2) Measurement of IOP in the study population.

REVIEW OF LITERATURE

History:

Large incision nuclear expression cataract surgery, or extracapsular cataract extraction (ECCE), has been the mainstay of cataract surgery for the past three decades. The experiences gained from this form of surgery have in many ways contributed to the development of the smaller incision techniques. Much of the value of the large incision procedure is the lower cost and minimal instrumentation with which the surgery can be performed, a significant factor in third-world care. ECCE is also less demanding in terms of skill yet provides excellent rehabilitation of blindness caused by cataract at the expense of only recovery time and stability of refraction over the first year. With time, cataract removal became more successful, thanks to complete cortical removal and IOL stabilization against the capsular bag. This was followed by the desire to improve optical outcome by making smaller incisions with consequently less astigmatism and instability. The means of doing this slowly became available with the development of phacoemulsification, but the costs of equipment and the steepness of the learning curve created the need for techniques that reduced the requirement for sophisticated means of decreasing the nuclear size in order to remove fragments through smaller incisions. This led to the development of manual nuclear expression surgery through smaller incisions, or the so-called mininuc technique.[8]

Changes induced in anatomy and physiology of eye by cataract surgery has been summarized by **Gillman AM** (1965) as follows:[8]

1. Changes at site of surgical manipulation

    a) Local tissue necrosis

    b) Axon - reflex vascular reaction.

    c) Liberation of intracellular metabolities, enzyme substrates and chemotactic substances.

    d) Surgical exposure permitting fortuitous airborne and instrument borne contamination with pathogenic organisms and agents.

It is the stimulus for the initial phase of wound repair, which is normally referred to as the 'lag phase'. We now know that this phase is in reality a period of intense biochemical activity. The cells in the area of repair become organized and develop their enzyme machinery before synthesis of new connective tissue, which occurs in the 'fibroblastic phase' of healing[8]. If the response is excessive or other post operative complications arise, the innocuous surgical inflammatory response may cause troublesome postoperative uveitis.[8]

There is little bleeding because of relative avascularity of tissue involved. Endowed with intense axon-reflex activity, these tissues respond to cutting capillaries and some of the larger vessels by an immediate localized vasoconstriction and vessel retraction. Axon reflexes are mediated through defense against mechanical injury. Arterial vessel tends to contract over a considerate length, often to complete obliteration. In a few seconds, contraction subsides but a clot has already formed and bleeding is prevented. The short limited phase of localized vasoconstriction and vessel

retraction results in immediate blanching in the adjacent sclera. However, reactions in iris vessels are probably of longer duration, this stage followed by a phase of hyperemia, vasocongestion, vasodilatation and increased capillary permeability. This reaction is more prolonged and is characterized by edema, localized bleeding, tissue swelling and increased aqueous turbidity[8].

2. Changes induced by AC decompression

    i. Loss of anatomic limits of AC and exteriorization of the AC

    ii. Incisional hypotension.

    iii. Early wound incarcerations.

3. Changes induced by posterior decompression

i. Vasocongestion, edema and hemorrhage in retina and choroids.

ii. Serous and hemorrhagic separation of retina.

iii. Perichoroidal serous and hemorrhagic separation with extension to the periciliary space with the implicit threat of inhibitional hyposecretion hypotension.

iv. Forward displacement of vitreous, loss of fluid in formed vitreous and anterior and posterior vitreous separations. Inflammatory response to surgical manipulation is still little understood, but evidently is essential for normal healing.[8]

Pathogenesis of ocular inflammation following cataract surgery:

Cataract surgery, which requires decompression of the globe, manipulation of the iris, lysis of zonules, irrigation of AC, injection of solutions and placement of sutures, would reasonably be expected to result in transitory uveitis. This reaction subsides rapidly and usually leaves no permanent sequelae. However some patients are troubled by a more persistent uveitis for which there is no cause; though the reason is unknown it is reasonable to assume that the tissues of some patients are sufficiently sensitive to respond to the manipulation of surgery by remaining chronically inflamed.[9]

Effects of surgical decompression on posterior portion of globe cause more dramatic and profound sudden release of intraocular pressure. This initiates profound changes in capillary beds of ciliary body, choroid and retina resulting in capillary dilatation, congestion, edema and hemorrhage. Marked changes in vitreous also occur exemplified by vitreous turbidity, anterior ectasia of vitreous and increase vitreous morbidity. In most eyes, these changes are tolerated well, with the cells in aqueous and vitreous disappearing after several days as global integrity is restored.[10]

Foreign material introduced during surgery:

Particles of lint, rubber, cilia, cotton and suture material are occasionally found in the AC resting on iris, posterior capsule or anterior vitreous face, remain unchanged for long periods, rarely cause significant intraocular inflammation. Introduction of materials in the eye like saline solutions BSS, α-chymotrypsin, acetylchloline, epinephrine, viscoelastic material and air play a role in some cases of unexplained postoperative uveitis. Jaffe NS in 1970 found an effective method of eliminating particulate debris from air and all ophthalmic solutions intended for

intraocular use. It involves using a pre-sterilized, disposable micropore filter. Laminar airflow systems are being used increasingly in operating rooms throughout the United States to prevent particulate atmospheric debris from entering the eye[11].

Based on research, Galin MA, Tuberville AW and Dotson RS**,** (1982) have outlined possible sequence of events following IOL implantation[12].

1. Surgery permits high levels of protein, to enter the eye (flare and cells).
2. IOL directly (nylon, polypropylene) and indirectly (PMMA) activate initial high levels of complement. This leads to generation of biologically active C_5-derived peptides.
3. C_5 derived peptides induce increased vascular permeability (anaphylatoxin activity, limbal flush, uveal leakage), which perpetuates leakage of complement components and IgG into the eye. Complement components "feed" or amplify the sequence of complement activation, while IgG coats PMMA, forming immuno-globulin aggregates.
4. PMMA bound IgG further activates the complement.
5. Leucocyte influx is augmented as a result of degeneration of C_5 derived chemotactic peptides (cells, KP's hypopyon)
6. Finally reaction of leucocytes with C_5 proteins and PMMA bound IgG can lead to marked tissue injury from the generation of oxygen derived free radicals, release of leucocyte lysosomal enzymes and white cell adhesion and aggregation to surrounding ocular tissues (corneal edema, CME) .

These sequences of events are self-limited. Inflammatory mediators are released from tissues during injury or inflammatory reactions[12].

Udell. J.J and Abelson M.B. (1983) classified mediators in groups according to their mode of action[13].

1. Substances that act directly at the cell membrane by way of specific receptors (e.g. histamine).
2. Substance of cell or plasma origin that cause direct damage to tissue (e.g. major basic protein and component).
3. Chemotactic factors that attract cells such as eosinophils and macrophages to site of inflammation. (e.g. arachidonic acid metabolities.)

Some of the mediators known to be relevant to Ophthalmology are histamine, prostaglandins, complement, bradykinin and serotonin.

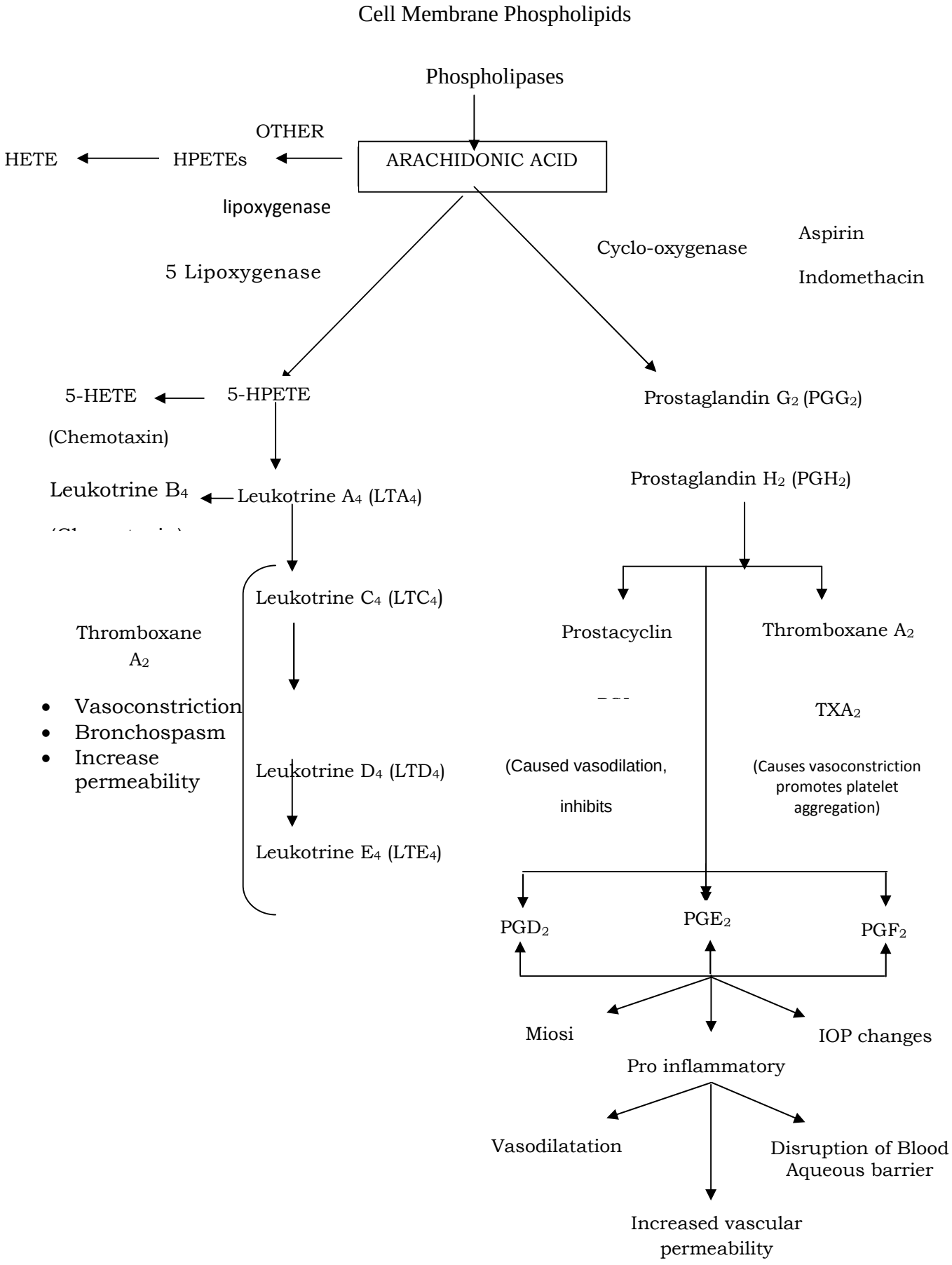
Prostaglandins

Ambache N and Brummer HC (1968) demonstrated that a substance of iris tissue extraction, irin, is capable of producing atropine-resistant miosis of the pupil. Ambache N in 1978 found that Irin was a mixture of prostaglandins that help to mediate the response of eye to acute trauma. It is becoming more apparent that prostaglandins help to mediate the response of eye to acute trauma[14]. Neufeld AH and Sears ML (1973) in their study found that this irritative response is characterized by hyperemia of the conjunctiva, miosis, disruption of the blood aqueous barrier and a transient increase in IOP by relative hypotension. Source of prostaglandins in uveitis may be leucocytes. Masuda K, Izawa Y and Mishima S (1973) reported that the prostaglandins play a role in the development of ocular inflammation, which may be associated with the effect on ocular permeability. Prostaglandins are not stored in the cells and their presence in aqueous is the result of denovo synthesis, because the eye does not contain the enzyme 15-prostaglandin dehydrogenase to deactive

prostaglandins, their removal depends on an active transport pump located in the ciliary epithelium.[15]

Malmsten C (1986), found that Prostaglandins are manufactured enzymatically in the cell from unsaturated fatty acids as Arachidonic acid. This precursor is itself formed by linoleic acid, an essential constituent of human and animal diets.[16]

Arachidonic acid is a poly-unsaturated fatty acid that is present in large amounts in phospholipids of cell membrane. It is released from membrane phospholipids due to activation of cellular phospholipases by inflammatory stimuli or other chemical mediators such as C5a. During inflammation, lysosomes of neutrophils are believed to be an important source of phospholipases. The inhibition of prostaglandin synthesis has been suggested as the mechanism of action of many NSAIDs.[16]


Cell Membrane Phospholipids
Phospholipases
OTHER
HETE
HPETEs
ARACHIDONIC ACID
lipoxygenase
5 Lipoxygenase
Cyclo-oxygenase
Aspirin
Indomethacin
5-HETE
5-HPETE
Prostaglandin G2 (PGG2)
(Chemotaxin)
Leukotrine B4
Leukotrine A4 (LTA4)
Prostaglandin H2 (PGH2)
Leukotrine C4 (LTC4)
Thromboxane A2
Prostacyclin
Thromboxane A2
TXA2
Vasoconstriction
Bronchospasm
Increase permeability
Leukotrine D4 (LTD4)
(Caused vasodilation, inhibits
(Causes vasoconstriction promotes platelet aggregation)
Leukotrine E4 (LTE4)
PGD2
PGE2
PGF2
Miosi
IOP changes
Pro inflammatory
Vasodilatation
Disruption of Blood Aqueous barrier
Increased vascular permeability

MANIFESTATION OF POST OPERATIVE OCULAR INFLAMMATION

The Manifestations of postoperative inflammation that is pain, redness and edema occur in every tissue of the eye.

Conjunctiva

Conjunctiva shows congestion and chemosis. Surgical trauma to the tissues results in increased permeability of vessels. Hence there is edema and exudation. Release of bradykinin and prostaglandins also causes pain.[17]

The inflammatory edema (chemosis) is caused by exudation of fluid and cells into the loose tissue. Release of bradykinin and prostaglandins also causes pain[17].

Cornea

Corneal edema is one of the most serious complications of cataract surgery. The refractive power of the anterior corneal surface is 49 D, whereas at its posterior surface it is 6 D. Thus irregularities at the anterior surface, such as epithelial edema, have a far greater effect on visual acuity than posterior surface irregularities.[17]

a) Epithelium:

Epithelial edema may be intracellular (from the hypoxia created by prolonged contact lens use) or intercellular (where high intraocular pressure forces aqueous through the endothelial barrier into the stroma and the intercellular spaces of the epithelium).Epithelial edema almost always is present in corneal inflammation and varies from mild edema to frank vesicle formation.[17]

The edema produces a loss of corneal transparency .In superficial lesions edema is due to absorption of relatively hypotonic tears, Edema of the epithelium appears clinically as fine vesicles. Accidental chemical cauterization with solutions

used for surgical preparation can cause epithelial clouding. Once dry ,the epithelium loses its opacity,both by loss of surface smoothness and by clouding. Balanced salt solution should be applied to keep the epithelium moist.[17]

b) Stroma:

Hedbys BO and Dohlman CH (1963) in their study reported that corneal stromal swelling results in a pressure exerted against Descemet's membrane and the endothelium that measures 50 to 60 mm Hg in the normal state[18]. Hedbys BO and Mishima S (1962) found that because the swelling pressure of the cornea depends on hydration, it has been correlated with corneal thickness. Hedbys BO, Mishima S and Maurice DM (1963) reported that the pressure of the interstitial fluid of the stroma (in contrast to the stromal solids) that draws fluid in is normally negative, and the magnitude, of the fluid pressure is equal to the difference between the swelling pressure and the intraocular pressure. Because the stromal fluid pressure is negative, the stroma tends to imbibe fluid from the surrounding tears, limbal vessels, and aqueous. The limiting cellular layers (epithelium and endothelium) serve as a barrier against the flow of fluid into the stroma.[18]

When the stromal fluid pressure becomes positive, the epithelium becomes edematous, with the edema beginning in the basal cell layers and spreading through the epithelium and occasionally resulting in subepithelial bullae. For the epithelium to be pushed off the stroma, or for this to appear so, the following obstacles must be overcome: The epithelial attachment to Bowman's membrane by the desmosomes must be severed, and the negative pressure of the stromal tissue, which draws the epithelium to the underlying tissue, must become positive. This is the cause of Bullous Keratopathy.[18]

In the early stages stroma is edematous. After months to years, opacification occurs in an irregular, stellate fashion just anterior to Descemet's membrane. Folds in Descemet's membrane persisting for a long period become fixed scars. The effect of this opacification decreases visual acuity, but it is not the main factor because epithelial edema is usually present long before opacification of the stroma occurs.[18]

c) Descemet's membrane

Clinically, Descemet's membrane can peel away from the stroma as a result of sharp instrument contact during cataract surgery. This will result in a localized area of stromal edema**.** Leber in 1887 found that the traumatic variety (the so called Deep Striate Keratitis) is the most common and is one of the frequent sequels of cataract surgery, appearing a few hours after the operation and persisting for some days or weeks, gradually fading away. Fine parallel lines of grey opacity run down the center of the cornea from the wound, sometimes they branch or cross each other in an irregular pattern, while straight reflex dark bands produced optically by folds in the membrane may cross them perpendicularly. Especially after difficult extractions where there has been some folding of the cornea there may be a added diffuse opacity, due to edema resulting from damage to endothelium.[19]

d) Endothelium:

The endothelial barrier is also damaged by mechanical or chemical insults, disease states, oxidation of intracellular glutathione with diamide, pH outside the range of 6.8 to 8.2, preservatives such as sodium bisulfite (the antioxidant for epinephrine), thimerosal, and benzalkonium chloride. If barrier function damage is great enough, the leak rate will exceed the pump rate, leading to increased corneal thickness and epithelial edema. The endothelial pump consists of enzymes in the

lateral plasma membrane of the cell. Alteration of the function of the pump leads to corneal edema.[19]

Yi DH and Dana MR (2002) in their study found that corneal edema from inadequate endothelial pump function is one of the most common complications of cataract surgery. Various causes for this endothelial dysfunction can be divided into four categories including [20]

(a) Mechanical injury,

(b) Inflammation/infection,

(c) Chemical injury,

(d) Concurrent eye disease.

Glasser D B, Schultz RO and Hyndiuk RA (1992) studied the role of viscoelastics, cannulas, and irrigating solution additives in post-cataract surgery corneal edema and found that cases of unexpected corneal edema can often be traced to unrecognized preoperative endothelial dysfunction or to toxicity of intraocular medications used during surgery and that the use of re-usable cannulas with viscoelastics is highly likely to result in toxic residues being introduced onto the eye, and must be avoided.[21]

There appear to be wide clinical variations in the onset of corneal edema among patients in response to similar insults. Irvine AR and Irvine AR Jr. (1953), in their study found a striking similarity between the concentration of these cells in the two eyes of one individual but a significant difference in cellular concentration in the eyes of other individuals. In addition endothelial population decreases with age. Thus it might be inferred that these variations in cellular concentration indicate the levels of intraocular pressure that must be exceeded before corneal edema ensues.[22]

Endothelial cell loss after cataract surgery:

Trauma during cataract surgery is the most common cause of postoperative corneal edema. Hayashi K and others (1996) reported the cases of such trauma recently in a prospective study of 859 consecutive eyes that had a phacoemulsification with either a polymethylmethacrylate (PMMA) lens, a three-piece silicone lens, or a three-piece acrylic lens. They found that the most significant factor in endothelial cell loss during phacoemulsification is the hardness of the nucleus. The chance of endothelial contact can occur when a segment of hard nucleus is emulsified.[22]

Anterior Chamber

Anterior chamber shows turbid or plasmoid aqueous containing proteins and cells. The plasmoid aqueous is visible as flare in the AC. Surgical trauma results in the breakdown of the blood-aqueous barrier. The blood-aqueous barrier is a cellular barrier constituted by the ciliary processes. The capillaries of the ciliary processes have fenestrated walls that are highly permeable but the pigmented and the non-pigmented ciliary epithelia are both provided with zonulae adherentes and occludentes as well as maculae occludentes, which serve as relative, but efficient diffusion barriers. Sander DR, Kraff MC, Lieberman HL, et al (1982) have reported that the breakdown of the blood aqueous barrier results in the leakage of proteins and cells into the AC and is accepted as clinical sign of anterior uveitis. This results in chemotaxis and further augmentation of the inflammatory processes. This can result in the formation of an exudative membrane over the pupil or the intra ocular lens.[23]

The conventional way of evaluating ocular inflammation using the slit lamp to estimate flare and cells gives, at best, semi quantitative results.

Fearnley IR, Spalton DJ and Smith SE (1987), have advocated the use of anterior segment ocular fluorophotometer to permit greater objectivity, better reproducibility and quantification of observations. This technique assumes the breakdown of the blood-aqueous barrier, as measured by following fluorescein accumulation of cells and proteins within the anterior chamber of the eye. However, this invasive, cumbersome method cannot be routinely used on a large scale. [24]

Spalton DJ and Shah SM (1991), have reported in their study that that Laser Flare-cell photometry provides an objective and quantitative measurement of anterior chamber flare and cells. It is easy and quick to perform and seems to be more precise than fluorophotometry.[25] El- Maghraby A, Marzouki A, Matheen TM et al (1992) have studied that the reproducibility and validity of laser flare–cell meter results in measuring inflammation after cataract surgery have been well established and are increasingly used to document postoperative inflammation.[25]

Cystoid Macular Edema

It has been known for some time that cystoid macular edema (CME; Irvine-Gass syndrome) may occur at some point during the postoperative period of cataract extraction. In 1953, Irvine described a syndrome that now bears his name. By definition, this condition included improvement of vision after cataract surgery followed by diminution of vision associated with postoperative rupture of the anterior hyaloid membrane, with or without adherence of vitreous to the surgical wound.[26]

The following are some of the proposed mechanisms that attempt to explain the altered blood retinal barrier[26]:

Mechanical disturbances such as vitreous incarceration in the wound, vitreous traction at the macula, hypotension and turbulence retinopathy associated with endophthalmodonesis can lead to CME.

Inflammatory disturbances that cause incompetence of capillary walls, osmotic gradients between vitreous and serum or primary retinal vein phlebitis can lead to altered blood retinal barrier and hence CME.

Systemic factors such as hypertension and diabetes mellitus also predispose the patient to CME.

Other proposed causes, such as topical adrenergic compounds, hyaluronidase in the anaesthetic solution, photic damage to the fovea from exposure to the light of the surgical microscope, inflammation and prostaglandins.

In the recent years considerable attention has been directed towards prostaglandins as chemical mediators of CME**.** Tennant and Yannuzzi and Wallyn in 1976 reported on treatment of chronic CME with oral indomethacin. Miyake in 1977 postulated that aphakic CME may occur as a result of prostaglandins synthesized intraoperatively in the iris.[26]

A study by Jaffe NS and coworkers in 1979 and 1982 indicated that an intact posterior capsule lessens the incidence of CME in eyes with implants compared with eyes that have had intracapsular cataract extractions. Winslow RL, Taylor BC, Harris WS (1978) showed a significantly lower incidence of this disorder in eyes after phacoemulsification when the posterior capsule was not excised. Moses L (1979) reported much lower incidence of CME in eyes after phacoemulsification or planned ECCE compared with intra capsular extraction.[26]

Clinical findings:

A more accurate picture of the Irvine-Gass syndrome has evolved as a result of observations made by Gass JDM and Norton EWD (1966). The typical patient undergoes an uneventful cataract extraction. During the postoperative course, good visual acuity is attained. However, 1 to 3 months after surgery, visual acuity decreases to 20/50 to 20/100. The onset may be delayed. The eye may become irritable and photophobic and suffer recurrences and remissions of circumcorneal injection. Ophthalmoscopy reveals little except the loss of the foveal reflex and a yellowish reflex or spot that appears to lie deep in or behind the retina. The cystoid spaces are best seen by using red-free light, which renders their inner walls visible.[26]

Biomicroscopic examination with a Hruby or fundus contact lens reveals a characteristic honeycomb lesion showing one or more larger cystoid spaces centrally, with any number of smaller oval spaces around them. Small perifoveal hemorrhages are common. The retina may be greatly thickened, and the lesion may occupy an area as large as 1.5 to 2 disc diameters. Capillary microaneurysms are frequent. Gass JDM and Norton EWD**, (**1969) found that wrinkling of the inner retinal surface (pucker) resulting from contraction of a semi translucent preretinal membrane has been reported. Papilledema may occur in some cases and may be associated with peripapillary hemorrhages.[26]

As a diagnostic tool, fluorescein angiography has proved of great value.

The macular pattern, which is well developed in most patients in 5 to 15 minutes (although it requires more than 30 minutes in others), consists of a stellate pattern with feathery margins. The dark septa in the macular area, which compartmentalize the pattern, are probably attributable to Muller's supporting fibers

of the retina. [26]

In the original definition of his syndrome, Irvine stated that the anterior hyaloid membrane must be ruptured. However, the same pathologic process is found in eyes with an intact anterior hyaloid membrane or after an extra capsular lens extraction with an intact posterior capsule. Of 64 eyes with postoperative CME, Gass JDM, Norton EWD (1969) observed an intact anterior hyaloid membrane in 34. In 30 eyes the anterior hyaloid membrane was ruptured, and in 25 vitreous was attached to the posterior surface of the wound.[26]

The report of the Colloborative Miami Study suggests an unacceptably high incidence of cystoid macular edema occurring in younger persons in whom a Copeland lens has been implanted.Subsequent studies have suggested that the use of iris-plane lenses and extracapsular cataract extractions may decrease the occurrence of this entity to the level associated with routine cataract extractions. Since, as previously,there is a high incidence of prolonged iridocyclitis associated with lens implantation,an increased incidence of corticosteroid responsive macular edema is often seen in these patients. Patients developing this type of presumptive cyclitic macular edema may require systemic, as well as periocular or topical corticosteroids, to alleviate the condition. If a patient maintains a persistent inflammatory response following lens implantation with cystic edema and this response is resistant to medical therapy ,removal of the pseudophake should be considered. [27]

Postoperative CME is associated with such a diversity of conditions that it is unlikely to have a single cause and surgical trauma is a significant cause.[27]

TOPICAL STEROID TREATMENT OF OCULAR INFLAMMATION

Introduction, History and Source

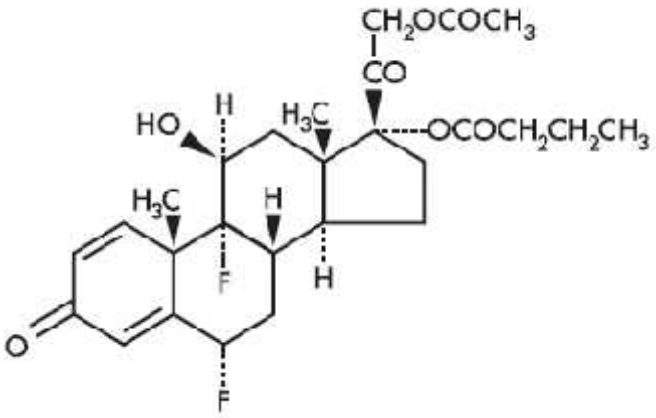
The isolation of cortisone (compound E) in 1935, by Edward C. Kendall and the subsequent clinical demonstration of the dramatic beneficial effects of this compound and adrenocorticotropin (ACTH) in the treatment of acute rheumatoid arthritis by Philip S. Hench and associates in 1948 marked a turning point in modern medical therapeutics. In 1950**,** Gordon and McLean introduced the use of corticosteroids and ACTH to ophthalmic practice.[28]

Official Drug Name And Chemistry

All corticosteroids are 21 carbon molecules consisting of a cyclopentoperhydrophenathrene nucleus, three hexane rings, and one pentane ring designated A, B, C, and D. Modifications in this basic structure at various sites result in this compounds with different biologic properties, (i.e, duration of action, relative anti-inflammatory activity, sodium retaining activity and transcorneal penetration).

DIFLUPREDNATE OPHTHALMIC EMULSION 0.5%

Structure



Mechanism of Action:

Corticosteroids have both anti-inflammatory and immunosuppressive effects that are non- specific. Abelson MB, Butrus S (1994) and Friedlander MH (1983) have described many different mechanisms by which corticosteroids mediate their anti-inflammatory and immuno-suppressive effects. These mechanisms may be summarized as follows;[29]

- Induction of lymphocytopenia.
- Neutrophilic leukocytosis.
- Reduction of circulating eosinophils and monocytes.
- Inhibition of macrophage recruitment with consequent alterations in cell-mediated immune responses (i.e. reduced skin-test reactivity).
- Inhibition of macrophage migration and antigen processing capability.
- Attenuation of bactericidal activity of macrophages and monocytes.
- Stabilization of intracellular lysozomal membranes.
- Stabilization of mast cell and basophil membranes.
- Inhibition of prostaglandin synthesis. Corticosteroids, via a protein called macrocortin, inhibit the enzyme phospholipase A_2 and thus inhibit the conversion of phospholipid to arachidonic acid. Consequently, the synthesis of both prostaglandins (via. the cyclooxygenase pathway) and leukotrienes (via. the lipoxygenase pathway) is prevented.

- Reduction of capillary permeability and suppression of vasodilation in the setting of acute inflammation. As a consequence, transudation of fluid, protein and inflammatory cells into the target site is reduced.
- Suppression of fibroplasia.

A variety of corticosteroid preparations are available for topical use in the treatment of inflammatory ocular disease.

Difluprednate ophthalmic emulsion acts by repressing the gene expression of inflammatory mediators as well as induces phospholipase A2 inhibitory proteins called lipocortins. These proteins control the biosynthesis of inflammatory mediators such as prostaglandins and leukotreines by inhibiting the release of arachidonic acid,the precursor. Corticosteroids inhibit the inflammatory response by inhibiting edema development, fibrin deposition, capillary dilation, leucocyte migration, capillary proliferation, collagen deposition, fibroblast proliferation, and scar formation associated with the inflammatory response.[29]

Absorption

Due to the low dose and rapid metabolism of topically administered difluprednate in ophthalmic tissue, no measurable systemic absorption of difluprednate or its primary active metabolite has been observed.[30]

Intraocular Penetration

Ocular tissues themselves may play an important role in local steroid metabolism and determine, to some degree, the efficacy of a certain topical preparation**.** Polansky JR, Weinres RN (1984) found that the clinical anti-

inflammatory efficacy of topically applied cortisone and prednisone suggest inherent 11- hydroxylase activity in the cornea and perhaps other ocular tissues.

Phosphate preparations, marketed as solutions, are highly water soluble and would be expected to penetrate lipophillic barriers (the corneal epithelium and endothelium) relatively poorly. In contrast, alcohol-based suspensions, particularly acetate suspensions, exhibit biphasic solubility and are theoretically better able to penetrate all corneal layers to reach the anterior chamber. Similarly, the presence or absence of the corneal epithelium affects the intracorneal and intraocular bioavailability of various steroid preparations. The experimental data, however, are not as clear-cut as the theoretic expectations.[30]

More practical considerations may dictate the choice between derivatives of the same steroid base in clinical practice. Acetate suspensions must be shaken adequately in order to distribute insoluble drug particles so that the maximal concentration of steroid is delivered with each dose (approximately 30 shakes). Apt L, Henrick A, Silverman LM (1979) in their study found that poor patient compliance has been demonstrated in individuals who were instructed to shake their suspension eye drops before topical instillation. Hence, there is a good rationale for preferring phosphate solutions that provide more consistent dosage of drug. [30]

Therapeutic Use

Steroids are the most widely used anti-inflammatory and immuno-suppressant drugs in ophthalmology in general and are the mainstay of therapy for patients with uveitis. They may be grouped into three broad therapeutic categories; postoperative inflammatory control, abnormalities of immune regulation, and entities with a combined immune and inflammatory mechanism. A sensible approach to the use of

topical steroids in anterior uveitis is to treat aggressively with a potent agent during the initial stage of inflammation, to reevaluate the patient at frequent intervals, and to taper the drug slowly, as dictated by the clinical response. [30]

Side Effects and Toxicity

The most clinically significant ocular complication of corticosteroid therapy is the development of cataract and secondary glaucoma.[31]

1. Secondary open-angle glaucoma is most likely to follow prolonged topical therapy with potent steroids.

A more pronounced steroid-induced IOP rise is noted in patients with open-angle glaucoma, relatives of patients with glaucoma, diabetics, and high myopes. The rise in IOP may occur as early as 1 week or may be delayed for years after the initiation of therapy, requiring periodic monitoring of all patients taking corticosteroid medications. The exact mechanism for this phenomenon is unclear; however, there is evidence that corticosteroids enhance the deposition of mucopolysaccharide in the trabecular meshwork. In a controlled study of topical steroids in nurses, Linner, E (1959) noted a small but significant increase in intra-ocular pressure without change in outflow facility.

Francois, J (1961) and Goldmann, H (1962) have recorded glaucomatous states after prolonged topical steroid therapy, but these were considered rare occurrences.

Recently Bernstein H. N, and Schwartz B. (1962) found that patients on a long term systematic corticosteroid therapy showed significantly higher mean applanation pressures when compared with non-treated individuals. They also noted lower facilities of outflow and ocular rigidities in steroid-treated patients.

Bernard Becker and Donald W. Mills (1963**)** administered corticosteroid drops 4 times daily for upto 2 months to one eye of (a) Patients with primary open angle glaucoma (b) Glaucoma suspects (c) Normals. They concluded that the glaucoma and the glaucoma-suspect groups demonstrated large and highly significant increases in intra-ocular pressure and striking decreases in facility of outflow in the steroid-treated eyes without significant changes in their opposite controlled eyes. In the volunteer group, a surprising number of steroid-treated eyes (30%) developed elevations of intra-ocular pressure to 21mmHg or higher, the pressure elevations was significant and were associated with significant decreases in the coefficients of aqueous outflow facility.[31]

2. Posterior subcapsular cataracts (PSCs) arise in a dose and duration-dependent manner after long-term corticosteroid therapy, although individual susceptibility appears to vary.

   Rubin B, Palestine AG (1989) in their study found that the mechanism of corticosteroid-induced cataract formation is believed to involve the binding of glucocorticoids to lens fibers, leading to biochemical alterations, with protein aggregation in the cells and a change in the refractive index.[31]

3. Susceptibility to microbial infections is enhanced by the corticosteroids because these agents suppress the inflammatory response. Herpetic, bacterial (particularly pseudomonal), and fungal keratitis may be potentiated by corticosteroid therapy without the concomitant use of the appropriate antiviral or antibiotic. Likewise, posterior segment inflammatory conditions, such as ocular syphilis, tuberculosis, and toxoplasmosis always should be treated with appropriate anti-infective agents before the introduction of corticosteroids.

4. Inhibition of corneal epithelial and stromal healing occurs with all corticosteroids, with the possible exception of medroxyprogesterone. Manifestations may be as trivial as superficial punctate staining of the cornea to relentless corneal-scleral melting and perforation. Corticosteroids retard collagen synthesis by fibroblasts and enhance collagenase activity. Cognizance of the effects of steroids on wound healing is particularly important in the presence of corneal-scleral ulceration or thinning, minor trauma, and during the postoperative period.

5. Mild mydriasis and ptosis are more often seen with topical steroid therapy as seen in a study by Armaly MF in 1963.

6. Krupin T, LeBlanc RP, Becker B et al (1970) found that paradoxic anterior uveitis induced by the corticosteroid itself rather than the vehicle may follow topical therapy. It has been suggested that the development of corticosteroid induced uveitis may be related to an activation of latent spirochetes in the eye, although there is no direct proof to substantiate this.

7. Other side effects of topical steroid therapy such as blurred vision and punctate keratopathy may relate to ocular irritation arising from mechanical effects of the steroid particles in suspension, allergy to the vehicle, or the underlying inflammatory condition. In addition, refractive changes, paralysis of accommodation, and altered corneal thickness has been described. Central serous retinopathy has been reported in association with systemic steroid therapy, and pseudotumor cerebri, especially in children, may occur after abrupt cessation or reduction of therapy.

A study was to done to assess the efficacy of Difluprednate 0.05% ophthalmic emulsion to a placebo in the treatment of inflammation associated with ocular surgery. The result was a greater proportion of Difluprednate-treated patients had a reduction in inflammation at 8 days and 15 days compared to placebo. The anterior chamber cell grade zero on day 8 of therapy occurred in 34.5% of patients in Difluprednate group versus 12.4% in placebo group. Difluprednate 0.05% Ophthalmic Emulsion safely clears post-operative inflammation with no serious adverse effects, fewer adverse affects were noted in Difluprednate treated groups than the placebo group[32].

The efficacy of Difluprednate 0.05% Ophthalmic Emulsion was also studied and proved in treatment of refractory macular edema after vitrectomy.

Another comparative study was done to know the dose uniformity of prednisolone Acetate Ophthalmic suspension to Difluprednate 0.05% Ophthalmic Emulsiuon, result was Difluprednate Ophthalmic emulsion exhibited excellent dose uniformity under all conditions studied[32].

PREDNISOLONE ACETATE 1% OPHTHALMIC SUSPENSION

Prednisolone acetate 1% ophthalmic suspension, USP is a topical anti-inflammatory agent for ophthalmic use.[33]

Chemical Name:

11ß,17, 21-Trihydroxypregna-1,4-diene-3, 20-dione 21-acetate

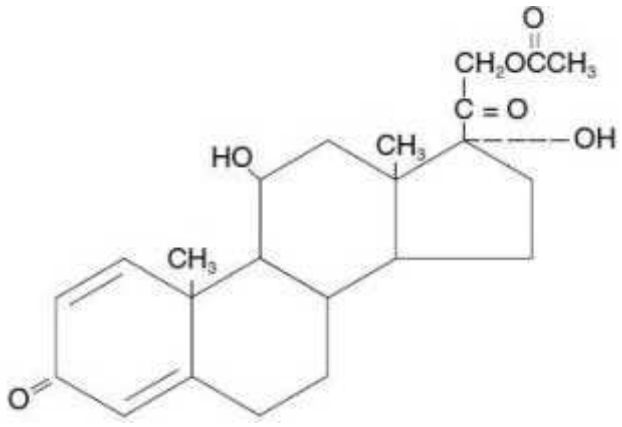
Contains: Active: prednisolone acetate (microfine suspension) 1.0%.

Preservative: benzalkonium chloride.

Inactives: boric acid; edetate disodium; hypromellose; polysorbate 80; purified water; sodium bisulfite; sodium chloride; and sodium citrate.

The pH during its shelf life ranges from 5.0 - 6.0.

Structure



Prednisolone acetate

Mechanism of Action:

Glucocorticoids inhibit the edema, fibrin deposition, capillary dilation, and phagocytic migration of the acute inflammatory response, as well as capillary proliferation, deposition of collagen, and scar formation.[33]

Therapeutic Use

Prednisolone acetate 1% ophthalmic suspension is indicated for the treatment of steroid-responsive inflammation of the palpebral and bulbar conjunctiva, cornea, and anterior segment of the globe.[33]

Side Effects

Corticosteroid-containing preparations have also been reported to cause acute anterior uveitis and perforation of the globe. Keratitis, conjunctivitis, corneal ulcers, mydriasis, conjunctival hyperemia, loss of accommodation and ptosis have occasionally been reported following local use of corticosteroids.[33]

MATERIALS AND METHODS

The present study was a comparative study to compare the safety and efficacy of Topical Difluprednate Ophthalmic Emulsion 0.05% eye Drops with Topical Prednisolone Acetate Ophthalmic Suspension 0.1% eye drops in the control of postoperative inflammation after cataract surgery.

A total of hundred patients diagnosed as senile cataracts coming to KLES Dr. Phabhakar Kore Hospital Belgaum over a one year period (Jan 2011-Jan 2012) who fulfilled the inclusion criteria were enrolled in the study.

Sample Size

A total of hundred patients with senile cataract were selected for the study. They were divided into two groups.

Group A- 50 cases (Difluprednate Ophthalmic Emulsion 0.05% group)

Group B- 50 cases (Prednisolone Acetate Ophthalmic Suspension 1% group)

The inclusion and exclusion criteria were as follows:

INCLUSION CRITERIA:

1. Patients between 50 to 80 years of age
2. Patients undergoing cataract surgery (SICS) with IOL implantation.

EXCLUSION CRITERIA:

1. Patients sensitive to any of the study or procedural medicines
2. Patients with preoperative inflammation in either eye.
3. Patients with H/O ocular trauma, previous intraocular surgery or wear of contact lens.

4. Patients with H/O uncontrolled or chronic ocular (glaucoma, uveitis, pseudoexfoliation) or systemic disease (hypertension, Diabetes).
5. Patients developing intraoperative complications

STUDY DESIGN- Randomized controlled study.

The patients were assessed preoperatively, a complete history was taken and they underwent a detailed slit lamp examination, assessment of visual acuity and fundoscopy. The details were entered in the clinical proforma. The investigations done were measurement of intraocular pressure (Schiotz), tests for lacrimal patency, urine for albumin and sugar and Blood Pressure. The local human experimentation ethics committee approved the study and informed consent was obtained from all patients.

The intraocular surgery was a conventional SICS with PCIOL implantation done by experienced surgeons. The pupil was dilated with a combination of 1% cyclopentolate hydrochloride and 10 % phenylephrine hydrochloride eyedrops. Ciprofloxacin eye drops were administered hourly preoperatively. Systemic antibiotics like tablet Ciprofloxacin 500mg BD and i.v. Cefotaxime 1gm BD was given. Peribulbar anesthesia was administered using 2% lignocaine, adrenaline and hyaluronidase. The procedure consisted of corneoscleral incision, canopener type of anterior capsulotomy, nucleus delivery and irrigation aspiration of cortical matter, and insertion of polymethymethaacrylate (PMMA) IOL. Viscoelastic substance used was hydroxypropylmethycellulose. The irrigating solution used was normal saline. Surgery was uncomplicated in all cases. The study medications topical Difluprednate Ophthalmic Emulsion (0.05%) or Prednisolone Acetate Ophthalmic Suspension (0.1%) was administered in a randomized fashion.

Methodology:

After the randomization procedure the patients were assessed pre-operatively by the investigator for a complete history and a detailed slit-lamp examination, assessment of visual acuity and fundoscopy and manual small incision cataract surgery.

The details were entered in a pretested questionnaire.

Investigation done :

1. Measurement of intra –ocular pressure
2. Tests for lacrimal patency
3. Urine for albumin and sugar
4. Blood pressure.

All the patients received the study medication as allotted. Patients in Group A received Difluprednate eye drops 6 times and Gatifloxacin eyedrops 4 times in a day.. Patients in Group B received Prednisolone eyedrops 6 times and Gatifloxacin eyedrops 4 times in a day. Cyclopentolate eye drops 2 times was given to both the groups.

The patients were instructed clearly regarding administration of the drops. They were told to lie down or with head tilted back, to form a conjunctival pouch and to instill the first drug without the dropper touching the eye area and then close the eye gently (without blinking, rubbing, squeezing). They were the instructed to apply pressure over the lacrimal puncta for one minute. They were told to administer the next drug after a gap of 5 minutes.

The patients were examined postoperatively on days 1, 7, 15 and 30. At each visit, symptoms like pain, watering and any other experienced by the patient was noted. Visual Acuity was assessed by Snellen's chart and a slit lamp examination was done for evaluation of inflammation. All Slit lamp examinations were conducted under standard conditions: dim room illumination, highest lamp voltage, 3x1 millimeter aperture for Anterior chamber Flare and Cells, illumination angle of 30 degrees and magnification of 16x. The following are the parameters that were recorded at each visit. Each parameter was graded as 0, 1, 2, and 3 as follows.

Lid edema:

Grade 0 - None

Grade 1 - Mild

Grade 2 - Moderate

Grade 3 - Severe

Conjunctival congestion:

Grade 0 - None

Grade 1 - Mild (some vessels injected)

Grade 2 - Moderate (diffuse injection)

Grade 3 - Severe (intense injection)

Ciliary congestion:

Grade 0 - None

Grade 1 - Mild (some vessels injected)

Grade 2 - Moderate (diffuse injection)

Grade 3 - Severe (intense injection)

Corneal edema:

Grade 0 - None

Grade 1 - Mild

Grade 2 - Moderate

Grade 3 - Severe

Presence of deep striate keratitis or keratic precipitates was also noted.

Anterior Chamber Flare:

Grade 0 - Absent

Grade 1 - Mild (barely detected)

Grade 2 - Moderate (iris and lens details seen)

Grade 3 - Severe (iris and lens details not seen)

Anterior Chamber Cells:

Grade 0 - Absent

Grade 1 - 5 to 10cells

Grade 2 - 11 to 20 cells

Grade 3 - 20 to 50 cells

If 50 cells and hypopyon was graded as 4.

Pupils were examined for any synechiae or any other abnormalities.

Direct Ophthalmoscopy was done and **Vitreous Haze** was graded as follows; Grade 0- Absent, Grade 1-Few scattered fine and coarse opacities (Fundus seen clearly), Grade2-Scattered fine and coarse opacities but fundus somewhat obscured, Grade3- many opacities with marked blurring of the fundus, and denser opacities which prevented viewing of the fundus was graded as 4.

The details of the fundus were also noted especially in the macula for the presence of cystoid macular edema.

The **total scoring** of all the parameters was then done at each visit and graded as follows-

None	-	0
Mild	-	1-3
Moderate	-	4-7
Severe	-	8 and above

At the end of six weeks, refraction was done to get the best-corrected visual acuity and intraocular pressure was measured using an Air Puff Tonometer. All the above details were recorded in the clinical proforma at each visit.

Data Analysis:

Statistical analysis was done using Student's 't' test, chi square test and paired t test.

Limitations of the study:

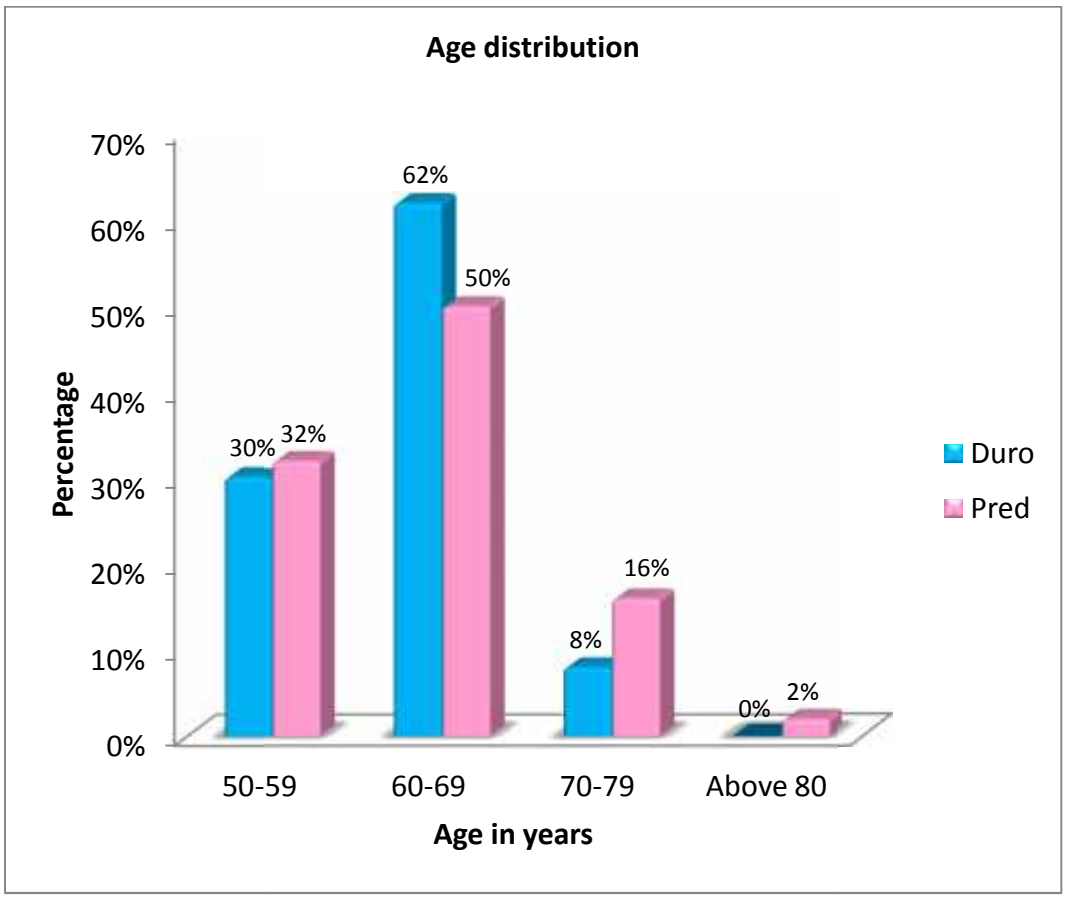
The evaluation of postoperative inflammation in this study was done using a slit lamp, which is subjective. It should have ideally been done using laser flare cell photometers, which could provide objective information but unfortunately, are not readily available.

RESULTS

TABLE 1 - AGE DISTRIBUTION

Age group in years	Difluprednate		Prednisolone	
	No	%	No	%
50-59	15	30	16	32
60-69	31	62	25	50
70-79	4	8	8	16
Above 80	0	0	1	2
Total	**50**	**100**	**50**	**100**

(p=0.272)

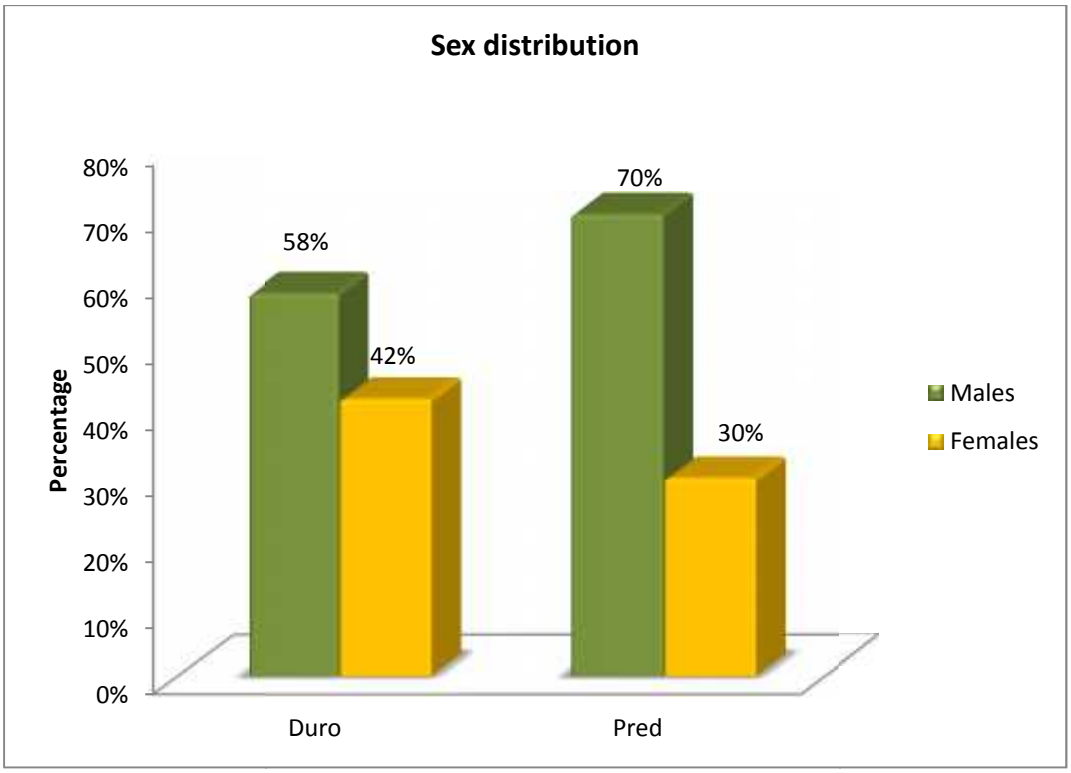


Total number of patients in age group of 50 to 59 years in Difluprednate group was 15 (30%) and in Prednisolone group was 16 (32%). Number of patients in the age group of 60 to 69 years in Difluprednate group was 31 (62%) and number of patients in Prednisolone group was 25(50%). Maximum number of patients were in this age group. There were 4 patients (8%) and 8 patients (16%) in Difluprednate and Prednisolone group respectively in the age group of 70-79 years. Only one patient of 80 years of age was in Prednisolone group. The mean age in Difluprednate group was 59.04 years and in Prednisolone group was 61.04

TABLE 2 - SEX DISTRIBUTION

Sex	Difluprednate		Prednisolone	
	No	%	No	%
Males	29	58	35	70
Females	21	42	15	30
Total	50	100	50	100

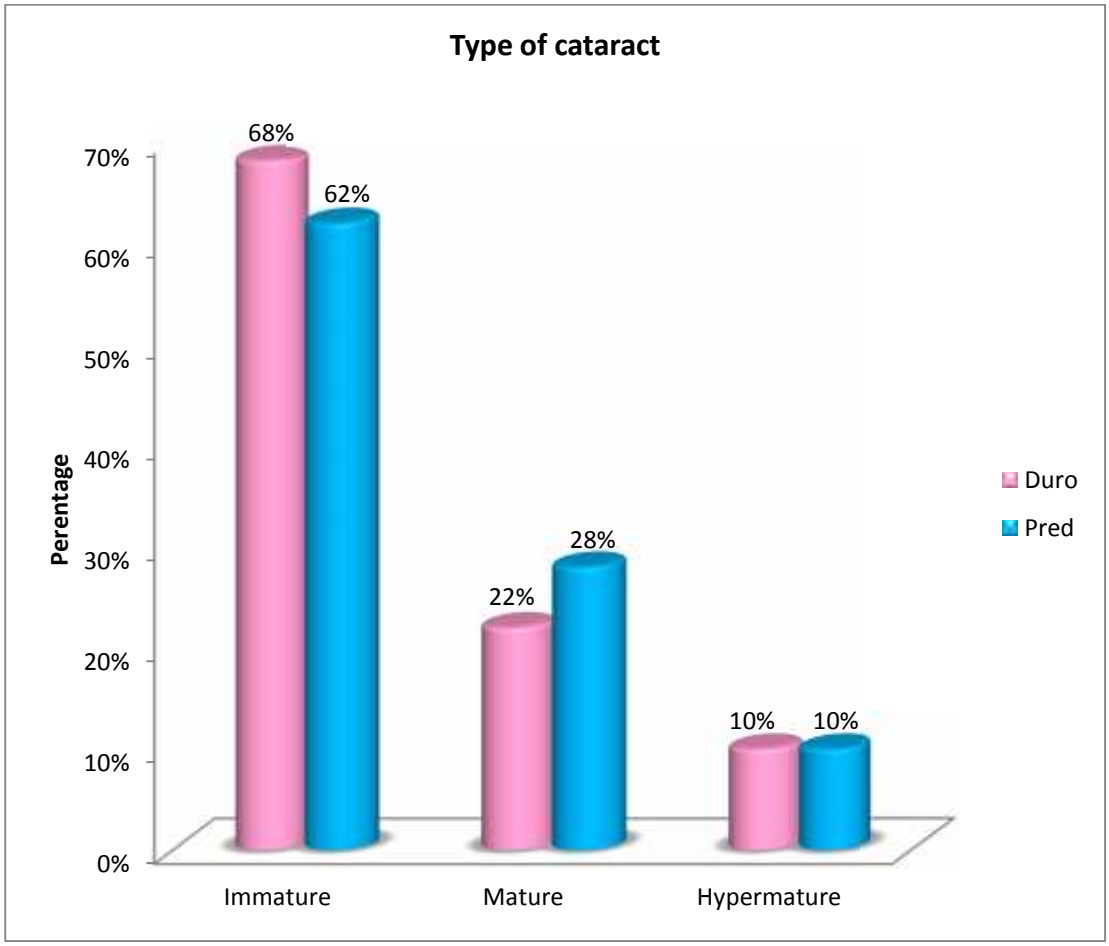
(p=0.562)



The number of males in Difluprednate group were 29(58%) and in Prednisolone group were 35(70%). Twenty one patients (42%) were females in Difluprednate group as compared with 15 patients (30%) in Diclofenac group.

TABLE 3 - TYPE OF CATARACT

Type of cataract	Difluprednate		Prednisolone	
	No	%	No	%
Immature	34	68	31	62
Mature	11	22	14	28
Hypermature	5	10	5	10
Total	50	100	50	100

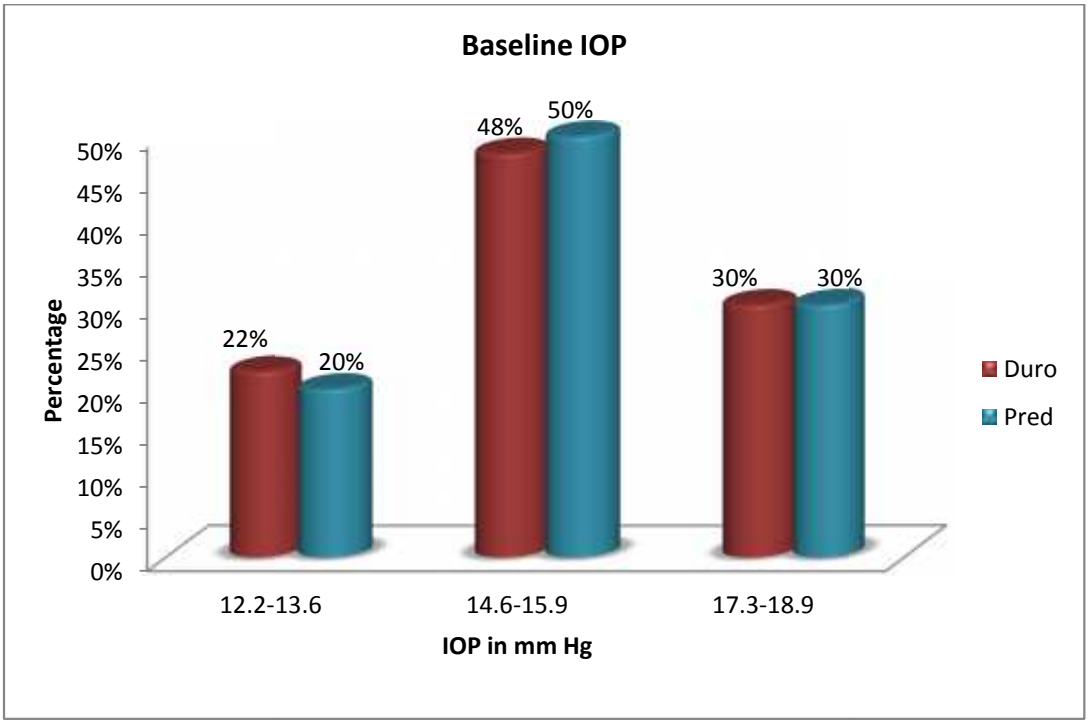


There were 34 patients (68%) in Difluprednate group and 31 patients (62%) in Prednisolone group with immature cataract. Majority of the patients were in this group. Eleven patients (22%) in Difluprednate group and 14 patients (28%) in Prednisolone group had mature cataract. The number of patients with hypermature cataracts was 5 patients (10%) in Difluprednate group and 5 patients (10%) in Prednisolone group.

TABLE 4 - BASELINE INTRAOCULAR PRESSURE

IOP	Difluprednate		Prednisolone	
	No	%	No	%
12.2 - 13.4	11	22	10	20
14.6 - 15.9	24	48	25	50
17.3-18.9	15	30	15	30
Total	50	100	50	100

(p=0.967)



As seen in Table 4, the number of patients in the range of 12.2 to 13.4 mmHg was 11 patients (22%) in Difluprednate group and 10 patients (20%) in Prednisolone group. 24 patients (48%) in Difluprednate group and 25 patients (50%) in Prednisolone group were in the range of14.6 to 15.9 mmHg. Fifteen patients (30%) were in range of 17.3 to 18.9 mmHg in Difluprednate group as compared with 15 patients (30%) of Prednisolone group.

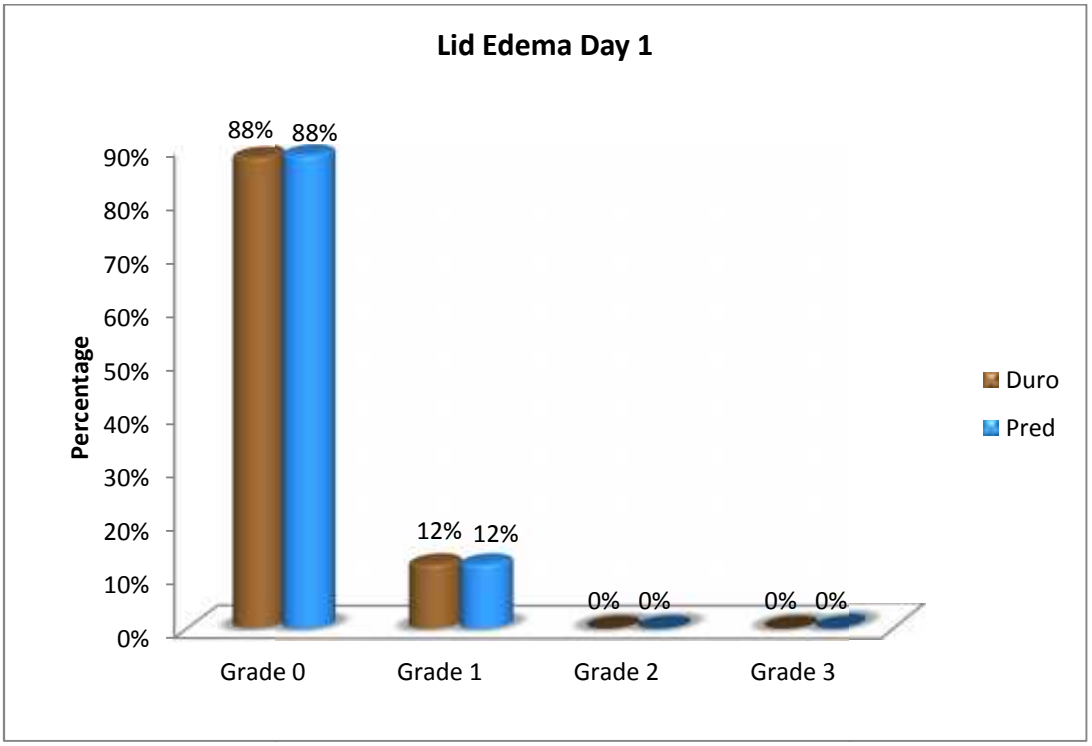
GRADES OF POSTOPERATIVE INFLAMMATION

TABLE 5 - LID EDEMA

Day 1

	Lid Edema	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 1	Grade 0 (None)	44	88	44	88
	Grade 1 (Mild)	6	12	6	12
	Grade 2 (Moderate)	0	0	0	0
	Grade 3 (Severe)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

(p=1.0)



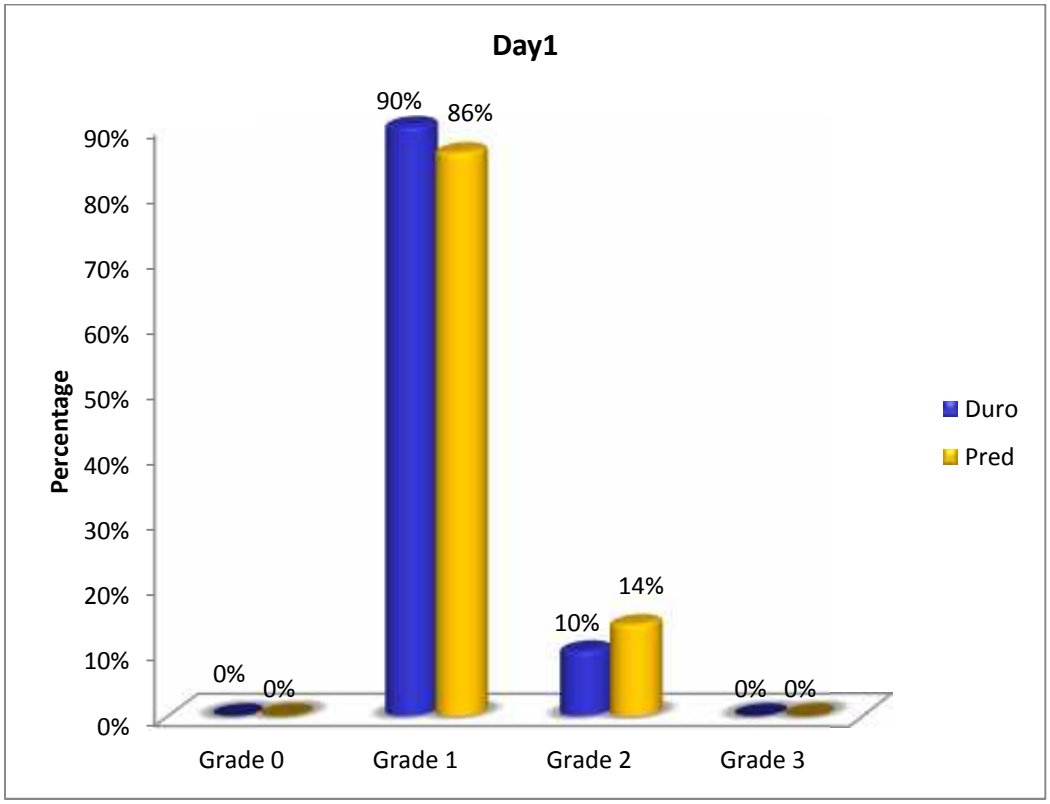
On day 1, only 6 patients (12%) in Difluprednate group and 6 patients (12%) in Prednisolone group had Grade1 lid edema.

TABLE 6 - CONJUNCTIVAL CONGESTION

Day 1

	Conjunctival congestion	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 1	Grade 0 (None)	0	0	0	0
	Grade 1 Mild (some vessels injected)	45	90	43	86
	Grade 2 Moderate (diffuse injection)	5	10	7	14
	Grade 3 Severe (intense injection)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

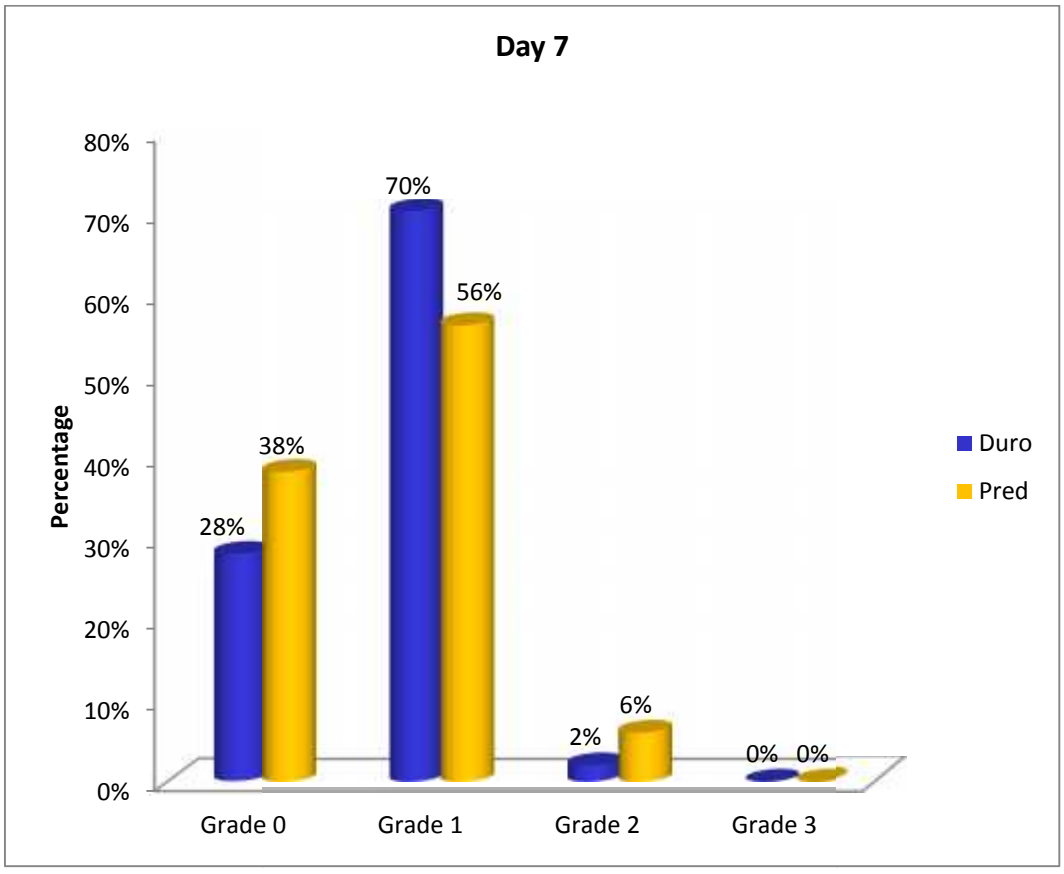
(p=0.758)



Day 7

	Conjunctival congestion	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 7	Grade 0 (None)	14	28	19	38
	Grade 1 Mild (some vessels injected)	35	70	28	56
	Grade 2 Moderate (diffuse injection)	1	2	3	6
	Grade 3 Severe (intense injection)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

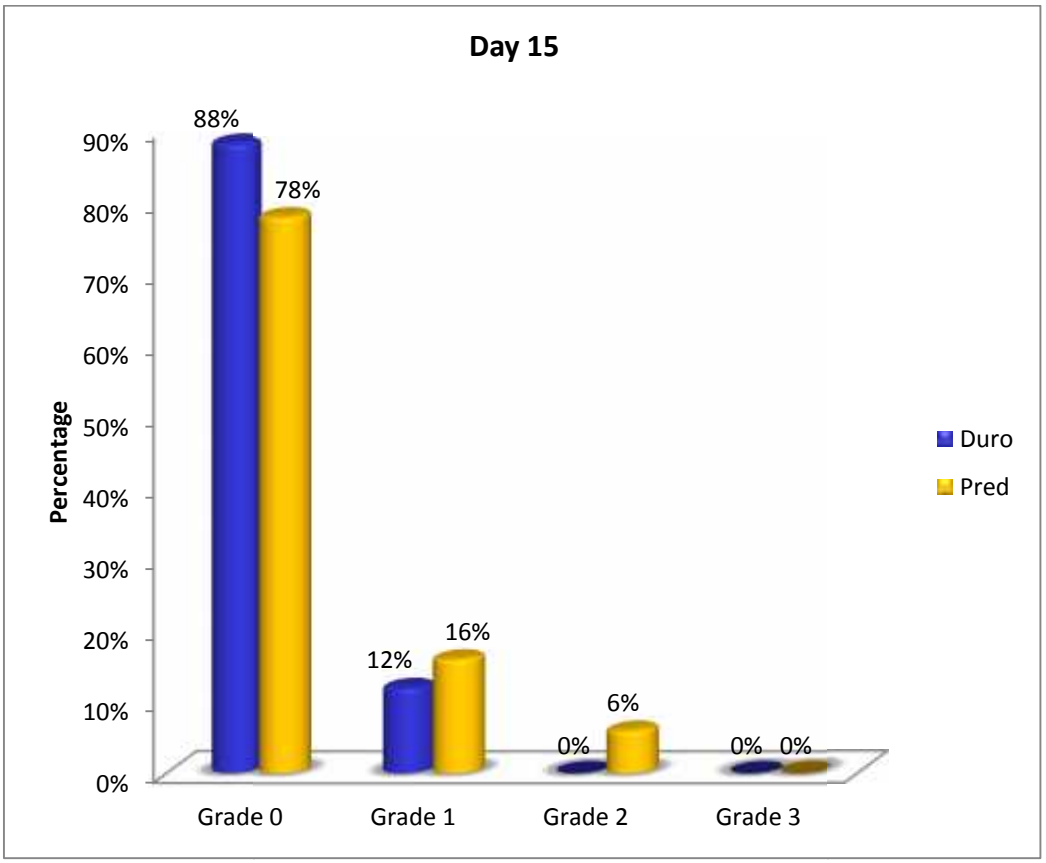
(p=0.395)



Day 15

	Conjunctival congestion	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 15	Grade 0 (None)	44	88	39	78
	Grade 1 Mild (some vessels injected)	6	12	8	16
	Grade 2 Moderate (diffuse injection)	0	0	3	6
	Grade 3 Severe (intense injection)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

(p=0.287)



Maximum number of patients had Grade 1 conjunctival congestion on Day 1. Out of which there were 45 patients (90%) in Difluprednate group and 43 patients (86%) in Prednisolone group. The numbers of patients with grade 2 were 5(10%) in Difluprednate and 7 (14%) in Prednisolone group.

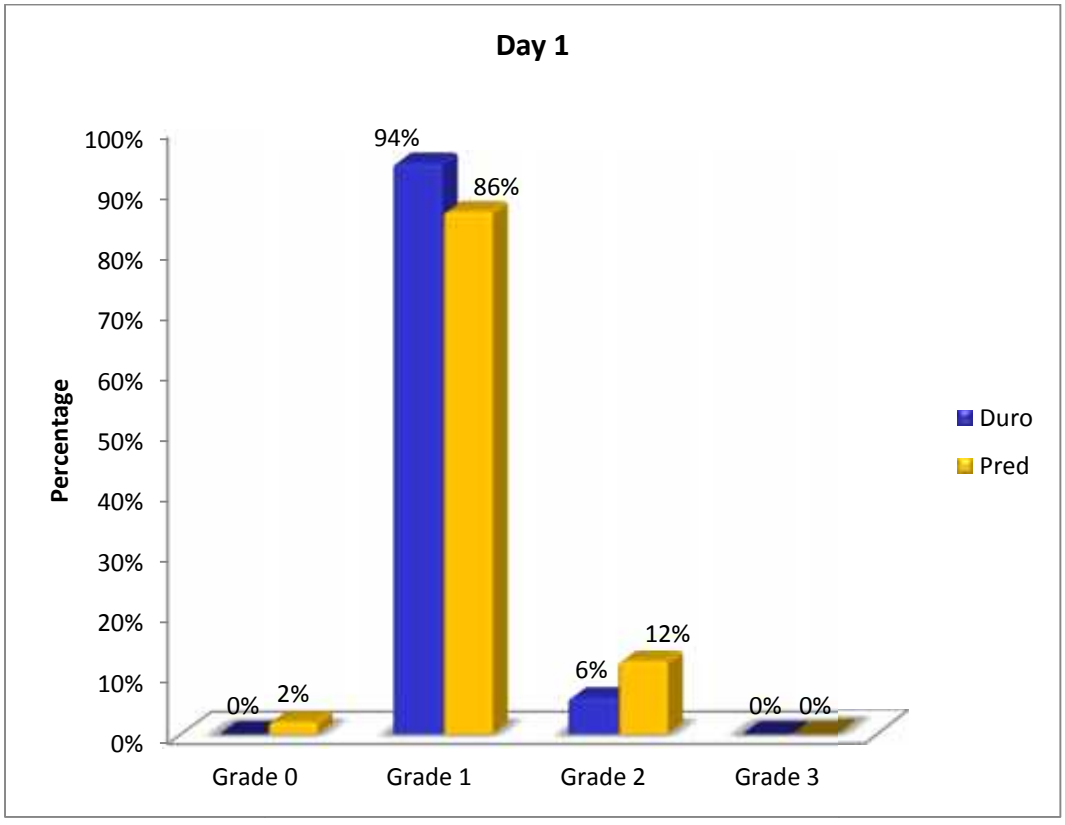
By Day 7, the number of patients with Grade 0 was 14(28%) in Difluprednate group and 19(38%) in Prednisolone group. The total number of patients with Grade 1 had come down to 35 patients (70%) in Difluprednate group and 28 patients (56%) in Prednisolone group.1 patient (2%) in Difluprednate group and 3 patients (6%) in Prednisolone group showed Grade 2 conjunctival congestion. Six patients (12%) in Difluprednate group still had Grade 1 as compared with 8 patient (16%) of Prednisolone group on day 15. Three patients (6%) in Prednisolone group stil had Grade 2 conjunctival congestion.

TABLE 7 - CILIARY CONGESTION

DAY 1

	Ciliary congestion	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 1	Grade 0 (None)	0	0	1	2
	Grade 1 Mild (some vessels injected)	47	94	43	86
	Grade 2 Moderate (diffuse injection)	3	6	6	12
	Grade 3 Severe (intense injection)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

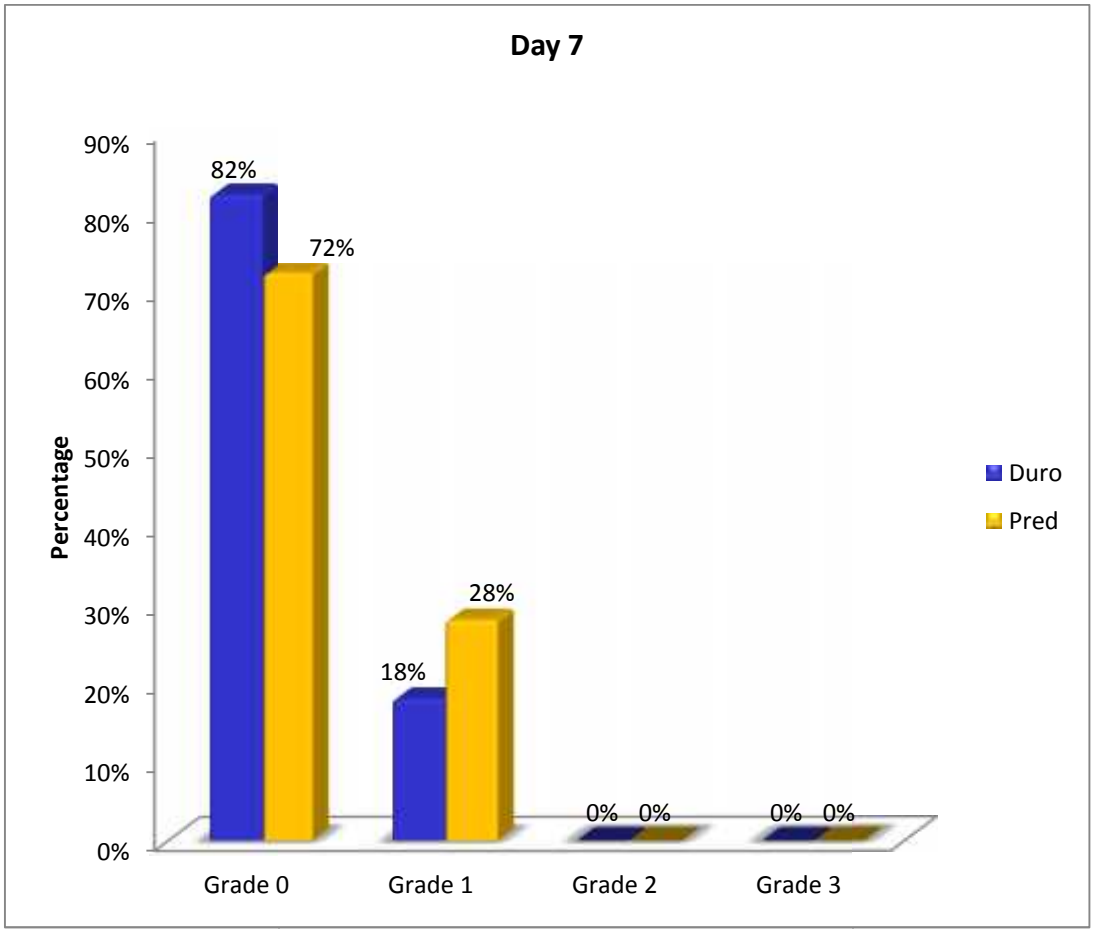
(p=0.318)



Day 7

	Ciliary congestion	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 7	Grade 0 (None)	41	82	36	72
	Grade 1 Mild (some vessels injected)	9	18	14	28
	Grade 2 Moderate (diffuse injection)	0	0	0	0
	Grade 3 Severe (intense injection)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

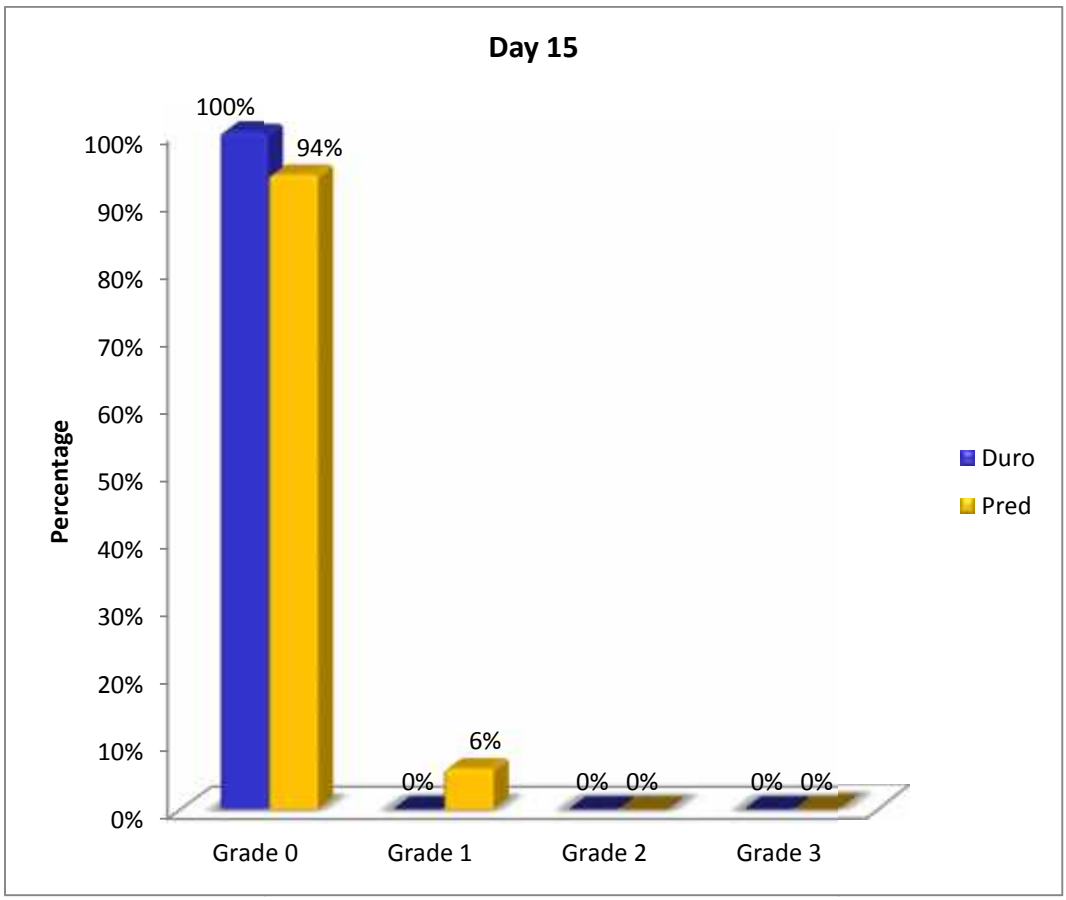
(p=0.342)



Day 15

	Ciliary congestion	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 15	Grade 0 (None)	50	100	47	94
	Grade 1 Mild (some vessels injected)	0	0	3	6
	Grade 2 Moderate (diffuse injection)	0	0	0	0
	Grade 3 Severe (intense injection)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

(p=0.242)



Maximum number of patients with ciliary congestion on Day 1 were of Grade 1 out of which 47 patients (94%) were of Difluprednate group and 43 patients (86%) were of Prednisolone group. Three patients (6%) in Difluprednate and 6 patients (12%) in Prednisolone group showed Grade 2 ciliary congestion.

Nine patients (18%) in Difluprednate group had Grade 1 as compared with 14 patients (28%) in Prednisolone group by Day 7.

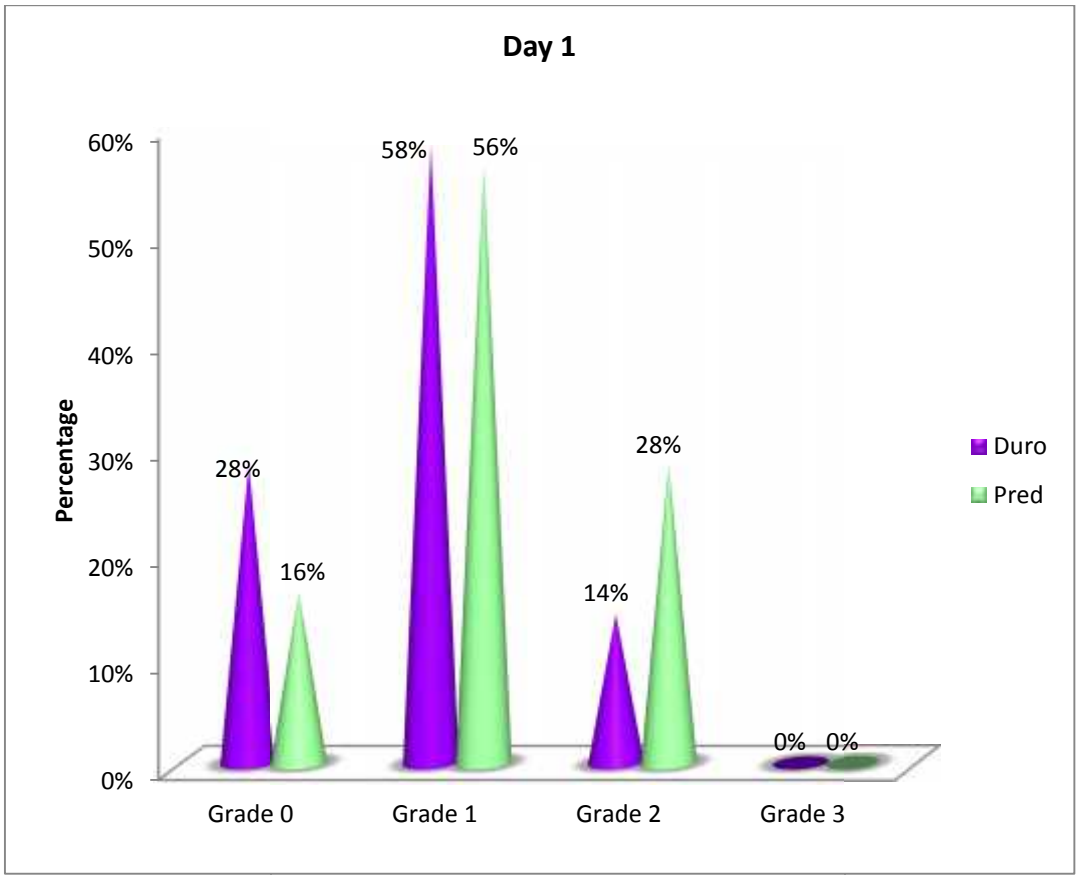
As of Day 15 none of the patients in Difluprednate group had ciliary congestion but 3 patients (6%) still had Grade 1 in Prednisolone group.

TABLE 8 - CORNEAL EDEMA

Day 1

	Corneal edema	**Difluprednate**		**Prednisolone**	
	Grades	**No**	**%**	**No**	**%**
Day 1	Grade 0 (None)	14	28	8	16
	Grade 1 (Mild)	29	58	28	56
	Grade 2 (Moderate)	7	14	14	28
	Grade 3 (Severe)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

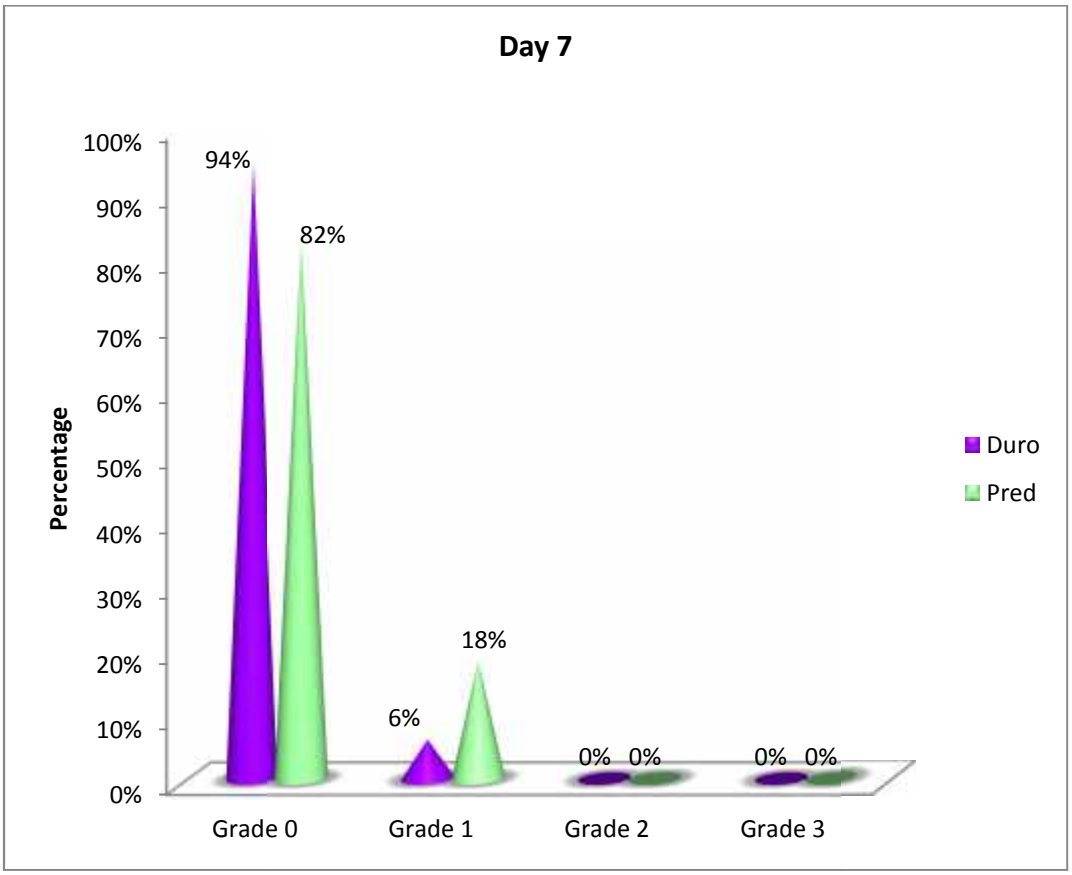
(p = 0.136)



Day 7

	Corneal edema	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 7	Grade 0 (None)	47	94	41	82
	Grade 1 (Mild)	3	6	9	18
	Grade 2 (Moderate)	0	0	0	0
	Grade 3 (Severe)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

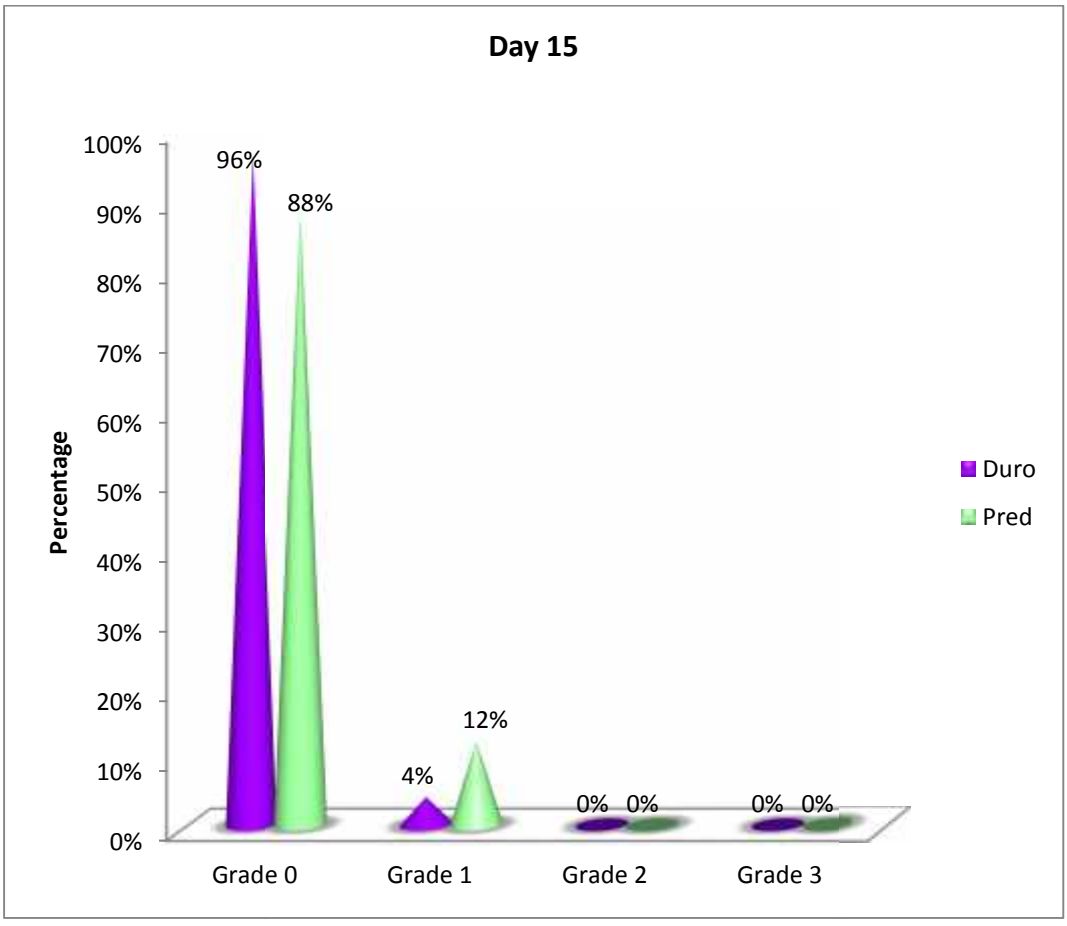
(p = 0.121)



Day 15

	Corneal edema	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 15	Grade 0 (None)	48	96	44	88
	Grade 1 (Mild)	2	4	6	12
	Grade 2 (Moderate)	0	0	0	0
	Grade 3 (Severe)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

(p = 0.269)



On Day 1, 29 patients (58%) in Difluprednate group and 28 patients (56%) in Prednisolone group had mild (grade1) corneal edema. Seven patients (14%) in Difluprednate group and 14 patients (28%) in Prednisolone group had Grade 2 corneal edema.

Three patients (6%) in Difluprednate group and 9 patients (18%) in Prednisolone group had Grade 1corneal edema on day 7.

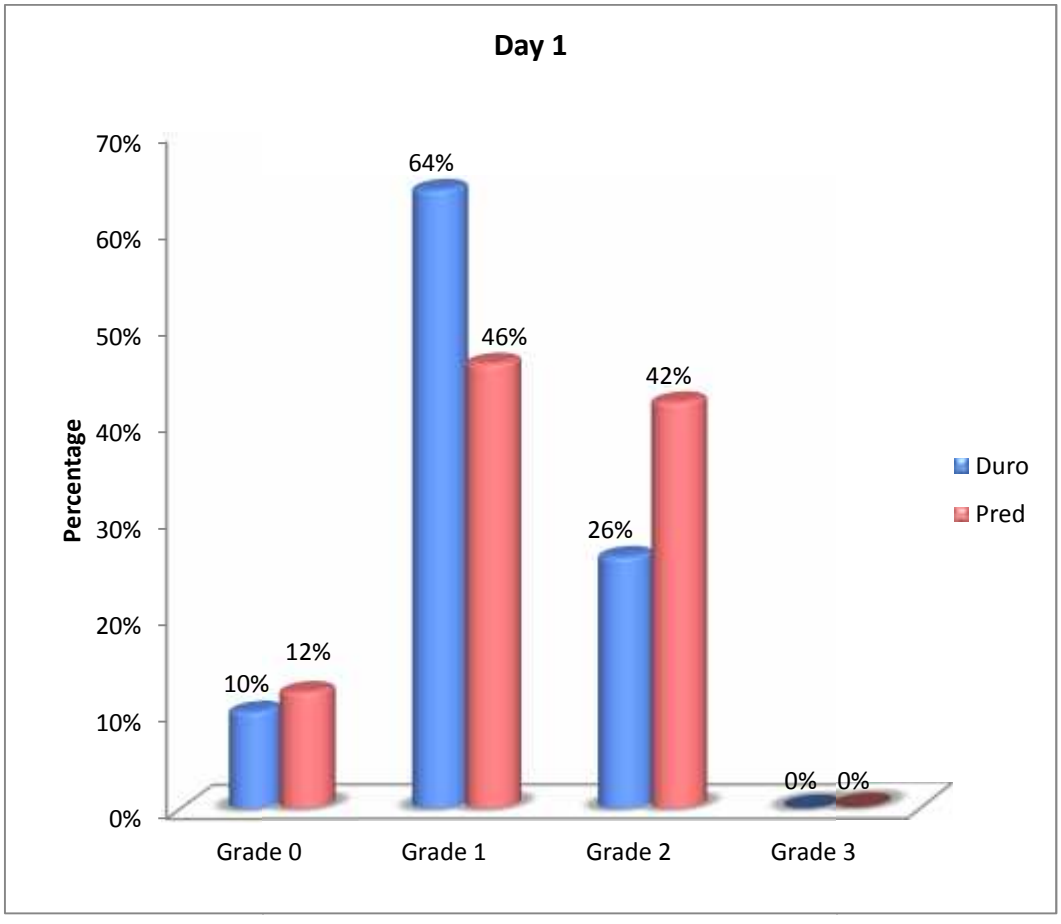
On day 15, only 2 patients (4%) in Difluprednate group and 6 patients (12%) in Prednisolone group had Grade 1 corneal edema.

TABLE 9 - ANTERIOR CHAMBER FLARE

Day 1

	Anterior chamber flare	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 1	Grade 0 (Absent)	5	10	6	12
	Grade 1 Mild (barely detected)	32	64	23	46
	Grade 2 Moderate (iris & lens details seen)	13	26	21	42
	Grade 3 Severe (Iris & lens details not seen)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

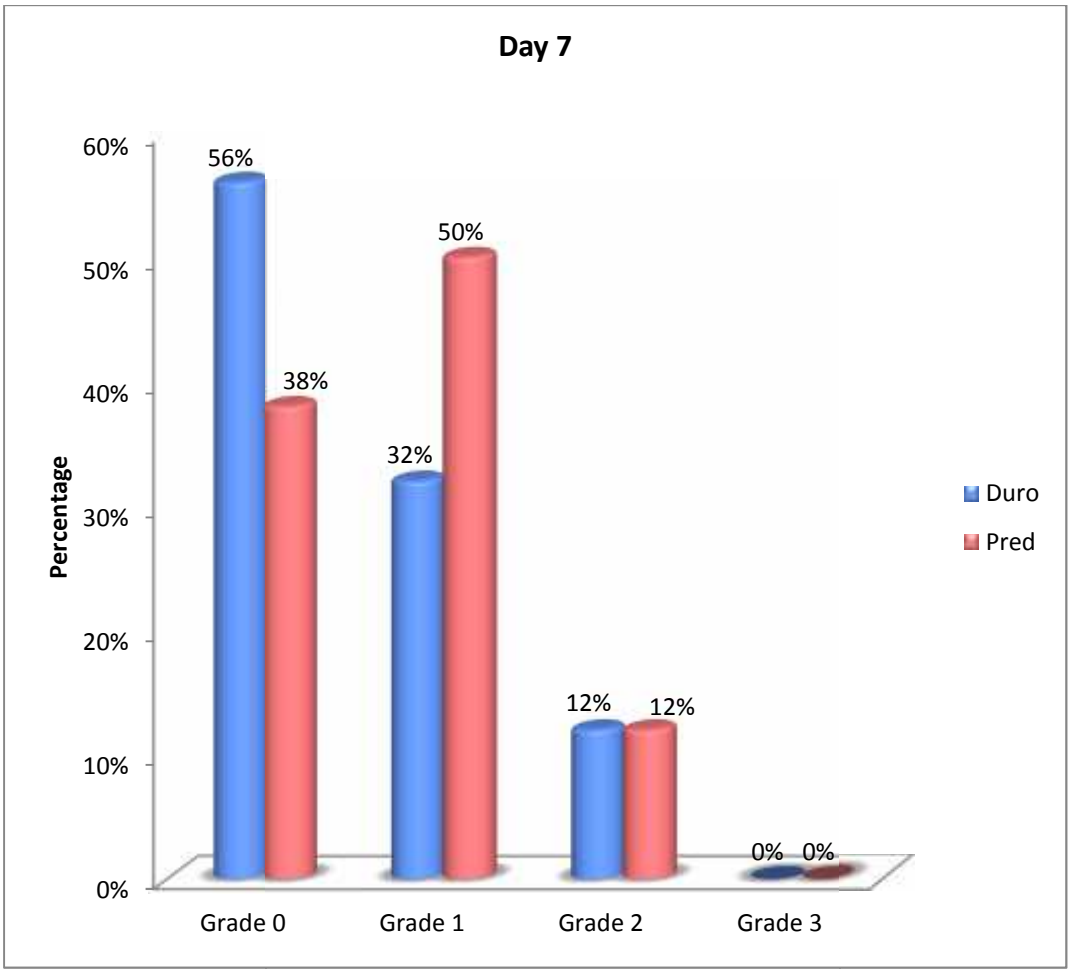
(p = 0.178)



Day 7

	Anterior chamber flare	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 7	Grade 0 (Absent)	28	56	19	38
	Grade 1 Mild (barely detected)	16	32	25	50
	Grade 2 Moderate (iris & lens details seen)	6	12	6	12
	Grade 3 Severe (Iris & lens details not seen)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

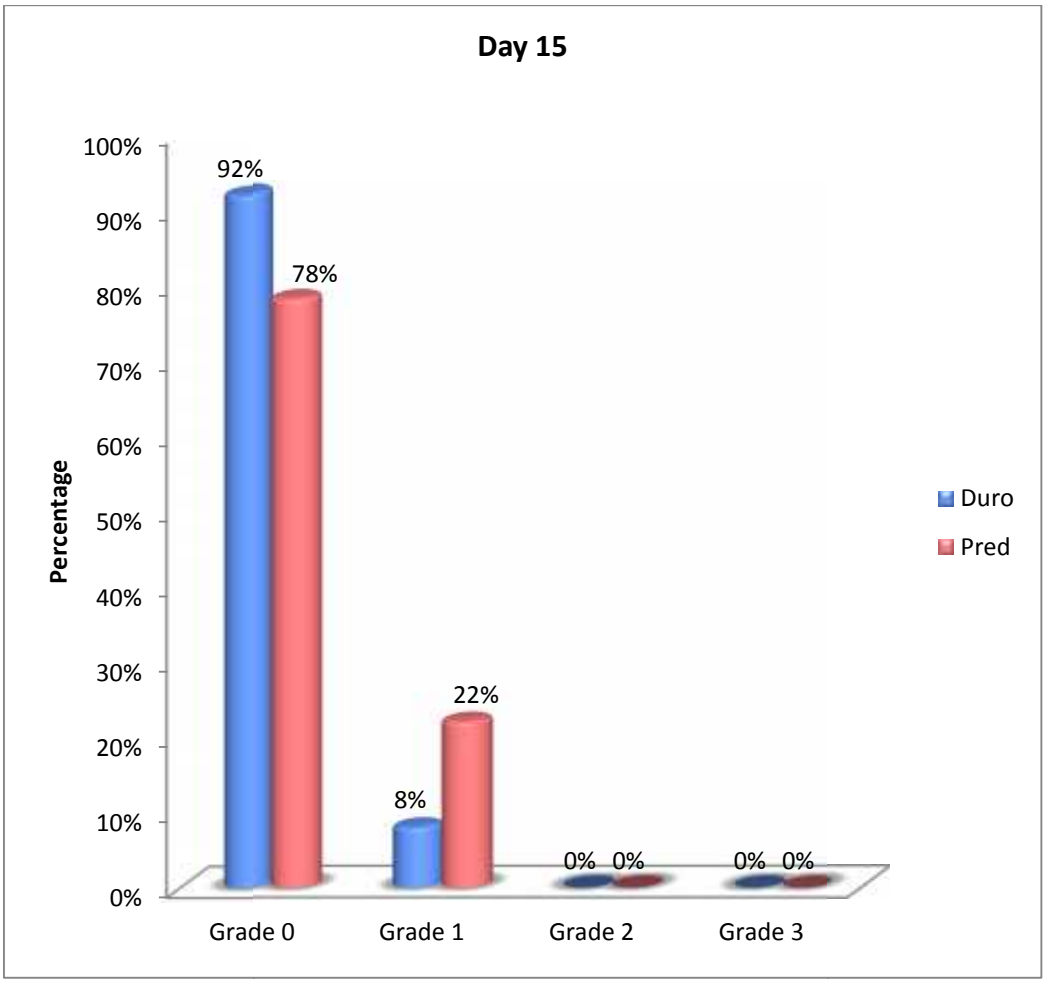
(p = 0.157)



Day 15

	Anterior chamber flare	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 15	Grade 0 (Absent)	46	92	39	78
	Grade 1 Mild (barely detected)	4	8	11	22
	Grade 2 Moderate (iris & lens details seen)	0	0	0	0
	Grade 3 Severe (Iris & lens details not seen)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

(p = 0.091)



The number of patients with Grade 1 Anterior Chamber Flare on Day 1 was 32 (64%) in Difluprednate group and 23(46%) in Prednisolone group.Thirteen patients (26%) of Difluprednate group and 21 patient (42%) of Prednisolone group had Grade 2 Flare

By Day 7, in Difluprednate group, 16 patients (32%) had Grade 1 Flare, where as 25 patients (50%) of Prednisolone group had Grade 1 Flare. Six patients (12%) and 6 patients (12%) in Difluprednate and Prednisolone group respectively had Grade 2 Flare.

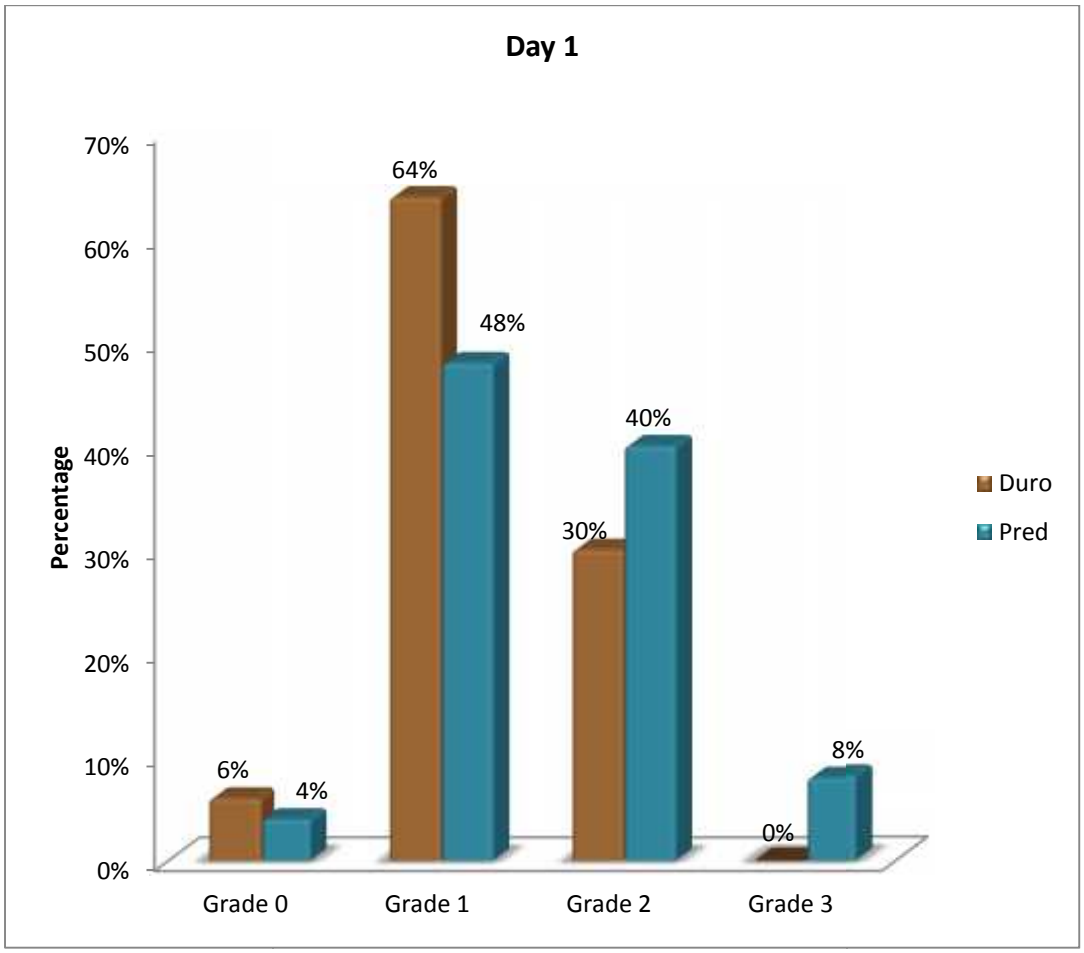
On Day 15, four patients (8%) in Difluprednate group and 11 patients (22%) in Prednisolone group had Grade 1 flare.

TABLE 10 - ANTERIOR CHAMBER CELLS

Day 1

	Anterior chamber cells	Difluprednate		Prednisolone	
		No	**%**	**No**	**%**
Day 1	Grade 0 (Absent)	3	6	2	4
	Grade 1 5-10 cells	32	64	24	48
	Grade 2 11-20 cells	15	30	20	40
	Grade 3 21-50 cells	0	0	4	8
	Total	**50**	**100**	**50**	**100**

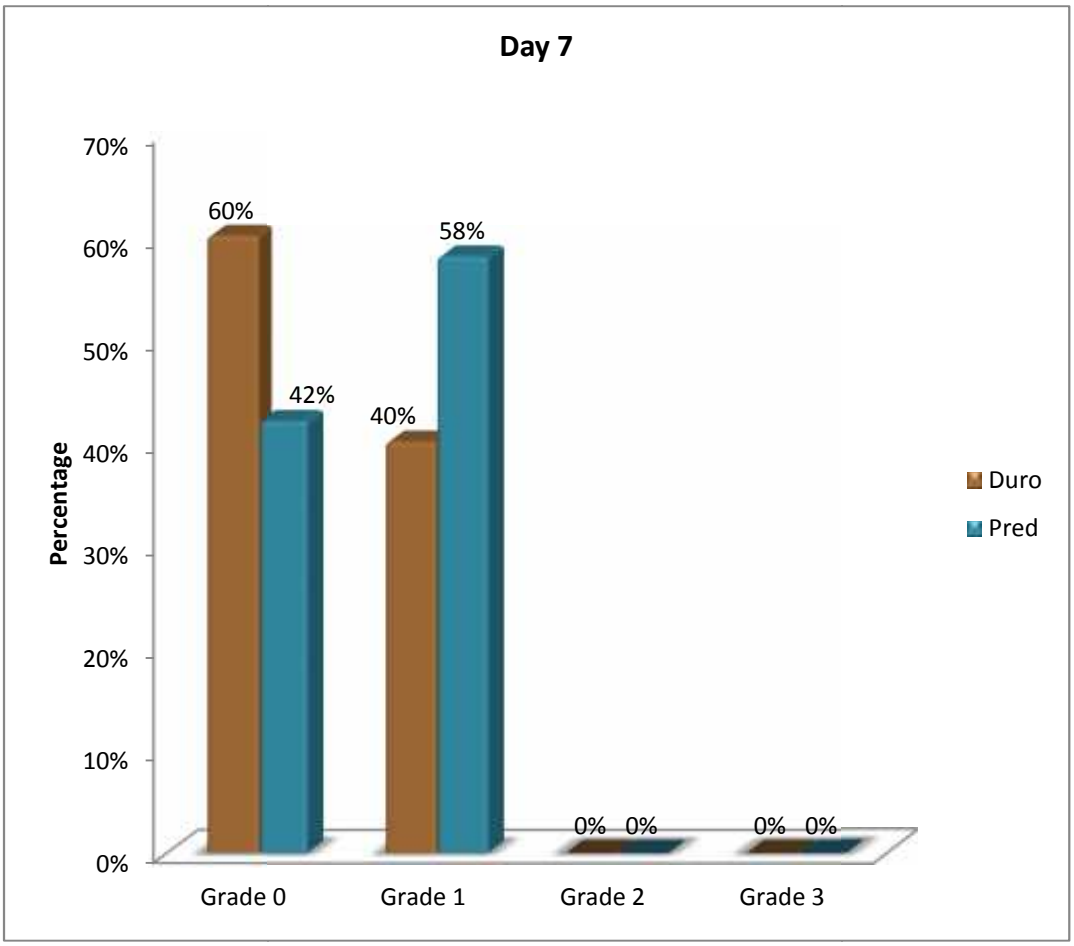
(p = 0.158)



Day 7

	Anterior chamber cells	Difluprednate		Prednisolone	
		No	**%**	**No**	**%**
Day 7	Grade 0 (Absent)	30	60	21	42
	Grade 1 5-10 cells	20	40	29	58
	Grade 2 11-20 cells	0	0	0	0
	Grade 3 21-50 cells	0	0	0	0
	Total	**50**	**100**	**50**	**100**

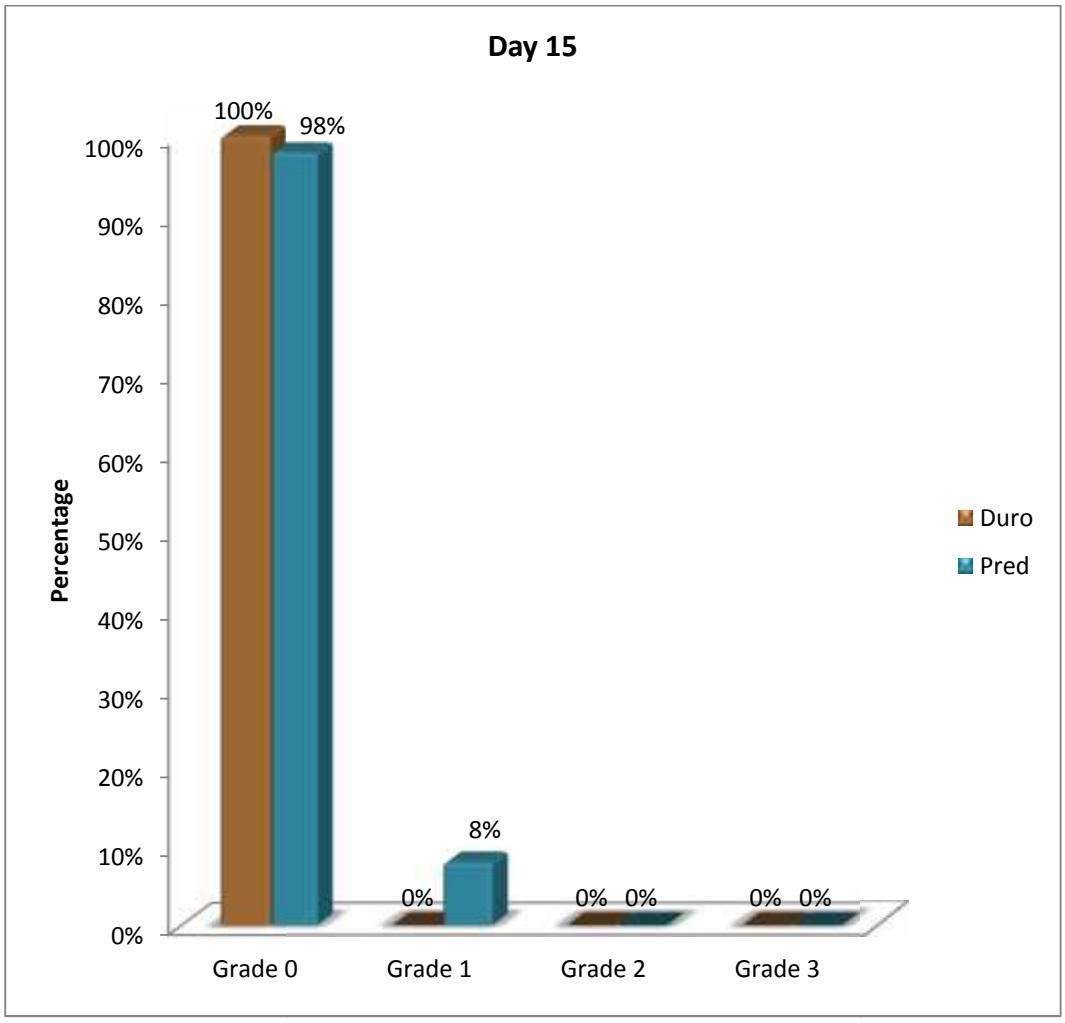
(p=0.109)



Day 15

	Anterior chamber cells	Difluprednate		Prednisolone	
		No	**%**	**No**	**%**
Day 15	Grade 0 (Absent)	50	100	46	92
	Grade 1 5-10 cells	0	0	4	8
	Grade 2 11-20 cells	0	0	0	0
	Grade 3 21-50 cells	0	0	0	0
	Total	**50**	**100**	**50**	**100**

(p=0.118)



Thirty-two patients (64%) in Difluprednate group and 24 patients (48%) in Prednisolone group had Grade 1 cells in the anterior chamber on day 1,15 patients (30%) in Difluprednate group and 20 patients (40%) in Prednisolone group had Grade 2 cells.Four patients(8%) in Prednisolone group had Grade-3 cells in the anterior chamber.

By Day 7, there were 20 patients (40%) in Difluprednate group and 29 patients(58%) in Prednisolone group showed Grade -1 cells.

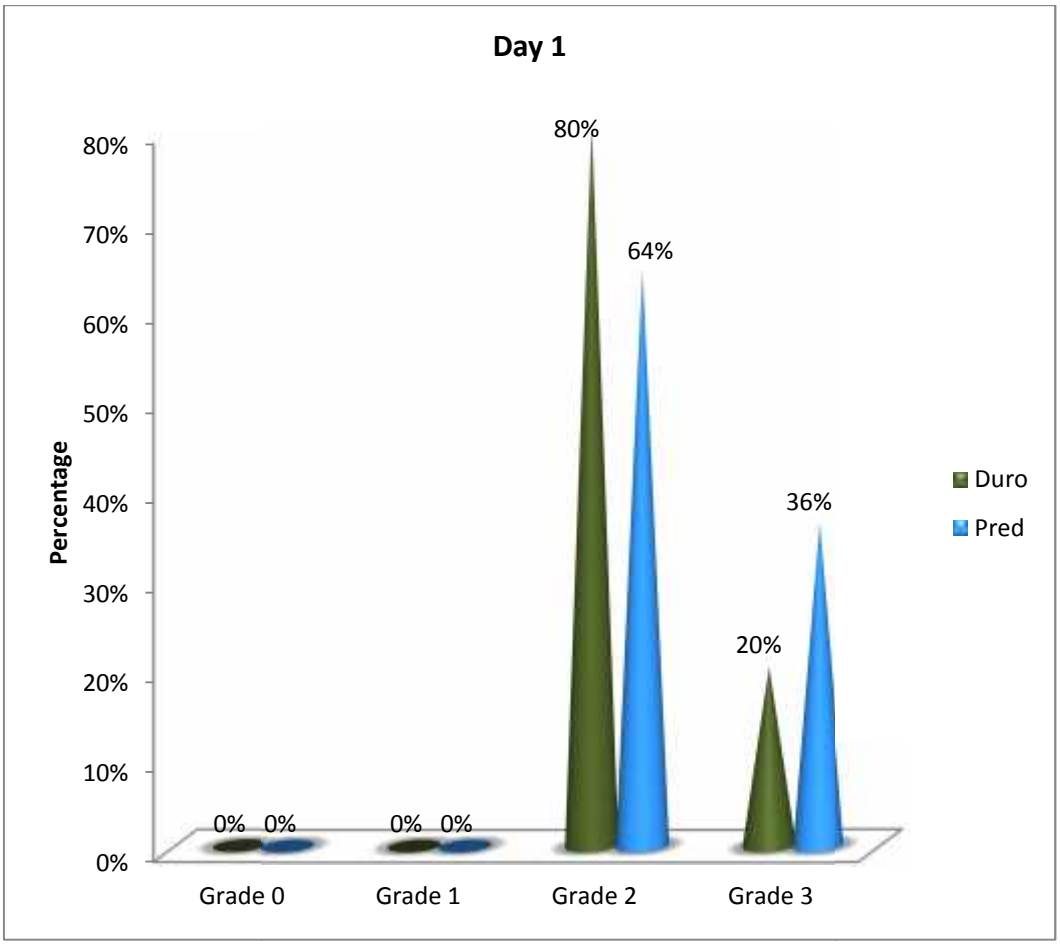
On Day 15, only 4 patients (8%) in Prednisolone group had Grade 1 cells.

TABLE 11 - TOTAL SCORE OF INFLAMMATORY PARAMETERS

Day 1

	Total Score	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 1	None (0)	0	0	0	0
	Mild (1-3)	0	0	0	0
	Moderate (4-7)	40	80	32	64
	Severe (8 and above)	10	20	18	36
	Total	**50**	**100**	**50**	**100**

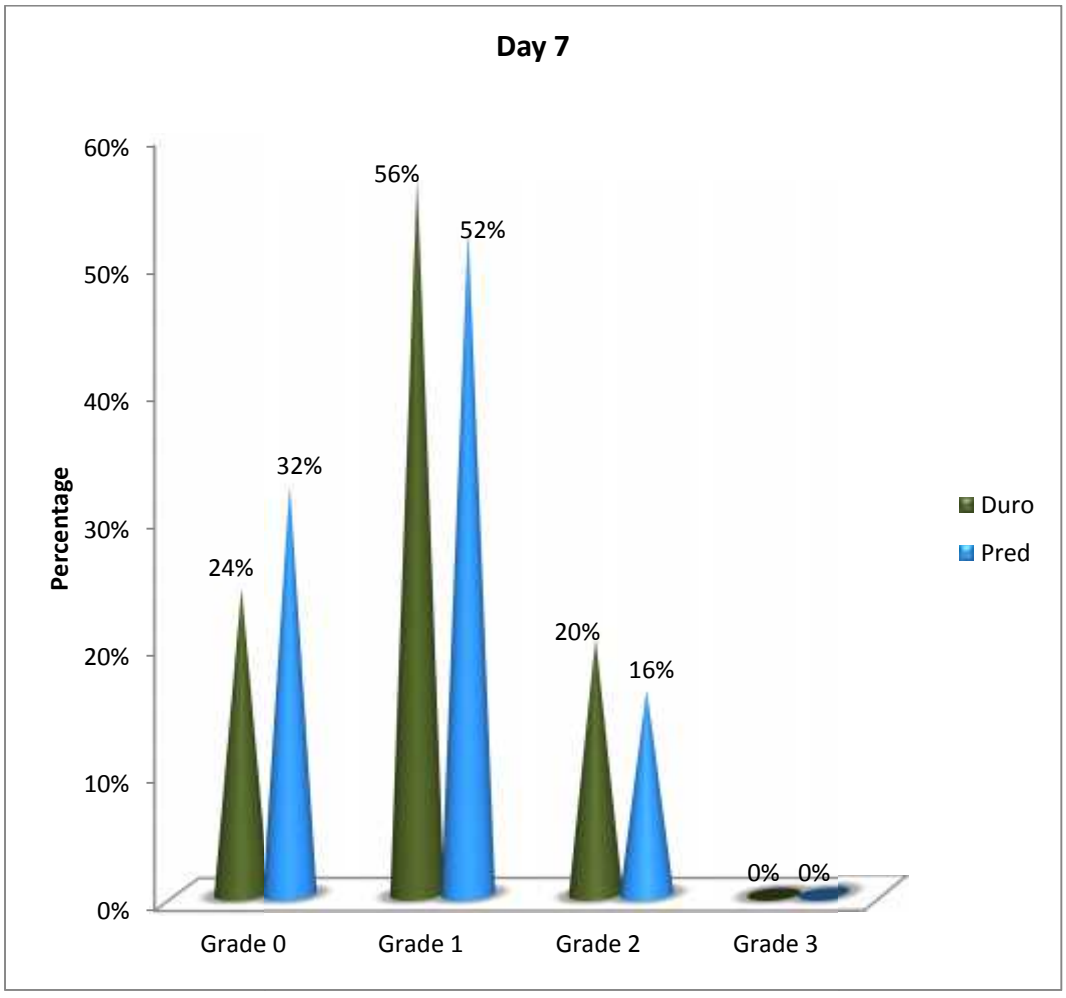
(p = 0.119)



Day 7

	Total Score	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 7	None (0)	12	24	16	32
	Mild (1-3)	28	56	26	52
	Moderate (4-7)	10	20	8	16
	Severe (8 and above)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

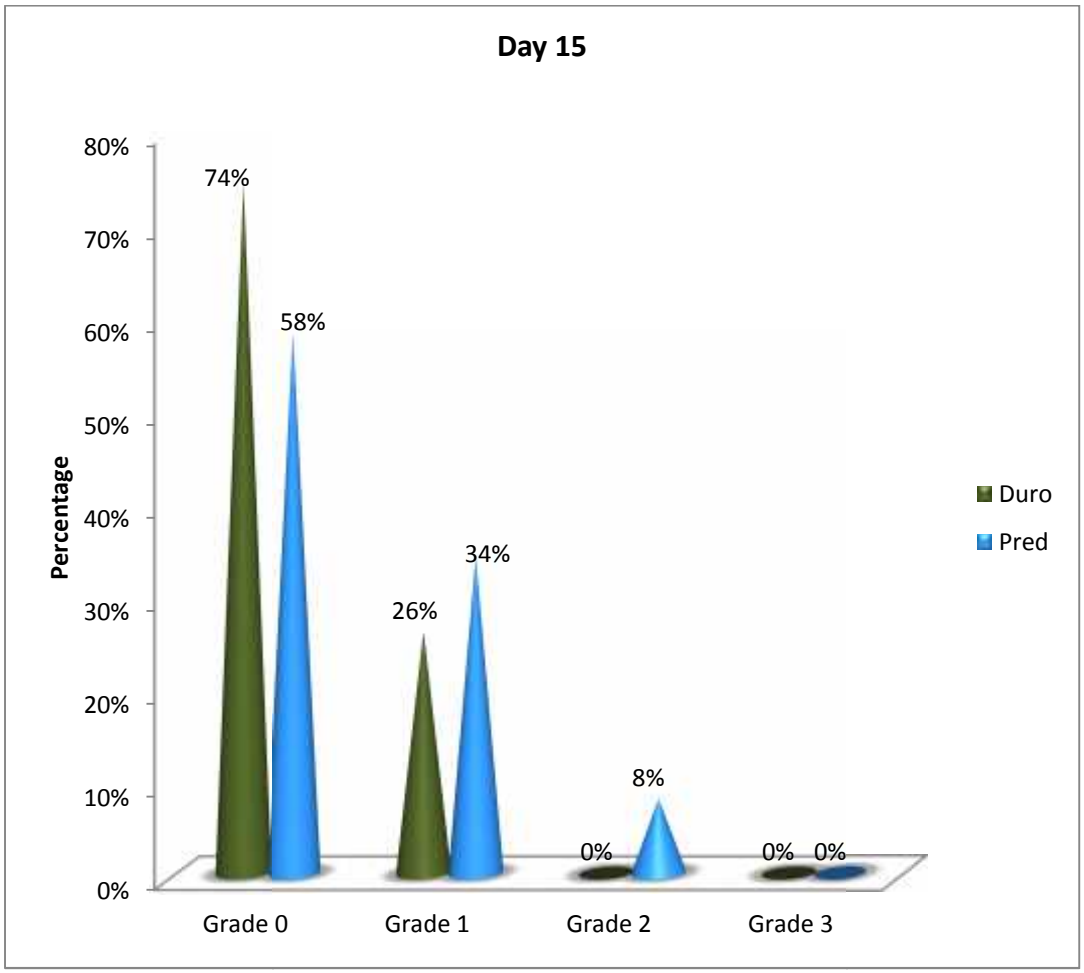
(p=0.648)



Day 15

	Total Score	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 15	None (0)	37	74	29	58
	Mild (1-3)	13	26	17	34
	Moderate (4-7)	0	0	4	8
	Severe (8 and above)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

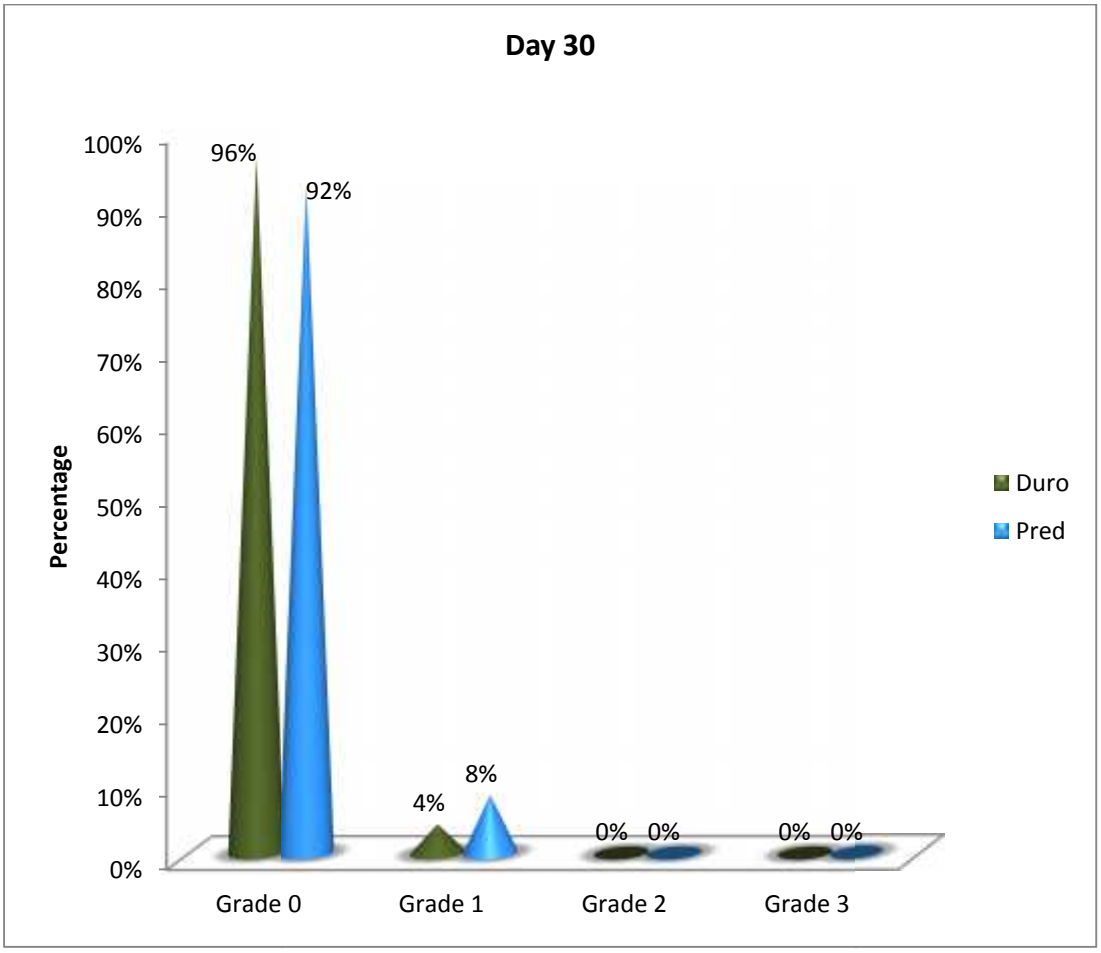
(p=0.064)



Day 30

	Total Score	Difluprednate		Prednisolone	
	Grades	**No**	**%**	**No**	**%**
Day 30	None (0)	48	96	46	92
	Mild (1-3)	2	4	4	8
	Moderate (4-7)	0	0	0	0
	Severe (8 and above)	0	0	0	0
	Total	**50**	**100**	**50**	**100**

(*p*=0.678)



The number of patients with moderate inflammation on day 1 was 40 patients (80%) in Difluprednate group and 32 patients (64%) in Prednisolone group. The other 10 patients (20%) in Difluprednate group and 18 patients (36%) in Prednisolone group had severe inflammation.

By Day 7, 28 patients (56%) in Difluprednate group and 26 patients (52%) in Prednisolone group had mild inflammation. Ten patients (20%) in Difluprednate group had moderate inflammation as compared with 8 patients (16%) in Prednisolone group.

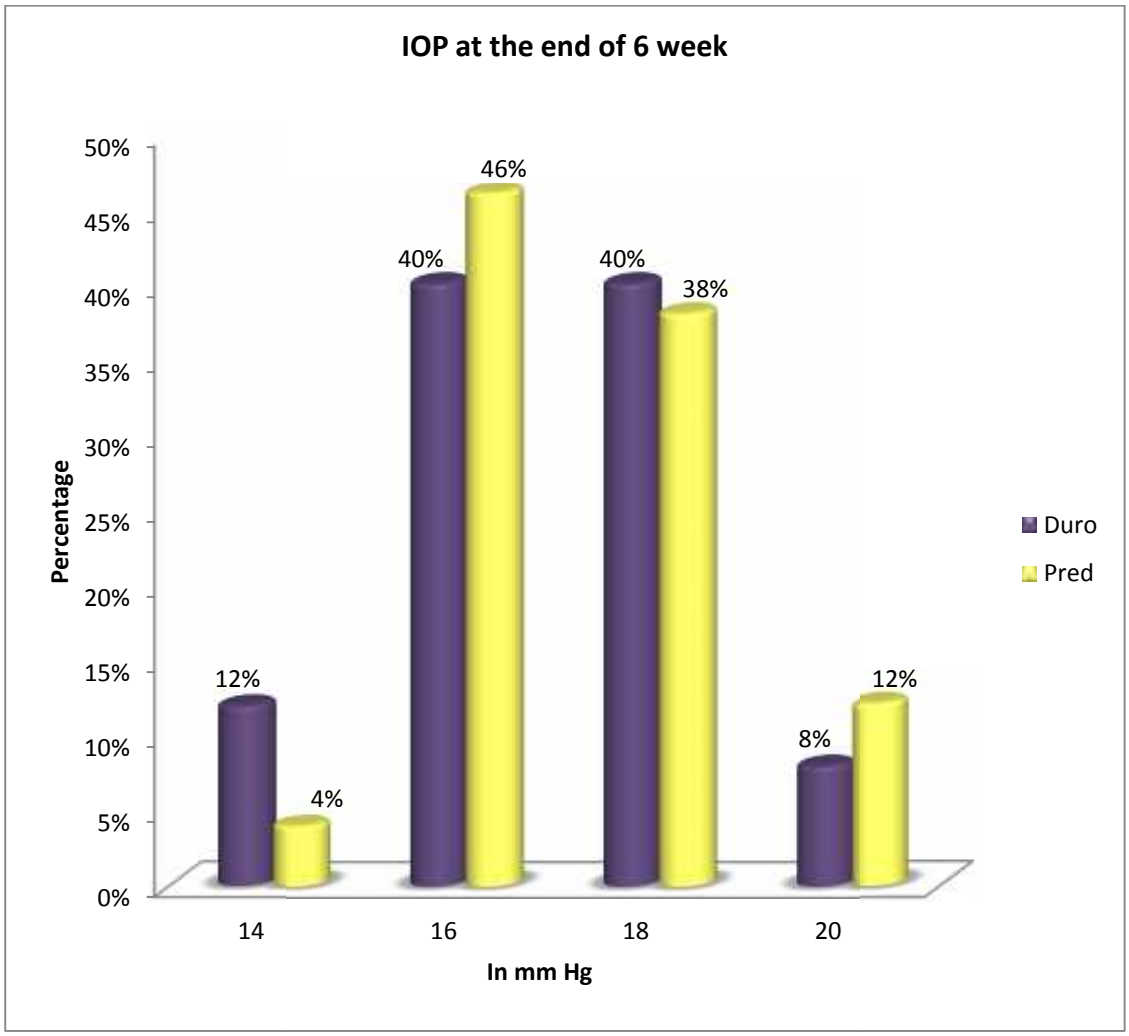
Thirteen patients (26 %) in Difluprednate group and 17 patients (34%) in Prednisolone group had mild inflammation on day 15. Only 4 patients (8%) in Prednisolone group had moderate inflammation.

By Day 30, two patients(4%) in Difluprednate group and 4 patients(8%) in Prednisolone group had mild inflammation.

TABLE 12 – INTRAOCULAR PRESSURE AT THE END OF 6 WEEKS

IOP	Difluprednate		Prednisolone		Total %
	No	%	No	%	
14	6	12	2	4	8
16	20	40	23	46	43
18	20	40	19	38	39
20	4	8	6	12	10
Total	**50**	**100**	**50**	**100**	**100**

(p=0.451)

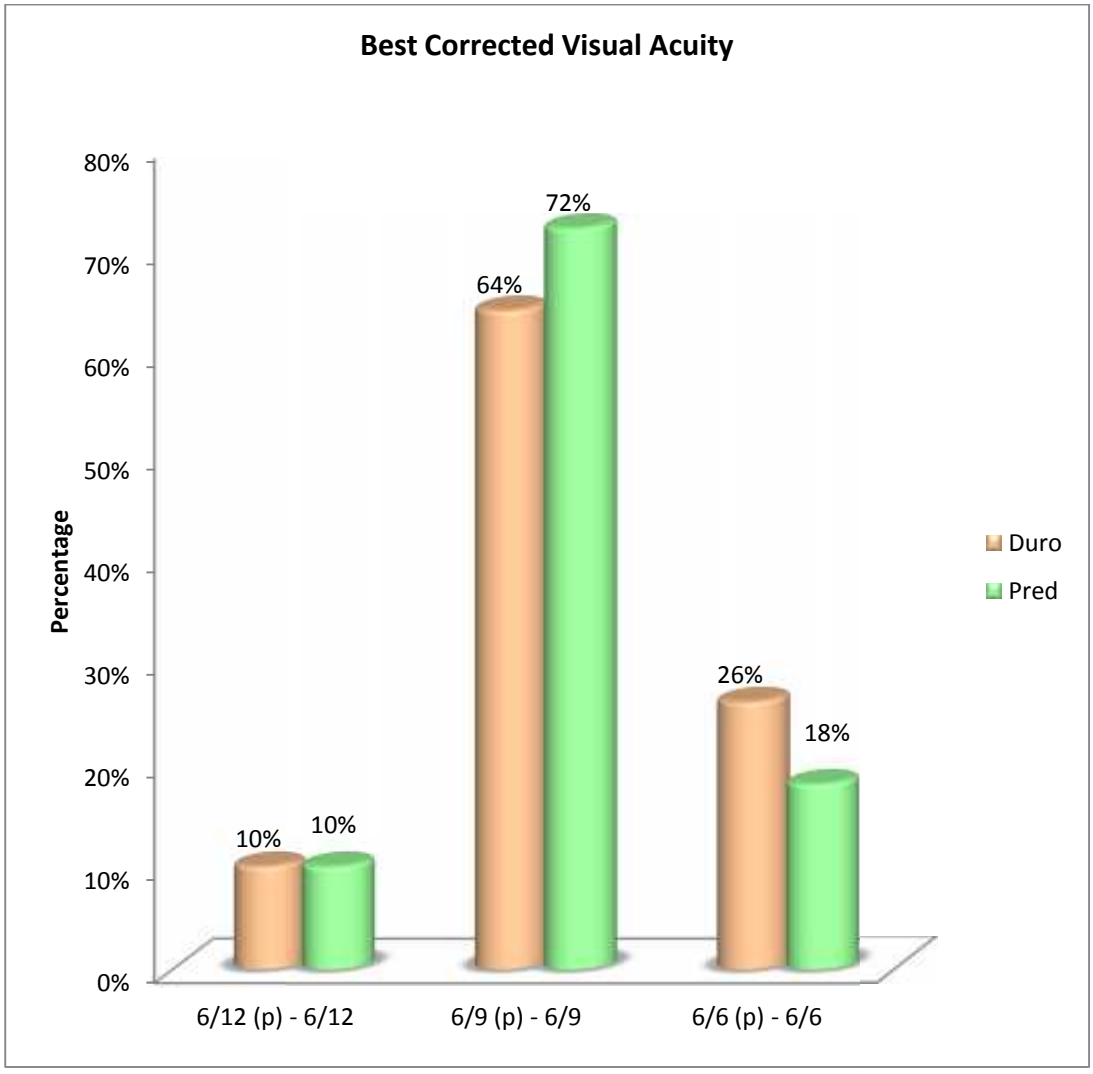


At the end of 6 weeks, the number of patients with IOP of 14 mm Hg was 6 patients(12%) each in Difluprednate and 2 patients (4%) in Prednisolone group. Twenty patients (40%) in Difluprednate group and 23 patients (46%) in Prednisolone group had IOP 16 mmHg. An IOP of 18 mmHg was seen in 20 patients (40%) in Difluprednate group and in 19 patients (38%) in Prednisolone group. Four patients (8%) in Difluprednate group and 6 patients(12%) in Prednisolone group had IOP of 20 mmHg.

TABLE 13 - BEST CORRECTED VISUAL ACUITY

Vision	Difluprednate		Prednisolone		Total %
	No	%	No	%	
6/12 (p) - 6/12	5	10	5	10	10
6/9 (p) - 6/9	32	64	36	72	68
6/6 (p) - 6/6	13	26	9	18	22
Total	**50**	**100**	**50**	**100**	**100**

(p=0.618)



At the end of 6 weeks, the number of patients with best corrected visual acuity of 6/12(p)- 6/12 was 5 patients (10%) in Difluprednate group and 5 patients (10%) in Prednisolone group. Thirty-two patients (64%) in Difluprednate group and 36 patients (72%) in Prednisolone group had visual acuity of 6/9(p) – 6/9. Thirteen patients (26%) in Difluprednate group and 9 patients (18%) in Prednisolone group achieved a vision of 6/6(p) – 6/6.

DISCUSSION

A total of hundred patients with senile cataract who underwent SICS in KLES. Dr.Phabhakar Kore Hospital Belgaum were selected for the study. They were divided into two groups.

Group A - 50 cases (Difluprednate ophthalmic emulsion 0.05% group)

Group B- 50 cases (Prednisolone Acetate Ophthalmic Suspension 1% group)

In the present study, the average age group of patients in Difluprednate group was 60.5 years while in Prednisolone group was 58.8 years. Maximum of the total patients were in age group of 60 to 69 years. The two groups were comparable in age. In a similar study done in USA mean age group of patients in Difluprednate group was 47 years and in Prednisolone group was 43 years. The mean age group in Difluprednate Group was 46.5 years and in Prednisolone group was 42.9 years, in another similar study[34,35].

In the present study, male: female ratio in Dfluprednate group was 29:21 and in Prednisolone group was 35:15. The two groups were similar in gender distribution. This was concurrent with similar studies done in USA where in Difluprednate group percentage of males was 46% where as in Prednisolone group percentage of males was 37.5%[34].

In the present study**,** majority of the cases were senile immature cortical cataract (50%) out of which 22 patients (44%) were in Difluprednate group and 25 patients (50%) in Prednisolone group. 6 patients (12%) in Difluprednate group and 6 patients (12%) in Prednisolone group had nuclear cataract. Six patients (12%) in Difluprednate group had posterior subcapsular cataract. Eleven patients (22%) in

Difluprednate group had mature cataract as compared with 14 patients (28%) in Prednisolone group. Five patients (10%) in Difluprednate group and 5 patients (10%) in Prednisolone group had hypermature cataract. The two groups were comparable in the type of cataract.

In the present study, for baseline IOP, 22% of patients were in the range of 12.2 to 13.4 mmHg in Difluprednate group as compared with 20% in Prednisolone group. Maximum number of patients were in the range of 14.6 to 15.9 mmHg. The two groups were comparable in baseline IOP. This is in concurrence with studies done in USA where the baseline IOP's were similar in both the groups[34,35].

In the present study, only12% in Difluprednate group and 12% patients in Prednisolone group had lid edema on Day 1. By Day 7 the lid edema subsided in both the groups. There was no statistical difference between the groups at any time.

In the present study, Maximum number of patients had Grade 1 conjunctival congestion on Day 1. Out of which there were 45 patients (90%) in Difluprednate group and 43 patients (86%) in Prednisolone group. The numbers of patients with grade 2 were 5(10%) in Difluprednate and 7 (14%) in Prednisolone group (p=0.758). By Day 7, the number of patients with Grade 0 of conjunctival congestion was 14(28%) in Difluprednate group and 19(38%) in Prednisolone group. The total number of patients with Grade 1 had come down to 35 patients (70%) in Difluprednate group and 28 patients (56%) in Prednisolone group. One patient (2%) in Difluprednate group and 3 patients (6%) in Prednisolone group showed Grade 2 conjunctival congestion (p=0.395). there was no statistical significance between the two groups . Six patients (12%) in Difluprednate group had Grade 1 as compared with 8 patient (16%) of Prednisolone group on day 15. Three patients (6%) in

Prednisolone group stil had Grade 2 conjunctival congestion (p=0.287).There was no statistical difference between the two groups at Day 15 and Day 30.

In the present study, Maximum number of patients with ciliary congestion on Day 1 were of Grade 1 out of which 47 patients (94%) were of Difluprednate group and 43 patients (86%) were of Prednisolone group. Three patients(6%) in Difluprednate and 6 patients(12%) in Prednisolone group showed Grade 2 ciliary congestion (p=0.342). This was further reduced to 9 patients (18%) in Difluprednate group had Grade 1 as compared with 14 patients (28%) in Prednisolone group by Day 7 (p=0.342). As of Day 15 none of the patients in Difluprednate group had ciliary congestion but 3 patients (6%) still had Grade 1 in Prednisolone group (p=0.242). There was no statistical difference between the two groups on Day 30.

In the present study, On Day 1, twenty nine patients (58%) in Difluprednate group and 28 patients (56%) in Prednisolone group had mild (grade1) corneal edema. Seven patients (14%) in Difluprednate group and 14 patients (28%) in Prednisolone group had Grade 2 corneal edema (p=0.136). There was no statistical difference between the two groups. 3 patients (6%) in Difluprednate group and 9 patients (18%) in Prednisolone group had Grade 1 corneal edema on day 7(p=0.121).There was no statistical difference here also. On day 15, only 2 patients (4%) in Difluprednate group and 6 patients (12%) in Prednisolone group had Grade 1 corneal edema (p=0.269). There was no statistical significance between the two groups here also. There was also no statistical significance between the two groups on Day 30. A similar study done in Newyork found that corneal thickness at Day 1 was 33μm (measured via pachymetry) in Difluprednate-treated eyes (p=0.026). More eyes were without corneal edema in the Difluprednate group at Day 1(62% v/s 38%

respectively; p=0.019)[36]. In another similar study done in Los Angeles found that at Day 30, endothelial cell density was 195.52 cells/mm^2 higher in the Difluprednate-treated eyes as compared to Prednisolone-treated eyes and at Day 15, retinal thickness was 7.74μm less in Difluprednate-treated eyes[37]. They also found that administration of Difluprednate had less corneal edema (measured via pachymetry) on Day 1 when compared with Prednisolone. The mean central corneal thickness at Day 1 in the Difluprednate group increased to 28μm(560 to 590 μm).This increase was about half of that observed in the Prednisolone group, which was *57*μm (from 562 to 619 μm). Additionally, central corneal thickness at Day 1 in the Difluprednate group averaged 32.59μm less than that of the Prednisolone group (p=0.026). At Day 1, corneal swelling in the Difluprednate group was 31.79μm less than the Prednisolone group (p=0.033) compared with baseline. One of the important assessment was endothelial cell counts. In the Difluprednate arm, there was significantly less endothelial cell loss at Day 30, with a difference of 180 cells between the two groups[37].

In the present study, The number of patients with Grade 1 Anterior Chamber Flare on Day 1 was 32 (64%) in Difluprednate group and 23 (46%) in Prednisolone group. 13 patients (26%) of Difluprednate group and 21 patient (42%) of Prednisolone group had Grade 2 Flare (p=0.178). There was no statistical difference between the two groups. By Day 7, in Difluprednate group, 16 patients (32%) had Grade 1 Flare, whereas 25 patients (50%) of Prednisolone group had Grade 1 Flare. Six patients (12%) and 6 patients (12%) in Difluprednate and Prednisolone group respectively had Grade 2 Flare (p=0.157). On Day 15, 4 patients (8%) in Difluprednate group and 11 patients (22%) in Prednisolone group had Grade 1 flare (p=0.091). There was no statistical difference between the two groups at any time. In USA a similar study was done, where Difluprednate was compared to a placebo found

that clearing of inflammation on Day 14, defined as an AC cell grade of 0 (<5 cells) and a flare grade of 0 (complete absence),was achieved in a significantly greater percentage of subjects treated with Difluprednate, compared with placebo (74.7% v/s 42.5% p=0.0006). A significantly greater percentage of Difluprednate-treated subjects were free of ocular pain/discomfort on Day 14 than placebo-treated subjects (64.6% v/s 30.0% ; p=0.0004)[38].

In the present study, 32 patients (64%) in Difluprednate group and 24 patients (48%) in Prednisolone group had Grade 1 cells in the anterior chamber on day 1,15 patients (30%) in Difluprednate group and 20 patients (40%) in Prednisolone group had Grade 2 cells. Four patients (8%) in Prednisolone group had Grade-3 cells in the anterior chamber (p=0.101) By Day 7, there were 20 patients (40%) in Difluprednate group and 29 patients (58%) in Prednisolone group showed Grade -1 cells (p=0.109). On Day 15, only 4 patients (8%) in Prednisolone group had Grade 1 cells (p=0.118). There was no statistical difference between the two groups at any time. Sirion therapeutics conducted a similar study where in the primary endpoint was the difference from baseline in AC cell grades between the Difluprednate and Prednisolone groups. At Day 14, the Difluprednate group achieved a mean cell grade reduction of 2.1, compared to 1.9 in the Prednisolone group, confirming the noninferiority of Difluprednate to Prednisolone[39]. Another similar study was done by Sirion therapeutics, where comparison was done between Difluprednate and Betamethasone found that Difluprednate was effective in reducing AC cell ,flare, and total signs and symptom scores. After 14 days of Difluprednate treatment, 72% of patients had less than 10 cells and 11% had no cells in the anterior chamber[39]. Another similar study was done in Arizona, USA, where comparison was done between Difluprednate and Betamathasone found that there were no statistically

significant differences between the treatment groups in mean AC cell count, mean AC flare on days 3,7 or 14.This study showed that treatment with Difluprednate 0.05% was atleast as effective as Betamethasone 0.1% in reducing postoperative inflammation and its safety profile was acceptable[40]. A similar study was also done in Florida where Difluprednate was compared to a placebo found that clearing of inflammation on Day 14, defined as an AC cell Grade of 0 (<5 cells) and a flare Grade of 0 (complete absence), was achieved in a significantly greater percentage of subjects treated with Difluprednate, compared with placebo (74.7% v/s 42.5% p=0.0006). A significantly greater percentage of Difluprednate-treated subjects were free of ocular pain/discomfort on Day 14 than placebo-treated subjects (64.6% v/s 30.0%; p=0.0004)[38].

In the present study, the number of patients with moderate inflammation on day 1 was 40 patients (80%) in Difluprednate group and 32 patients (64%) in Prednisolone group. The other 10 patients (20%) in Difluprednate group and 18 patients (36%) in Prednisolone group had severe inflammation (p=0.119). By Day 7, 28 patients (56%) in Difluprednate group and 26 patients (52%) in Prednisolone group had mild inflammation. Ten patients (20%) in Difluprednate group had moderate inflammation as compared with 8 patients (16%) in Prednisolone group (p=0.648). 13 patients (26%) in Difluprednate group and 17 patients (34%) in Prednisolone group had mild inflammation on day 15. Only 4 patients (8%) in Prednisolone group had moderate inflammation (p=0.064) By Day 30, 2 patients (4%) in Difluprednate group and 4 patients (8%) in Prednisolone group had mild inflammation (p=0.678). There was no statistical difference between the two groups at any time. This correlates with a study by Sirion therapeutics **(**2008), where the primary endpoint was the difference from baseline in AC cell grades between the

Difluprednate and Prednisolone groups. At Day 14, the Difluprednate group achieved a mean cell grade reduction of 2.1, compared to 1.9 in the Prednisolone group, confirming the noninferiority of Difluprednate to Prednisolone[39]. A similar study was done in Texas, USA where comparison was done between Difluprednate and Betamathasone found that there were no statistically significant differences between the treatment groups in mean AC cell count,mean AC flare on days 3,7 or 14.This study showed that treatment with Difluprednate 0.05% was atleast as effective as Betamethasone 0.1% in reducing postoperative inflammation and that its safety profile was acceptable[40].

In the present study, at the end of 6 weeks, the number of patients with IOP of 14 mm Hg was 6 patients (12%) each in Difluprednate and 2 patients (4%) in Prednisolone group. 20 patients (40%) in Difluprednate group and 23 patients (46%) in Prednisolone group had IOP 16 mmHg. An IOP of 18 mmHg was seen in 20 patients (40%) in Difluprednate group and in 19 patients (38%) in Prednisolone group. 4 patients (8%) in Difluprednate group and 6 patients (12%) in Prednisolone group had IOP of 20 mmHg (p=0.451). There was no statistical difference between the baseline IOP and IOP at 6 weeks. Sirion therapeutics found no significant differences in IOP between the two groups at baseline and at 21 days[39]. In USA, similar study was done where in Difluprednate was compared to a placebo found that 3 subjects (3.7%) in the Difluprednate group had a clinically significant IOP rise (defined as >21 mm Hg and a change from baseline > 10 mm Hg at the same visit)[40]. The three subjects were children.

In the present study, at the end of 6 weeks, the number of patients with best corrected visual acuity of 6/12(p)- 6/12 were 5 patients (10%) in Difluprednate group

and 5 patients (10%) in Prednisolone group. Thirty two patients (64%) in Difluprednate group and 36 patients (72%) in Prednisolone group had visual acuity of 6/9(p) – 6/9. 13 patients (26%) in Difluprednate group and 9 patients (18%) in Prednisolone group achieved a vision of 6/6(p)–6/6 (p=0.618). There was no statistical difference between the two groups at any time. This was concurrent with a similar study done in USA where there were no significant differences in visual acuity between the groups[40].

CONCLUSION

Several steroids have been introduced over the last few decades, still Prednisolone has been considered the "gold standard" and indeed has enjoyed a status as the "go-to" steroid for many inflammatory conditions. All Ophthalmic corticosteroids, both topical and systemic, have the potential to provoke a rise in intraocular pressure (IOP). Difluprednate can also be associated with elevated IOP. Thus, standard of care practices must be employed, with frequent measurement of eye pressure for anyone using this medication. In our study, none of the patients showed raised IOP in both groups from baseline till the end of 6 weeks at any time.

With respect to scoring of inflammation in our study, the patients in Difluprednate group responded faster as compared to patients in Prednisolone group. Difluprednate Ophthalmic Emulsion 0.05% was as efficacious as Prednisolone Acetate 1% Ophthalmic Suspension in controlling the inflammation.

In conclusion, in our study Difluprednate Ophthalmic Emulsion 0.05% was as effective as Prednisolone Acetate 1% Ophthalmic Suspension in treating postoperative inflammation following catatract surgery. Thus, Difluprednate Emulsion 0.05% appears to be a promising addition to the surgical armamentarium for treating postoperative inflammatory conditions. With proven efficacy of Difluprednate, we now have a new standard for potency in a topical corticosteroid, with excellent anti-inflammatory properties and an ideal formulation for our patients.

SUMMARY

The present study included 100 cases with senile cataract, who underwent SICS with PCIOL implantation in KLES Dr. Prabhakar Kore Hospital, Belgaum. They were divided into two groups receiving the following study medications postoperatively. Group A consisted of 50 patients in Difluprednate group and Group B consisted of 50 patients in Prednisolone group.

The evaluation of postoperative inflammation has been summarized as follows.

Lid edema was not found in any patient by the end of the week.

Conjunctival congestion persisted in 6% patients in Prednisolone group as compared with none of the patients in Difluprednate group on day 15. By Day 30, patients in both the groups had reduced conjunctival congestion completely.

Ciliary congestion was seen in 3% patients in the Prednisolone group as compared with none of the patients in Difluprednate group by Day 15.By day 30, none of the patients in either of the groups had ciliary congestion.

Corneal edema was seen in 4% patients in Difluprednate group and 12% in Prednisolone group by day 15. By day 30 none of the patients in either of the groups had corneal edema.

Anterior chamber flare and cells –Mild flare and cells was seen in 22% of patients in Prednisolone group and in only 8% of patients in the Difluprednate group by day 15, which regressed completely in both the groups by day 30. Difluprednate controlled inflammation faster when compared clinically with Prednisolone.

Total score-When the total scoring of all these parameters was compared, in Prednisolone group 34% of the patients persisted with mild (Grade1-3) inflammation on Day 15 as compared with 26% of the patients in Difluprednate group. But by Day 30, both the groups were equally effective in controlling the inflammation.

Intraocular pressure in both the groups was similar at baseline and there was no significant difference between the two groups by 6 weeks.

There were no patients with cystoid macular edema at any time during the study.

Though the time taken to achieve anti-inflammatory activity was slower in Prednisolone group as compared with Difluprednate group, there was no difference in the visual outcome in both the groups.

BIBLIOGRAPHY

1. Jaffe NS: Elimination of contamination from ophthalmic solution for intraocular surgery. *Trans Am Acad Ophthalmol Otolaryngol* . 1970;74: 406-416.
2. Korenfeld Ms, Silverstein SM, Cooke Dl, Roger Vogel, Robert Crockett. Difluprednate Ophthalmic Emulsion 0.05% for post-operative inflammation and pain-Journal of cataract and Refractive surgery.2009; 35:26-34.
3. Foster SC: Topical steroid treatment of ocular inflammation. In Advances on Ocular pharmacology, Ophthalmology Clinics of North America.1997; 10: 389-403.
4. Friedlander MH: Coticosteroid therapy of ocular inflammation. *Int Ophthalmol Clin* .1983; 23: 175.
5. Apt L, Henrick A, Silverman LM: Patient compliance with the use of topical ophthalmic corticosteroid suspensions. *Am J Ophthalmol* .1979; 87: 210.
6. William Stringer, Sirion Therapeutics, Tampa, FL.com.
7. Sakiko Nakano. Steroid eye drop treatment (difluprednate ophthalmic emulsion) is effective in reducing refractory diabetic macular edema.Indian Journal of Ophthalmology.2009;58.
8. Hayashi K and others: Risk factors for corneal endothelial injury during phacoemulsification. *J Cataract Refract Surg* .1996;22: 1079-1084.
9. Emmett T. Cunningham, Jon D. Wender: Practical approach to the use of corticosteroids in patients with uveitis. *Clinical J Ophthalmology* 2010.

10. E Mayer, Cadman .A 10 year retrospective survey of cataract surgery in a single eye unit.*Br J Ophthalmology.* 2003;87:867-869.

11. Glasser DB, Schultz RO, Hyndiuk RA: The role of viscoelastics , canulas and irrigating solution additives in post -cataract surgery corneal edema: a brief review. *Lens Eye Toxic Res* .1992;9: 3-4.

12. Galin MA, Tuberville AW, Dotson RS: Immunologic aspects of intraocular lenses. *Int Ophthalmol Clin* .1982;22: 227-234.

13. Udell JJ, Abelson MB: Chemical mediators of inflammation. *Int Ophthalmol Clin*. 1983;23: 15-26.

14. Ambache N, Brummer HC: A simple chemical procedure for distinguishing E from F prostaglandins, with application to tissue extracts. *Br J Pharmacol Chemother*. 1970; 33: 162-170.

15. Neufeld AH, Sears ML: Prostaglandins and the eye. *Prostaglandins* . 1973; 4: 157-168.

16. Malmsten C: Prostaglandins, thromboxanes and Leukotrienes in Inflammation. *Am J Med* .1986; 10: 11-15.

17. Sander DR Kraff MC, Lieberman HL, Talley A : Breakdown and reestablishment of the blood aqueous barrier with implant surgery. *Arch Ophthalmol*. 1982; 100: 588-590.

18. Timothy L, Comstock and Heleen H. Decory: Advances in corticosteroids therapy for Ocular Inflammation: Loteprednol Elabonate. *Int.J of Inflammation*. 2012; 86:1-11.

19. El- Maghraby A, Marzouki A, Matheen TM: Reproducibility and validity of laser flare/cell meter measurements as an objective method of assessing intraocular inflammation. *Arch Ophthalmol* .1992; 110: 960-962.

20. Yi DH, Dana MR: Corneal edema after cataract surgery: incidence and etiology. *Semin Ophthalmol* 2002;17:110-4.

21. George L, Spaeth MD, Merlyn M et.al: Steroid-Induced Glaucoma. *Trans Am Ophthal Soc.*1977;75:353-381.

22. Laurell C-G, Zetterstrom C: Effects of dexamethasone, diclofenac or placebo on the inflammatory response after cataract surgery. *Br J Ophthalmol.* 2002; 86: 1380-1384.

23. Lieberman HL, Wallyn RH: Breakdown and reestablishment of the blood aqueous barrier with implant surgery. *Arch Ophthalmol* .1982;100: 588-590.

24. Fearnley IR, Spalton DJ, Smith SE: Anterior segment fluorophotometry in acute anterior uveitis. *Arch Ophthalmol* .1987;105: 1550-1555.

25. Spalton DJ, Shah SM: A comparative study of the laser flare cell meter and fluorophotometry in the assessment of the blood aqueous barrier in the pathological eye. ARVO Abstracts. *Invest Ophthalmol Vis Sci* .1991; 32: 886.

26. Othenin-Girard P:Dexamethasone versus Diclofenac Sodium eye drops to treat Inflammation after cataract surgery.*J Cataract And Refract. Surgery.* 1994; 20: 9-12.

27. Gass JDM, Norton EWD: Follow-up study of cystoid macular edema following cataract extraction. *Trans Am Acad Ophthalmol Otolaryngol.* 1970;73: 665-682.

28. Rho DS: Treatment of acute pseudophakic cystoid macular edema. *J Cataract and Refrac.Surg.*2003.

29. Abelson MB, Brutus S: Corticosteroids in ophthalmic practice. In Albert DM, Jakobiec FA (eds): Principles and Practice of Ophthalmology: Basic Sciences, Philadelphia, WB Saunders, 1994; 45:1013.

30. Polansky JR, Weinres RN: Anti-inflammatory agents, steroids as anti-inflammatory agents. In Sears ML(ed): Pharmacology of the eye. Berlin, Springer-Verlag.1984; 31:460.

31. Rubin B, Palestine AG: Complications of corticosteroids and immunosuppressive drugs. *Int Ophthalmol Clin.*1989; 29: 159.

32. Dunne J A Jacobs N. Morrison A .Efficacy in anterior uveitis of two known steroids and Topical Tolmetin. British Journal of Ophthalmology. 1985;69.

33. Krupin T, LeBlanc RP, Becker B : Uveitis in association with topically administered corticosteroid. *Am J Ophthalmol* .1970;76: 883.

34. Nancy Groves: Difluprednate is noninferior to prednisolone, data show; Ophthalmology Times; 2009.

35. Foster CS: Durezol (Difluprednate Ophthalmic Emulsion 0.05%) compared with Pred Forte 1% ophthalmic suspension in the treatment of endogenous anterior uveitis: Ocular Immunology and Uveitis Foundation.

36. Eric D Donnenfold: Difluprednate for the prevention of ocular inflammation postsurgery: an update. Clinical Ophthalmology. 2011;5:811-816.

37. Uday Devgan MD, Cataract Surgery in the Patient with Uveitis; *J* Clinical Ophthalmology 2010.

38. Stephen Smith, Douglas Lorenz, James Peace: Difluprednate ophthalmic emulsion 0.05% administered two times daily for managing ocular inflammation and pain following cataract surgery: *J* Clinical Ophthalmology. 2010;33:83-91.

39. William Stringer, Sirion Therapeutics, com.

40. Karim N Jamal and David G Callanan: The role of difluprednate ophthalmic emulsion in clinical practice: *J* Clinical Ophthalmology. 2009; 25: 381-390.

ANNEXURE - I

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr/Mrs/Ms_____

You are invited to participate in our research study titled "A ONE YEAR RANDOMISED CLINICAL TRIAL TO COMPARE EFFICACY OF TOPICAL DIFLUPREDNATE O.O5% OPHTHALMIC EMULSION WITH PREDNISOLONE ACETATE IN CONTROL OF POST-OPERATIVE INFLAMMATION FOLLOWING CATARACT SURGERY IN KLES HOSPITAL BELGAUM.." conducted by Dr._____, Post Graduate in M.S. Ophthalmology under the guidance of Dr. _____., M.S., D.O.M.S., Professor in the Department of Ophthalmology, J .N. Medical College, Belgaum.

Respected Sir/Madam we request you to enroll yourself to participate in our study as you are eligible for participating in the study. Your participation in research is voluntary. If you decide to participate you are free to withdraw at any time.

Purpose of the Study:

The purpose of research is to COMPARE THE EFFICACY OF TOPICAL DIFLUPREDNATE OPHTHALMIC 0.05% WITH TOPICAL PREDNISOLONE ACETATE IN CONTROL OF INFLAMMATION FOLLOWING CATARACT SURGERY..

Procedure Involved:

If you agree to enroll yourself in this study, I will ask your present, past and family history. You will be clinically examined and data of your relevant

investigations will be accessed. Then you will be subjected to slit lamp examination, direct ophthalmoscopy. and cataract surgery. The hence obtained data will be monitored and documented.

Risks and Benefits :

There are no major risks involved in the above mentioned procedures however some discomfort may occur, for which all precautions will be taken.

Your participation may benefit you and others in getting a new ophthalmic eye drops with less frequent dosing and early of inflammation in a shorter duration of time.

Alternatives :

If you are not willing to participate you will be treated according to the existing protocol & it will not affect your relationship with this hospital.

Costs for participating in this research:

The participant will have to pay for the investigations which are the part of the existing management protocol for this ailment.

Privacy and Confidentiality:

No information about you or information provided by you during the research will be disclosed to others without your written permission.

Authorization to Publish Results:

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity.

Compensation:

In the event of injury related to the study, treatment will be made available through KLE Prabhakar Kore Hospital & MRC, Belgaum. There is no compensation or payment for such medical treatment by law. The doctors and the staff will provide facilities and medical attention to you.

Questions:

If you have any questions about the research you may please contact:

1) Chief investigator, Dr. _____,

P.G., Department of Ophthalmology, JNMC, Belgaum. Contact No. _____.

2) Dr. _____, Guide,

Professor, Department of Ophthalmology, JNMC, Belgaum. Ph: _____

3) Dr. _____ ,

Principal, JNMC, Belgaum and chairman of Institutional Ethics Committee.

Ph. _____

Statement for participation in research trial

I, Mr./Ms./Mrs _____ voluntarily agree for the participation as a subject of this study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in my own vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Witness Name :_____

Signature of Witness:_____

Investigators Name: _____

Signature of Investigator : _____

Date:

Place:

Name of Guide:_____

Signature of guide:_____

ANNEXURE – II: PROFORMA

Sr. No:	Date of Surgery:
Name:	Group:
Age/Sex:	Dosage:
I.P.No:	Date of Admission:
Address:	Diagnosis:

CHIEF COMPLAINTS:

Diminision of vision	RE	LE
Duration		

HISTORY OF PRESENT ILLNESS:

1. Diminision of vision	Gradual	Sudden
	Progressive	Static
	Painless	Painful
	For Distant	For near
Visual Improvement	Dim Light	Bright Light

2. Diplopia

3. Coloured Haloes

4. Black spots before the eyes.

5. Second sight.

6. Watering	Constant	Intermittent
7. Redness	Yes	No
8. H/o wearing spectacles	Duration	
9. H/o Diabetes, Hypertension	Duration	

PAST HISTORY:

- Conjunctivitis/Dacryocystitis/Any others
- Redness/Pain/Ocular trauma
- Surgery
- Chronic Respiratory Disease/Chronic Constipation/Bleeding Disorders/Rheumatoid Arthritis

DRUG HISTORY

Oral Medication – Indication

Duration

Frequency

Topical Steroids – Indication

Duration

Frequency

H/o Drug Allergy –

FAMILY HISTORY

PERSONAL HISTORY

GENERAL PHYSICAL EXAMINATION:

Pallor	Yes/No	Lymphadenopathy	Yes/No	Pulse:
Edema	Yes/No	Clubbing	Yes/No	BP
Icterus	Yes/No	Cyanosis	Yes/No	Temperature

SYSTEMIC EXAMINATION:

CVS

RS

PA

CNS

OCULAR EXAMINATION: RE LE

1. Visual Acuity

    Distant

    Pinhole

    With old glasses

    Near

2. Adnexa

3. Sclera

4. Conjunctiva

    Congestion – Palpebral/Bulbar/Ciliary

    Chemosis

5. Cornea – Surface/Clarity/Vascularisation

6. Anterior Chamber

7. Iris-Color/Pattern

8. Pupils-Size/Shape/Reaction

9. Lens

10. Fundus

11. Ocular Movements

SLIT LAMP EXAMINATION:

1. Conjunctiva
2. Cornea
3. Anterior Chamber
4. Iris
5. Pupil
6. Lens
7. Any other

FINAL DIAGNOSIS: SENILE CORTICAL CATARACT

INCIPIENT IMMATURE

SENILE NUCLEAR CATARACT

SENILE MIXED CATARACT

SENILE MATURE CATARACT

HYPERMATURE CATARACT

POSTERIOR SUBCAPSULAR CATARACT

INVESTIGATIONS:

1. Ocular

a) Lacrimal Patency Patent Blocked

RE

LE

b) IOP(Schiotz Tonometry)

RE

LE

2. Urine-alb

- sug

3. Any other

A – Scan Reading: PC IOL:

DETAILS OF SURGERY:

Type of Surgery :

Small Incision Cataract Surgery with PCIOL

Date of Surgery: Name of Surgeon:

Pre – Operative Medications:

Anaesthesia: Peribulbar Lignocaine-with hyalase & adrenaline

-with sodium bicarbonate

Facial Block & Adrenaline

Adequate Inadequate

Viscoelastic Used:

IOL Type:

Postoperative medications:

Group Dosage

POST OPERATIVE EVALUATION OF INFLAMMATION

Symptoms	**Pain**	**Watering**	**Visual**
Day 1			
Day 7			
Day 15			
Day 30			

1. LID EDEMA	DAY 1	DAY 7	DAY 15	DAY 30
(None) 0				
Mild 1				
Moderate 2				
Severe3				

2. CONJUNCTIVAL CONGESTION	Day 1	Day 7	Day 15	Day 30
None 0				
Mild (some vessels injected) 1				
Moderate (diffuse injection 2				
Severe (intense injection 3				

3. CILIARY FLUSH	Day 1	Day 7	Day 15	Day 30
Non 0				
Mild (some vessels injected) 1				
Moderate (diffuse injection) 2				
Severe (intense injection) 3				

4. CORNEA (edema/Descemet's folds/KP's)	**Day 1**	**Day 7**	**Day 15**	**Day 30**
None 0				
Mild 1				
Moderate 2				
Severe 3				

5. ANTERIOR CHAMBER FLARE (FINE SLIT)	**Day 1**	**Day 7**	**Day 15**	**Day 30**
Absent 0				
Mild (barely detected)1				
Moderate (iris & lens details seen) 2				
Severe (Iris & lens details not seen) 3				

6. ANTERIOR CHAMBER CELLS	**Day 1**	**Day 7**	**Day15**	**Day 30**
Absent 0				
5 – 10 cells 1				
11 – 20 cells 2				
21 – 50 cells 3				
>50 cells/hypopyon 4				

7. Pupils	Day 1	Day 7	Day15	Day 30
Synechiae absent				
Synechiae present				

8. Vitreous Haze	Day 1	Day 7	Day15	Day 30
Absent 0				
Few scattered fine and coarse opacities. Fundus seen clearly 1				
Scattered fine and coarse opacities but fundus somewhat obscured 2				
Many opacities with marked blurring of the fundus 3				
Dense opacities prevent view of the fundus 4				

TOTAL SCORE:

0 = None

1 – 3 = mild

4 – 7 = moderate

8 & above/exudative membrane/KP's = severe

TOTAL SCORE

Day 1

Day 7

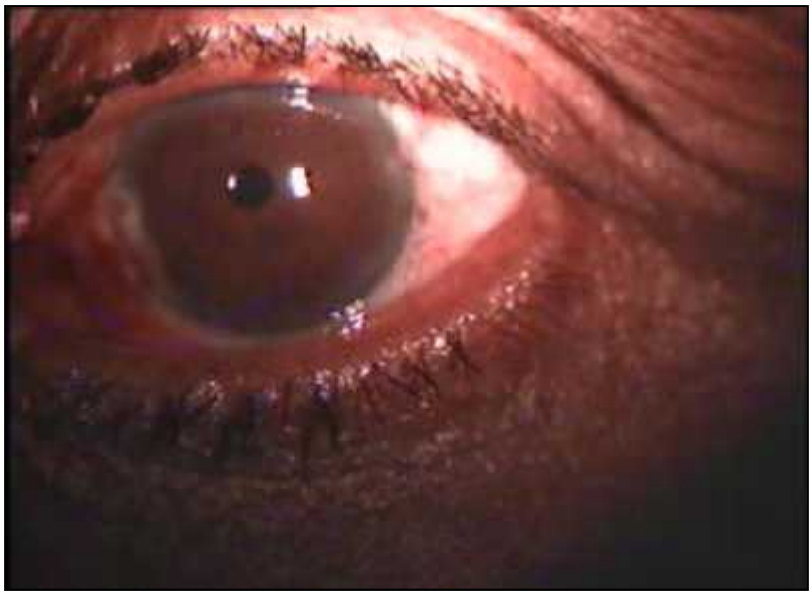
Day 15

Day 30

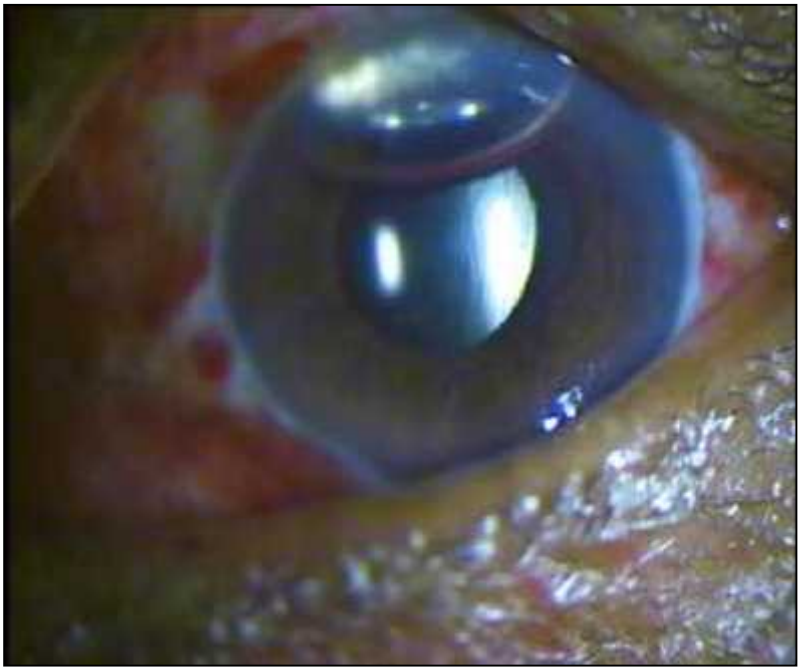
Days	**Vision**			**Fundus**
	Dv	**PH**	**Nv**	
1				
7				
15				
30				

REFRACTION:

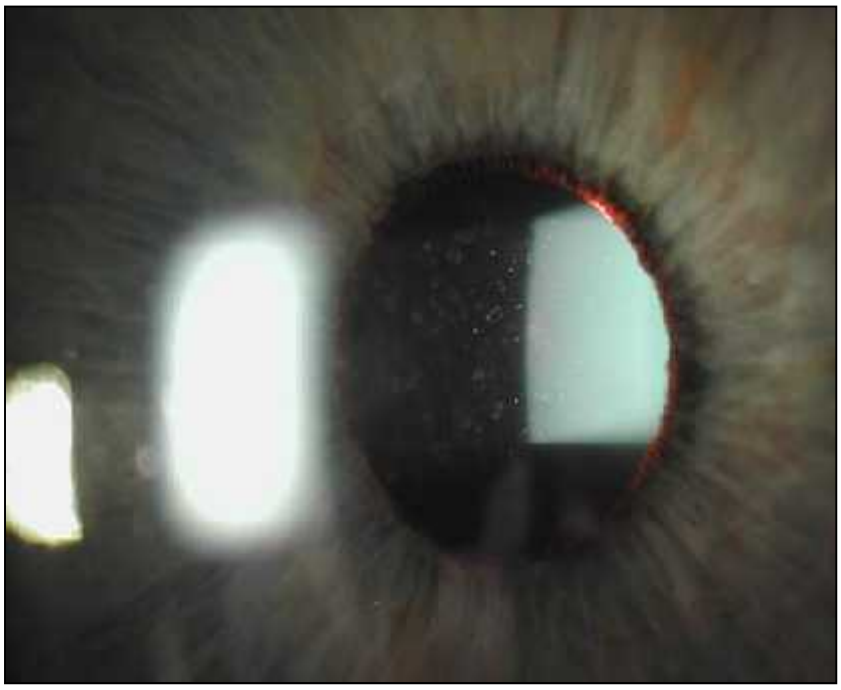
ANNEXURE – III: PHOTOGRAPHS



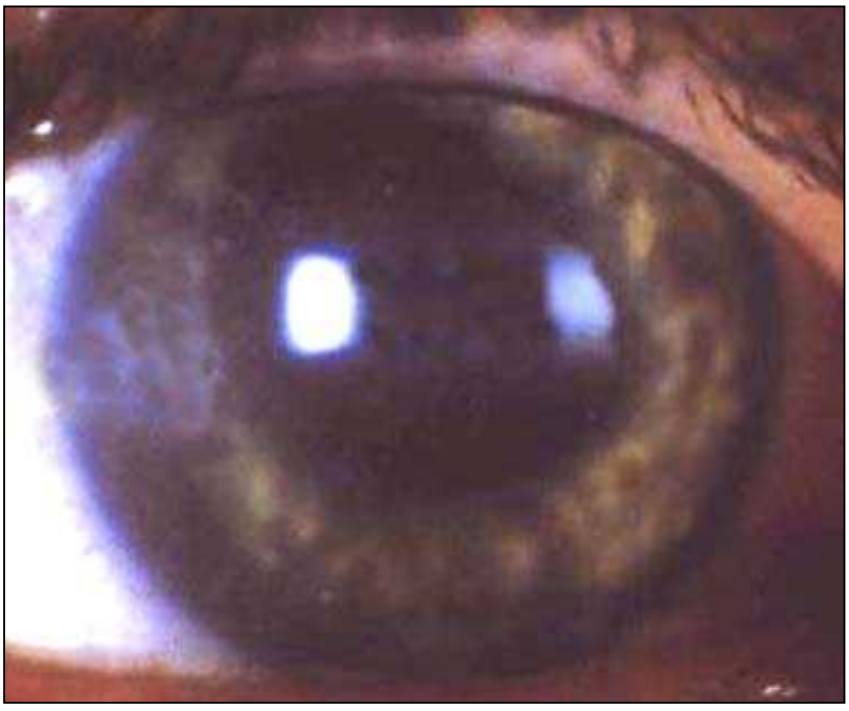
NO CONJUNCTIVAL AND CILIARY CONGESTION IN DIFLUPREDNATE GROUP BY DAY 15



PRESENCE OF MILD CONJUNCTIVAL AND CILIARY CONGESTION IN PREDNISOLONE GROUP ON DAY 15



PRESENCE OF ANTERIOR CHAMBER CELLS AND FLARE IN PREDNISOLONE GROUP ON DAY 15



NO ANTERIOR CHAMBER FLARE IN DIFLUPREDNATE GROUP BY DAY 15

ANNEXURE – IV: MASTER CHART

KEY TO MASTER CHART

AC : Anterior Chamber

BCVA : Best Corrected Visual Activity

C : Congestion

Cap : Capture

Cp : Complaints

Cm : Cortical Matter

Conj : Conjunctival

D : Diopters

Duro : Difluprednate

Pred : Prednisolone

DV : Distant Vision

DOV : Diminision of vision

Dt :Date

Fd : Fundus

G : Gradual

IO : IntraOperative

I.P.No : In patient number

I.O.P : Intraocular pressure

LE : Left eye

op : Operative

PCO : Posterior capsular opacification

PD : Pigment Deposit

PH : Pinhole

PL : Painless

Pp : Pupillary

Pr : Progressive

RE : Right eye

SHMC : Senile hypermature cataract

SIMC : Senile immature cataract

SK : Striate keratopathy

SNC : Senile nuclear cataract

SMC : Senile mature cataract

SPSC : Senile posterior sub capsular cataract

Srg : Surgery

Sup : Superiorly

Seg : Segment

Grade of inflammation

Lid edema : Grade 0-None, Grade 1-Mild, Grade 2- Moderate, Grade3-Severe.

Conjunctival congestion: Grade 0-None, Grade 1-Mild (some vessels injected), Grade 2- Moderate (diffuse injection), Grade 3-Severe (intense injection).

Ciliary congestion: Grade 0-None, Grade1-Mild (some vessels injected).Grade 2-Moderate (diffuse injection), Grade 3-Severe (intense injection).

Corneal edema: Grade 0-None, Grade 1-Mild, Grade 2-Moderate, Grade 3-Severe.

Anterior Chamber Flare: Grade 0- Absent, Grade1-Mild (barely detected), Grade 2-Moderate (iris and lens details seen), Grade 3-Severe (iris and lens details not seen).

Anterior Chamber Cells: Grade 0-Absent, Grade 1-5 to 10cells,

Grade 2-11 to 20 cells, Grade 3-20 to 50 cells.

Vitreous Haze: Grade 0- Absent, Grade 1-Few scattered fine and coarse opacities (Fundus seen clearly), Grade2-Scattered fine and coarse opacities but fundus somewhat obscured, Grade3- many opacities with marked blurring of the fundus.

MASTER CHART

S.No	Name	I.P.No.	Age	Sex	Diagnosis	Eye	Dt of srg	Gp	Preop.Evaluation: Cp G/Pr/PI	DOV Duration	Ant seg	Base line IOP	A scan	Type of Srg	Io Cp
1	chandrawwa		60	F	SPSC	RE	19.1.11	DURO	+	6 months	N	17.3	25 D	SICS WITHPCIOL	Nil
2	tengawwa	413093	70	M	SMC	RE	19.1.11	duro	+	1 yr	N	17.3	23.0 D	SICS WITHPCIOL	Nil
3	leela desai	413812	65	F	SIMC	LE	24.1.11	pred	+	6 months	N	17.3	22.0 D	SICS WITHPCIOL	Nil
4	yellubai	418127	60	F	SMC	RE	24.1.11	pred	+	1 yr	N	15.9	21.5 D	SICS WITHPCIOL	Nil
5	yashoda	418521	74	M	SIMC	LE	2.2.11	pred	+	2 months	N	14.6	22 D	SICS WITHPCIOL	Nil
6	leela pujari	423675	60	F	SIMC	RE	2.2.11	duro	+	3 yrs	N	17.3	26 D	SICS WITHPCIOL	Nil
7	parvatawwa	417656	60	F	SNC	LE	7.2.11	duro	+	1 yr	N	14.6	25 D	SICS WITHPCIOL	Nil
8	basavraj pujari	428675	55	F	SMC	LE	16.2.11	pred	+	3 months	N	14.6	22 D	SICS WITHPCIOL	Nil
9	sheshappa malage	168432	55	M	SIMC	LE	16.2.11	pred	+	4 months	N	14.6	21 D	SICS WITHPCIOL	Nil
10	sugandha patil	401383	65	M	SMC	RE	21.2.11	pred	+	3 months	N	12.2	25.5 D	SICS WITHPCIOL	Nil
11	yallawwa banaj	420693	56	M	SMC	LE	21.2.11	pred	+	6 months	N	12.2	21.5 D	SICS WITHPCIOL	Nil
12	kasturi ingale	453308	65	F	SMC	LE	28.2.11	pred	+	1 yr	N	17.3	25 D	SICS WITHPCIOL	Nil
13	channawwa	401384	50	F	SHMC	RE	28.2,11	DURO	+	6 months	N	15.9	25 D	SICS WITHPCIOL	Nil
14	satyappa	401356	70	F	SMC	LE	2.3.11	pred	+	6 months	N	15.9	21.5 D	SICS WITHPCIOL	Nil
15	balram	420307	63	F	SHMC	RE	7.3.11	pred	+	6 months	N	14.6	21.5 D	SICS WITHPCIOL	Nil
16	maruti patil	450678	70	F	SIMC	LE	7.3.11	pred	+	6 months	N	12.2	20D	SICS WITHPCIOL	Nil
17	mangala kumbhar	453134	58	M	SMC	RE	14.3.11	pred	+	6 months	N	17.3	22 D	SICS WITHPCIOL	Nil
18	ningappa patil	401630	56	M	SPSC	LE	16.3.11	DURO	+	1 yr	N	12.2	22 D	SICS WITHPCIOL	Nil
19	subhadra	443695	73	M	SMC	RE	16.3.11	pred	+	1 yr	N	14.6	21 D	SICS WITHPCIOL	Nil
20	rama	1833092	65	M	SMC	LE	21.3.11	pred	+	1 yr	N	13.4	23.5 D	SICS WITHPCIOL	Nil
21	parvati	444063	60	M	SMC	RE	21.3.11	DURO	+	6 months	N	13.4	22 D	SICS WITHPCIOL	Nil
22	tulsabai	453318	50	F	SIMC	RE	23.3.11	pred	+	1 yr	N	12.2	23.5 D	SICS WITHPCIOL	Nil
23	shivappa	443704	70	M	SIMC	LE	23.3.11	pred	+	6 months	N	15.9	24 D	SICS WITHPCIOL	Nil
24	madiwalappa	418144	55	F	SIMC	LE	30.3.11	DURO	+	8 months	N	14.6	21.5 D	SICS WITHPCIOL	Nil
25	basavraj talwar	406839	50	F	SPSC	LE	30.3.11	DURO	+	2 months	N	15.9	9.50 D	SICS WITHPCIOL	Nil
26	savitri	418140	65	M	SIMC	LE	6.4.11	pred	+	6 months	N	17.3	22 D	SICS WITHPCIOL	Nil
27	ramappa	452415	75	M	SNC	RE	13.4.11	pred	+	6 months	N	17.3	22 D	SICS WITHPCIOL	Nil
28	vittal patil	453479	68	M	SMC	RE	20.4.11	DURO	+	6 months	N	14.6	+ 21.0 D	SICS WITHPCIOL	Nil
29	dareppa	420691	50	F	SIMC	LE	20.4.11	DURO	+	6 months	N	15.9	24.0 D	SICS WITHPCIOL	Nil
30	gourawwa	420692	60	F	SIMC	RE	25.4.11	pred	+	4 months	N	17.3	+ 20.0 D	SICS WITHPCIOL	Nil
31	krishna mugale	450976	60	M	SIMC	LE	27.4.11	pred	+	6 months	N	14.6	+ 20D	SICS WITHPCIOL	Nil
32	shantawwaB	413815	50	F	SIMC	RE	4.5.11	DURO	+	1 yr	N	17.3	22 D	SICS WITHPCIOL	Nil
33	basappa shinde	432189	50	F	SIMC	RE	9.5.11	DURO	+	6 months	N	15.9	22 D	SICS WITHPCIOL	Nil
34	nazir ahmed	418551	61	M	SIMC	RE	11.5.11	pred	+	6 months	N	14.6	21.5 D	SICS WITHPCIOL	Nil
35	rayawwa	420690	60	F	SMC	LE	16.5.11	DURO	+	8 m	N	17.3	21.5	SICS WITHPCIOL	Nil
36	YALLAWWA J	420696	50	F	SNC	RE	23.5.11	pred	+	6 months	N	17.3	21.5 D	SICS WITHPCIOL	Nil
37	suresh	439889	50	M	SIMC	LE	25.5.11	DURO	+	6 months	N	17.3	20 D	SICS WITHPCIOL	Nil
38	shivappa	4033211	65	M	SIMC	RE	30.5.11	DURO	+	1 yr	N	17.3	22 D	SICS WITHPCIOL	Nil
39	saroja	453543	65	M	SIMC	LE	30.5.11	pred	+	6 months	N	17.3	21.5 D	SICS WITHPCIOL	Nil
40	sonawwa	456786	60	M	SIMC	RE	1.6.11	DURO	+	1 yr	N	12.2	22 D	SICS WITHPCIOL	Nil
41	kallawwa	432678	71	M	SIMC	LE	8.6.11	pred	+	6 months	N	17.3	22 D	SICS WITHPCIOL	Nil
42	kallappa	432176	60	F	SMC	RE	15.6.11	pred	+	8 months	N	17.3	23 D	SICS WITHPCIOL	Nil
43	rudrappa	478695	50	M	SNC	RE	15.6.11	DURO	+	6 months	N	15.9	22 D	SICS WITHPCIOL	Nil
44	yashoda	435393	75	F	SMC	RE	20.6.11	duro	+	1 yr	N	14.6	22 D	SICS WITHPCIOL	Nil
45	nagawwa	447338	50	F	SIMC	RE	20.6.11	duro	+	8 months	N	13.4	24 D	SICS WITHPCIOL	Nil
46	shevanti	446702	50	M	SIMC	LE	27.6.11	pred	+	6 months	N	15.9	24.5 D	SICS WITHPCIOL	Nil
47	ambakka	447329	65	M	SMC	RE	27.6.11	pred	+	8 months	N	12.2	21 D	SICS WITHPCIOL	Nil
48	iravva	424613	60	F	SIMC	RE	1.7.11	duro	+	8 months	N	14.6	23.5 D	SICS WITHPCIOL	Nil
49	Gangappa	424628	50	M	SHMC	RE	1.7.11	pred	+	6 months	N	13.6	23 D	SICS WITHPCIOL	Nil
50	drupata	425535	60	F	SMC	LE	6.7.11	pred	+	1 yr	N	14.6	23 D	SICS WITHPCIOL	Nil
51	Kallappa R.C.	425891	63	M	SHMC	RE	8.7.11	pred	+	6 months	N	14.6		SICS WITHPCIOL	Nil
52	janabai	425870	60	M	SIMC	RE	8.7.11	duro	+	4 months	N	15.9	23 D	SICS WITHPCIOL	Nil
53	rachayya	425875	51	M	SMC	LE	8.7.11	duro	+	6 months	N	15.9	23.0 D	SICS WITHPCIOL	Nil
54	mallawwa	425877	60	F	SIMC	RE	8.7.11	pred	+	2 months	N	15.9	21 D	SICS WITHPCIOL	Nil
55	satyawwa	425903	72	M	SNC	RE	8.7.11	pred	+	1 yr	N	15.9	21 D	SICS WITHPCIOL	Nil
56	ratnabai	426157	55	M	SIMC	RE	11.7.11	duro	+	8 months	N	17.3	20.5 D	SICS WITHPCIOL	Nil
57	bhojawwa	426159	50	F	SIMC	RE	11.7.11	duro	+	8 months	N	14.6	22 D	SICS WITHPCIOL	Nil
58	basavanth	426153	50	M	SIMC	LE	11.7.11	pred	+	6 months	N	17.3	21 D	SICS WITHPCIOL	Nil
59	prakash	426646	60	F	SIMC	RE	13.7.11	duro	+	3 months	N	15.9	24 D	SICS WITHPCIOL	Nil
60	lokawwa	426608	60	M	SHMC	RE	13.7.11	duro	+	1 yr	N	15.9	22 D	SICS WITHPCIOL	Nil
61	annappa	427033	55	M	SMC	LE	15.7.11	pred	+	6 months	N	13.4	21.6 D	SICS WITHPCIOL	Nil
62	sonabai	426981	50	M	SIMC	RE	15.7.11	pred	+	6 months	N	13.4	22 D	SICS WITHPCIOL	Nil
63	amrut	427785	60	F	SIMC	LE	20.7.11	duro		4 months	N	17.3	22 D	SICS WITHPCIOL	Nil
64	basappa	427816	62	M	SNC	LE	20.7.11	duro	+	6 months	N	17.3	24.5 D	SICS WITHPCIOL	Nil
65	dareppa	427836	72	M	SIMC	RE	20.7.11	pred	+	4 months	N	15.9	23 D	SICS WITHPCIOL	Nil
66	siddappa	427820	60	M	SIMC	RE	25.7.11	pred	+	8 months	N	14.6	25 D	SICS WITHPCIOL	Nil
67	basavani	427834	60	F	SIMC	RE	25.7.11	duro	+	4 months	N	12.2	23.5 D	SICS WITHPCIOL	Nil
68	shanta	427772	60	M	SHMC	RE	29.7.11	pred	+	1 yr	N	18.9	23 D	SICS WITHPCIOL	Nil
69	gadigewwa	427776	62	F	SMC	RE	29.7.11	duro	+	6 months	N	14.6	22 D	SICS WITHPCIOL	Nil
70	fakirawwa	427777	60	F	SIMC	LE	5.8.11	pred	+	6 months	N	17.3	22 D	SICS WITHPCIOL	Nil
71	gangawwa	427663	50	M	SNC	RE	10.8.11	pred	+	8 months	N	17.3	22 D	SICS WITHPCIOL	Nil
72	anandi	428508	52	M	SIMC	RE	9.9.11	duro	+	8 months	N	17.3	22 D	SICS WITHPCIOL	Nil

Post Operative Evaluation

Grades of Inflammation

S.No	Pain Day 1	Pain Day 7	Pain Day 15	Pain Day 30	Watering Day 1	Watering Day 7	Watering Day 15	Watering Day 30	Lid Edema Day 1	Lid Edema Day 7	Lid Edema Day 15	Lid Edema Day 30	Conj C Day 1	Conj C Day 7	Conj C Day 15	Conj C Day 30	Ciliary C Day 1	Ciliary C Day 7	Ciliary C Day 15	Ciliary C Day 30	Corneal edema Day 1	Corneal edema Day 7	Corneal edema Day 15	Corneal edema Day 30	AC Flare Day 1	AC Flare Day 7	AC Flare Day 15	AC Flare Day 30	AC cells Day 1	AC cells Day 7	AC cells Day 15	AC cells Day 30	Pupils Day 1	Pupils Day 7	Pupils (Synechiae +/-) Day 15	Pupils Day 30	Vitreous Haze Day 1	Vitreous Haze Day 7	Vitreous Haze Day 15	Vitreous Haze Day 30	Total score Day 1	Total score Day 7	Total score Day 15	Total score Day 30
1	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	1	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	1	0	0
2	+	-	-	-	+	-	-	-	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	1	0	0	0	0	0	0	-	-	-	-	0	0	0	0	5	1	0	0
3	-	-	-	-	-	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	2	1	1	0	1	1	0	0	-	-	Pp Cap	Pp Cap	0	0	0	0	6	3	1	1
4	-	-	-	-	-	-	-	-	0	0	0	0	1	1	0	0	1	1	0	0	0	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	4	4	1	0
5	+	-	-	-	+	-	-	-	1	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	6	1	0	0
6	+	-	-	-	+	-	-		0	0	0	0	1	1	0	0	1	0	0	0	1, Mild SK +	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	1	1	0
7	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	6	1	1	0
8	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0	3	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	1	0	0
9	+	+	-	-	+	+	-	-	0	0	0	0	1	0	0	0	1	0	0	0	2, SK +	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	1	0	0	0	7	0	0	0
10	+	+	+		+	+	+		0	0	0		1	1	1		1	1	1		1	1	1		2	2	2		1	1	1		-	-	-	-	0	0	0		6	6	6	
11	+	-	-	-	+	-	-	-	1	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	6	1	0	0
12	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1, Mild SK +	0	0	0	0	2	0	0	2	0	0	0	-	-	-	-	2	0	0	0	9	1	0	0
13	+	-	-	-	+	-	-	-	1	0	0	0	1	1	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	1	0	0	-	-	-	-	0	0	0	0	6	3	0	0
14	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1, Mild SK +	0	0	0	2	1	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	2	0	0
15	+	-	-	-	+	-	-	-	0	0	0	0	1	1	1	0	1	1	1	0	1, Mild SK +	1	0	0	1	1	1	0	1	1	0	0	-	-	-	-	0	0	0	0	5	5	3	0
16	+	-	-	-	+	-	-	-	0	0	0	0	2	1	0	0	2	0	0	0	2, SK +	1	1	0	0	2	0	0	1	0	0	0	-	-	-	-	1	0	0	0	10	2	1	0
17	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	1	1	0	3	1	0	0	-	-	-	-	0	0	0	0	7	2	1	0
18	-	-	-	-	-	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	0	0	0
19	-	-	-	-	-	-	-	-	0	0	0	0	2	1	0	0	2	1	0	0	2, SK +	1	1	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	10	3	1	0
20	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	2	0	0	0	9	0	0	0
21	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	1	0	0
22	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	0	0	0
23	-	-	-	-	-	-	-	-	0	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	0	0	0
24	+	-	-	-	+	-	-	-	1	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	6	3	0	0
25	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	2	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	7	3	0	0
26	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	8	0	0	0
27	-	-	-	-	-	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	2, SK +	1	1	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	7	1	1	0
28	+	+			+	+			0	0			1	2			1	1			1	0			2	2			1	1			-	-	-	-	1	1			7	7		
29	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	0	0	0
30	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	2, SK +	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	8	1	0	0
31	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	0	0	0
32	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	2	0	0
33	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	0	0	0
34	+	-	-	-	-	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	2, SK +	0	0	0	1	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	0	0	0
35	-	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	0	0	0
36	+	-	-	-	+	-	-	-	0	0	0	0	1	1	1	0	1	0	0	0	1	0	0	0	2	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	7	1	1	0
37	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	6	3	0	0
38	+	-	-	-	+	-	-	-	1	0	0	0	2	1	1	0	1	0	0	0	1	1	1	0	2	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	10	4	2	0
39	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	2, SK +	1	0	0	2	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	7	1	0	0
40	-	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	0	0	0
41	+	-	-	-	+	-	-	-	1	0	0	0	1	0	0	0	1	1	0	0	0	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	4	0	0
42	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	1	0	0	2	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	6	4	1	0
43	-	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	0	0	0
44	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	1	0	0	1	0	0	0	1	0	0	0	0	0	0	0	-	-	-	-	0	0	0	0	5	2	1	0
45	+	+	-	-	+	+	-	-	0	0	0	0	1	1	0	0	1	1	0	0	1	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	4	0	0
46	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	3	2	1	0	2	1	0	0	-	-	-	-	0	0	0	0	8	1	0	0
47	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	3	1	1	0	2	1	0	0	-	-	-	-	0	0	0	0	8	1	0	0
48	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	2	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	7	1	0	0
49	-	-	-	-	-	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	0	0	0
50	-	-	-	-	-	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	0	0	0
51	+	-	-	-	+	-	-	-	1	0	0	0	1	0	0	0	1	0	0	0	2, SK +	0	0	0	2	1	0	0	2	1	0	0	-	-	-	-	1	0	0	0	9	1	0	0
52	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	4	1	0
53	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1, Mild SK +	1	0	0	1	1	0	0	1	0	0	0	-	-	-	-	0	0	0	0	4	3	1	1
54	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	2	0	0
55	+	-	-	-	+	+	-	-	0	0	0	0	1	0	0	0	1	0	0	0	2, SK +	0	0	0	2	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	8	0	0	0
56	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	1	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	3	0	0
57	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1,sk	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	6	0	0	0
58	+	+	-	-	+	-	-	-	0	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	2	0	0	0	1	0	0	0	-	-	-	-	1	0	0	0	8	0	0	0
59	+	+	-	-	+	-	-	-	0	0	0	0	1	1	1	0	1	1	0	0	1	0	0	0	1	1	0	0	1	0	0	0	-	-	-	-	0	0	0	0	6	4	1	0
60	+	-	-	-	+	+	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	3	0	0
61	+	+	-	-	+	+	-	-	0	0	0	0	2	0	0	0	1	0	0	0	1	0	0	0	2	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	8	0	0	0
62	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	1	1	0
63	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	3	0	0
64	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	0	0	0	2	0	0	0	-	-	-	-	0	0	0	0	7	0	0	0
65	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	1	0	0	1	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	3	0	0
66	+	-	-	-	+	-	-	-	0	0	0	0	1	1	1	0	1	0	0	0	1	0	0	0	1	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	6	3	0	0
67	+	-	-	-	+	-	-	-	0	0	0	0	1	1	1	0	1	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0	-	-	-	-	0	0	0	0	4	1	0	0
68	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	2, SK +	0	0	0	2	1	0	0	2	1	0	0	-	-	-	-	1	0	0	0	7	1	0	0
69	+	-	-	-	+	+	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	2	0	0	0	-	-	-	-	1	0	0	0	7	0	0	0
70	+	-	-	-	+	-	-	-	0	0	0	0	2	1	1	0	2	1	0	0	1	0	0	0	1	0	0	0	1	0	0	0	-	-	-	-	1	0	0	0	8	2	1	0
71	+	+	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0	2	1	1	0	1	1	0	0	-	-	-	-	1	0	0	0	7	2	2	0
72	+	+	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	1	0	0	0	0	0	0	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	4	2	0

Post Operative Vision

S.No	Day 1 DV	Day 1 PH	Day 7 DV	Day 7 PH	Day 15 DV	Day 15 PH	Day 30 DV	Day 30 PH	BCVA	Fd (30 days)	IOP at 6 weeks	Other findings
1	Cf 5mt	6/24	6/36	6/12	6/36	6/12	6/36	6/12	6/12	WNL	20	-
2	6/60	6/80	6/36	6/18	6/36	6/12	6/36	6/12	6/9(p)	WNL	18	PD on lens from day7
3	6/60	6/36	6/36	6/24	6/36	6/24	6/24	6/12	6/9	WNL	18	PD on lens from day 15
4	6/60	6/24	6/36	6/24	6/24	6/9	6/18	6/19	6/9 (p)	WNL	16	-
5	6/60	6/36	6/18	6/12	6/12	6/9	6/12	6/9	6/9(p)	WNL	16	-
6	CF 2mt	6/60	Cf 4mt	6/60	Cf 4mt	6/60	CF4mt	6/36	6/12	WNL	18	PCO+ PD on lens from day 7
7	6/36	6/24	6/24	6/18	6/24	6/18	6/18	6/19	6/6 (p)	WNL	18	PCO + PD on lens day 1
8	6/36	6/24	6/12	6/9	6/12	6/9	6/9	6/9	6/6(p)	WNL	18	-
9	CF 5mt	-	6/36	6/24	6/18	6/9	6/12	6/9	6/9	WNL	18	-
10	6/60	6/36	6/12	6/9	6/12	6/9				WNL		Pt shifted to steriods on day 15
11	6/60	6/36	6/36	6/18	6/12	6/9	6/12	6/9	6/9	WNL	18	-
12	CF 3 mt	-	6/60	6/24	6/24	6/18	6/18	6/12	6/9 (p)	WNL	20	PD on lens from Day 7
13	6/36	6/24	6/24	6/9	6/18	6/9	6/12	6/9	6/9	WNL	16	-
14	CF5mt	6/60	6/24	6/18	6/24	6/18	6/24	6/12	6/9	WNL	16	Sup Cm, PD on lens +
15	6/60	6/18	6/36	6/18	6/24	6/9	6/12	6/9	6/9(p)	WNL	16	-
16	CF 2mt	-	6/24	6/18	6/18	6/12	6/18	6/12	6/9	WNL	16	-
17	6/24	6/18	6/18	6/12	6/18	6/12	6/18	6/9	6/9	WNL	18	PD on lens from Day 7
18	6/60	6/36	6/36	6/24	6/36	6/24	6/24	6/9	6/6	WNL	14	-
19	CF 4mt	-	6/60	6/36	6/36	6/24	6/24	6/12	6/9(p)	WNL	18	Cm, PD on lens from Day 7
20	CF 5 mt	-	6/24	6/12	6/12	6/9	6/12	6/9	6/6	WNL	16	-
21	6/18	6/12	6/12	6/9	6/12	6/9	6/12	6/9	6/9	WNL	16	Sup Cm, PD on lens from day 7
22	6/60	6/24	6/24	6/18	6/18	6/12	6/18	6/9	6/9	WNL	14	-
23	CF 4mt	6/60	6/60	6/36	6/36	6/18	6/36	6/12	6/12	WNL	16	PD on lens from Day 7
24	CF 5mt	6/60	6/12	6/9	6/12	6/9	6/9	6/9	6/9	WNL	16	PD on lens from Day 7, PCO +
25	CF 4mt	6/60	6/36	6/24	6/24	6/12	6/12	6/9	6/6	WNL	16	-
26	6/60	6/36	6/36	6/12	6/36	6/12	6/18	6/9	6/6	WNL	20	-
27	CF 3 mt	6/60	6/60	6/24	6/24	6/12	6/24	6/9	6/9	WNL	18	-
28	6/60	6/24	6/60	6/24						WNL		Pt shifted to steriods on day 7
29	6/36	6/24	6/24	6/12	6/24	6/12	6/12	6/9	6/9	WNL	18	PD from Day 7 on lens
30	6/60	6/36	6/36	6/24	6/36	6/24	6/24	6/12	6/9	WNL	18	PD from Day 7 on lens
31	6/60	6/36	6/36	6/12	6/36	6/12	6/12	6/9	6/9	WNL	16	-
32	6/60	6/24	6/36	6/24	6/24	6/18	6/12	6/9	6/9	WNL	18	-
33	6/60	6/24	6/36	6/24	6/36	6/24	6/24	6/12	6/6	WNL	16	PD from Day 7 on lens
34	CF 4mt	6/60	6/60	6/36	6/36	6/24	6/36	6/12	6/9	WNL	18	PCO+
35	6/36	6/24	6/24	6/18	6/24	6/18	6/24	6/9	6/6	WNL	18	PD on lens from Day 7, PCO +
36	6/24	6/18	6/24	6/12	6/12	6/12	6/12	6/9	6/9(p)	WNL	18	-
37	6/60	6/12	6/60	6/12	6/18	6/9	6/12	6/9	6/6	WNL	18	PD on lens from Day 7
38	6/60	6/36	6/60	6/36	6/60	6/24	6/24	6/12	6/6	WNL	18	PD on lens from Day 7
39	6/60	6/36	6/36	6/24	6/24	6/18	6/24	6/18	6/6	WNL	16	-
40	6/60	6/24	6/24	6/12	6/24	6/12	6/12	6/9	6/9(p)	WNL	14	-
41	6/60	6/36	6/60	6/24	6/24	6/18	6/18	6/9	6/9	WNL	18	PD on lens from Day 7
42	6/24	6/18	6/24	6/12	6/12	6/9	6/12	6/9	6/9(p)	WNL	18	-
43	6/60	6/36	6/36	6/24	6/36	6/24	6/24	6/12	6/9	WNL	18	-
44	6/18	6/12	6/18	6/12	6/12	6/9	6/12	6/9	6/9	WNL	16	PD on lens from Day 7
45	6/60	6/36	6/36	6/24	6/36	6/18	6/24	6/12	6/9	WNL	14	-
46	6/60	6/36	6/24	6/18	6/18	6/12	6/18	6/12	6/9	WNL	18	Sup Cm
47	6/60	6/36	6/24	6/18	6/18	6/12	6/18	6/9	6/9(p)	WNL	16	-
48	6/24	6/18	6/18	6/12	6/18	6/12	6/12	6/9	6/6(p)	WNL	16	-
49	6/36	6/18	6/24	6/18	6/24	6/12	6/18	6/9	6/6(p)	WNL	18	-
50	6/60	6/36	6/60	6/24	6/36	6/18	6/18	6/12	6/9	WNL	16	-
51	6/60	6/18	6/36	6/18	6/24	6/9	6/12	6/9	6/9	WNL	18	PD on lens from Day 7
52	6/60	6/36	6/24	6/18	6/24	6/18	6/18	6/12	6/12(p)	WNL	16	PD on lens from Day 7
53	6/60	6/36	6/36	6/18	6/24	6/18	6/24	6/12	6/9	WNL	16	-
54	6/24	6/12	6/18	6/12	6/12	6/9	6/12	6/9	6/6(p)	WNL	16	-
55	6/36	6/24	6/36	6/18	6/18	6/12	6/18	6/12	6/9	WNL	16	-
56	CF 5mt	6/60	6/60	6/36	6/36	6/18	6/18	6/12	6/9(p)	WNL	18	PD on lens from Day 7
57	6/24	6/18	6/18	6/12	6/18	6/12	6/18	6/9	6/9(p)	WNL	16	-
58	6/24	6/18	6/24	6/12	6/12	6/9	6/12	6/9	6/6(p)	WNL	20	-
59	CF 5mt	6/60	6/36	6/24	6/36	6/18	6/24	6/12	6/9	WNL	16	-
60	6/36	6/24	6/24	6/9	6/18	6/9	6/12	6/9	6/9	WNL	16	-
61	6/36	6/18	6/24	6/18	6/24	6/12	6/12	6/9	6/6(p)	WNL	16	-
62	CF 5mt	6/60	6/24	6/18	6/18	6/12	6/18	6/9	6/9	WNL	14	PD on lens from Day 7
63	6/18	6/12	6/18	6/12	6/12	6/9	6/12	6/9	6/6(p)	WNL	18	Cm Sup
64	6/60	6/36	6/60	6/36	6/36	6/24	6/36	6/12	6/12	WNL	20	-
65	CF 5mt	6/60	6/60	6/36	6/36	6/24	6/24	6/12	6/9	WNL	16	PD on lens from Day 7
66	6/18	6/12	6/18	6/12	6/12	6/9	6/12	6/9	6/6(p)	WNL	16	PD on lens from Day 7
67	6/60	6/24	6/36	6/24	6/12	6/9	6/12	6/9	6/9	WNL	14	PD on lens from Day 7
68	CF 4mt	6/60	6/60	6/36	6/36	6/24	6/24	6/18	6/9	WNL	20	PCO +
69	6/60	6/36	6/36	6/18	6/24	6/18	6/18	6/9	6/6	WNL	18	-
70	6/24	6/18	6/18	6/12	6/24	6/9	6/12	6/9	6/9	WNL	18	-
71	6/60	6/36	6/36	6/24	6/24	6/18	6/24	6/12	6/12	WNL	18	PD on lens from Day 7
72	CF 4mt	6/60	6/60	6/36	6/36	6/18	6/36	6/18	6/12	WNL	16	PD on lens from Day 7

S.No	Name	I.P.No.	Age	Sex	Diagnosis	Eye	Dt of srg	Gp	Preop.Evaluation: Cp: G/Pr/Pl	DOV: Duration	Ant seg	Base line IOP	A scan	Type of Srg	Io Cp
73	gurulingawwa	428505	60	F	SHMC	LE	26.11.11	duro	+	6 months	N	14.6	21 D	SICSWITH PCIOL	Nil
74	isac	428514	50	M	SMC	RE	26.11.11	pred	+	1 yr	N	17.3	20 D	SICSWITH PCIOL	Nil
75	irayya	428504	80	M	SIMC	LE	26.11.11	pred	+	8 months	N	15.9	21 D	SICSWITH PCIOL	Nil
76	yallawwa	429328	70	F	SIMC	LE	26.11.11	duro	+	6 months	N	12.2	22 D	SICSWITH PCIOL	Nil
77	abdul	426751	60	M	SIMC	RE	28.11.11	duro	+		N	14.6	20 D	SICSWITH PCIOL	Nil
78	pundalik	430285	60	M	SIMC	LE	28.11.11	pred	+	4 months	N	15.9	20 D	SICSWITH PCIOL	Nil
79	shonakka	431012	68	M	SIMC	LE	1.12.11	duro	+	6 months	N	17.3	21.5 D	SICSWITH PCIOL	Nil
80	bhimappa	431013	55	F	SIMC	LE	3.12.11	pred	+	1 yr	N	15.9	22.5 D	SICSWITH PCIOL	Nil
81	shahajadbi	449678	65	M	SNC	RE	6.12.11	duro	+	6 months	N	15.9	22.5 D	SICSWITH PCIOL	Nil
82	Bhimappa	445324	50	M	SNC	LE	6.12.11	pred	+	8 months	N	17.3	23 D	SICSWITH PCIOL	Nil
83	Honappa K	465321	60	M	SMC	LE	14.12.11	duro	+	6 months	N	15.9	23.5 D	SICSWITH PCIOL	Nil
84	Basvant D	478654	55	M	SMC	RE	14.12.11	duro	+	1 yr	N	13.4	23 D	SICSWITH PCIOL	Nil
85	Gangaram	423165	60	M	SPSC	RE	19.12.11	duro	+	4 months	N	17.3	21 D	SICSWITH PCIOL	Nil
86	Hussainbe	489765	68	F	SMC	RE	21.12.11	pred	+	8 months	N	14.6	23.5 D	SICSWITH PCIOL	Nil
87	Sumitra Patil	486754	65	F	SIMC	RE	21.12.11	duro	+	6 months	N	17.3	23.5 D	SICSWITH PCIOL	Nil
88	Veerabhadrappa	412687	60	M	SNC	LE	26.12.11	duro	+	8 months	N	12.2	21.5 D	SICSWITH PCIOL	Nil
89	Maruti H.D	409807	53	M	SMC	RE	26.12.11	duro	+	1 yr	N	17.3	22 D	SICSWITH PCIOL	Nil
90	Vithal	403487	59	M	SIMC	LE	28.12.11	pred	+	6 months	N	15.9	22 D	SICSWITH PCIOL	Nil
91	Hanmant I	402478	60	M	SMC	RE	28.12.11	duro	+	1 yr	N	13.4	20 D	SICSWITH PCIOL	Nil
92	Jadav	400987	65	M	SMC	LE	4.1.12	pred	+	1 yr	N	14.6	21.5 D	SICSWITH PCIOL	Nil
93	Pandurang	498076	65	M	SNC	RE	9.1.12	duro	+	4 months	N	17.3	21.5 D	SICSWITH PCIOL	Nil
94	Mahadev P	400076	60	M	SHMC	LE	9.1.12	duro	+	8 months	N	15.6	22 D	SICSWITH PCIOL	Nil
95	Mahantesh	411167	50	M	SHMC	RE	11.1.12	pred	+	1 yr	N	14.9	22.5 D	SICSWITH PCIOL	Nil
96	Sushila B	477743	61	F	SPSC	RE	16.1.12	duro	+	4 months	N	15.9	23.5 D	SICSWITH PCIOL	Nil
97	B. S. Menshe	400076	55	M	SMC	RE	16.1.12	duro	+	1 yr	N	14.6	21.5 D	SICSWITH PCIOL	Nil
98	Timawwa	438897	65	F	SNC	RE	18.1.12	pred	+	8 months	N	14.6	22 D	SICSWITH PCIOL	Nil
99	S. K. Patil	477798	60	M	SMC	RE	23.1.12	duro	+	6 months	N	17.3	22.5 D	SICSWITH PCIOL	Nil
100	Gopal S	455587	60	M	SPSC	LE	23.1.12	duro	+	4 months	N	15.9	21.5 D	SICSWITH PCIOL	Nil

Post Operative Evaluation — Grades of Inflammation

S.No	Pain Day 1	Pain Day 7	Pain Day 15	Pain Day 30	Watering Day 1	Watering Day 7	Watering Day 15	Watering Day 30	Lid Edema Day 1	Lid Edema Day 7	Lid Edema Day 15	Lid Edema Day 30	Conj C Day 1	Conj C Day 7	Conj C Day 15	Conj C Day 30	Ciliary C Day 1	Ciliary C Day 7	Ciliary C Day 15	Ciliary C Day 30	Corneal edema Day 1	Corneal edema Day 7	Corneal edema Day 15	Corneal edema Day 30
73	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0
74	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	1	0	0	1	0	0	0
75	+	-	-	-	+	+	-	-	0	0	0	0	1	0	0	0	1	0	0	0	2, SK +	0	0	0
76	+	-	-	-	+	-	-	-	0	0	0	0	2	1	0	0	1	0	0	0	1SK	0	0	0
77	+	+	-	-	+	+	-	-	1	0	0	0	1	1	0	0	1	1	0	0	1, Mild SK +	0	0	0
78	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	1	0	0	1	0	0	0
79	+	-	-	-	+	+	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1,SK	0	0	0
80	+	-	-	-	-	-	-	-	0	0	0	0	1	1	0	0	1	1	0	0	2, SK +	0	0	0
81	+	-	-	-	+	-	-	-	0	0	0	0	2	1	1	0	1	1	0	0	0	0	0	0
82	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0
83	+	-	-	-	+	-	-	-	0	0	0	0	1	1	1	0	2	1	0	0	0	0	0	0
84	+	+	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0
85	+	-	-	-	-	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0
86	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0
87	+	-	-	-	+	+	-	-	0	0	0	0	2	1	1	0	1	1	0	0	0	0	0	0
88	+	-	-	-	+	-	-	-	0	0	0	0	2	0	0	0	1	0	0	0	1	0	0	0
89	+	+	-	-	+	+	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0
90	+	-	-	-	+	-	-	-	0	0	0	0	2	1	1	0	1	0	0	0	1	0	0	0
91	+	-	-	-	+	-	-	-	0	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0
92	+	-	-	-	+	-	-	-	0	0	0	0	1	1	1	0	1	1	0	0	1	0	0	0
93	+	+	-	-	+	-	-	-	0	0	0	0	2	1	1	0	1	1	0	0	1	0	0	0
94	+	-	-	-	+	-	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0
95	+	-	-	-	+	+	-	-	0	0	0	0	1	1	1	0	1	0	0	0	1	0	0	0
96	+	-	-	-	+	+	-	-	0	0	0	0	1	1	1	0	1	0	0	0	0	0	0	0
97	+	-	-	-	+	+	-	-	0	0	0	0	2	1	0	0	1	0	0	0	0	0	0	0
98	+	+	-	-	+	+	-	-	0	0	0	0	1	1	0	0	1	0	0	0	1	0	0	0
99	+	+	-	-	+	+	-	-	0	0	0	0	1	1	1	0	1	1	0	0		0	0	0
100	+	-	-	-	+	+	-	-	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0

S.No	AC Flare Day 1	AC Flare Day 7	AC Flare Day 15	AC Flare Day 30	AC cells Day 1	AC cells Day 7	AC cells Day 15	AC cells Day 30	Pupils (Synechiae +/-) Day 1	Pupils Day 7	Pupils Day 15	Pupils Day 30	Vitreous Haze Day 1	Vitreous Haze Day 7	Vitreous Haze Day 15	Vitreous Haze Day 30	Total score Day 1	Total score Day 7	Total score Day 15	Total score Day 30
73	1	1	0	0	1	1	0	0	-	-	-	-	1	0	0	0	5	3	0	0
74	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	4	0	0
75	1	0	0	0	1	0	0	0	-	-	-	-	2	0	0	0	8	0	0	0
76	2	0	0	0	2	0	0	0	-	-	-	-	1	0	0	0	10	1	0	0
77	2	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	8	4	0	0
78	1	0	0	0	1	0	0	0	-	-	-	-	1	0	0	0	5	2	1	0
79	2	0	0	0	1	0	0	0	-	-	-	-	1	0	0	0	8	0	0	0
80	1	1	0	0	2	1	0	0	-	-	-	-	1	0	0	0	9	4	1	0
81	2	1	0	0	2	1	0	0	-	-	-	-	1	0	0	0	9	4	1	0
82	1	1	0	0	1	1	0	0	-	-	-	-	0	0	0	0	5	3	0	0
83	1	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	8	3	1	0
84	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	1	0	0
85	1	0	0	0	1	0	0	0	-	-	-	-	1	0	0	0	6	1	0	0
86	1	0	0	0	0	0	0	0	-	-	-	-	0	0	0	0	4	1	1	0
87	1	1	0	0	2	1	0	0	-	-	-	-	1	0	0	0	8	4	1	0
88	2	0	0	0	1	0	0	0	-	-	-	-	1	0	0	0	8	4	0	0
89	1	1	1	0	1	1	0	0	-	-	-	-	0	0	0	0	6	3	2	0
90	1	1	0	0	0	0	0	0	-	-	-	-	0	0	0	0	5	1	1	0
91	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	5	0	0	0
92	2	2	1	0	2	1	0	0	-	-	-	-	0	0	0	0	8	5	2	0
93	1	1	0	0	1	1	0	0	-	-	-	-	1	0	0	0	6	4	1	0
94	2	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	6	1	0	0
95	1	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	6	1	1	0
96	1	1	0	0	2	1	0	0	-	-	-	-	1	0	0	0	7	4	1	0
97	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	6	0	0	0
98	2	1	0	0	2	1	0	0	-	-	-	-	0	0	0	0	8	3	0	0
99	1	1	1	0	2	1	0	0	-	-	-	-	0	0	0	0	6	4	2	0
100	1	0	0	0	1	0	0	0	-	-	-	-	0	0	0	0	4	1	0	0

Post Operative Vision

S.No	Day 1 DV	Day 1 PH	Day 7 DV	Day 7 PH	Day 15 DV	Day 15 PH	Day 30 DV	Day 30 PH	BCVA	Fd (30 days)	IOP at 6 weeks	Other findings
73	6/60	6/36	6/36	6/18	6/18	6/12	6/18	6/9	6/9	WNL	16	-
74	6/60	6/24	6/24	6/18	6/24	6/9	6/12	6/9	6/9	WNL	18	-
75	CF 3mt	CF 5mt	6/60	6/36	6/36	6/18	6/18	6/12	6/9	WNL	18	PD on lens from Day 7
76	CF 2mt		6/24	6/18	6/24	6/12	6/24	6/12	6/12	WNL	16	-
77	6/60	6/36	6/36	6/24	6/36	6/24	6/36	6/18	6/9	WNL	16	PD on lens from Day 7
78	CF 4mt		6/36	6/12	6/24	6/12	6/24	6/12	6/9	WNL	16	-
79	CF 4mt	CF 5mt	6/60	6/36	6/36	6/24	6/12	6/9	6/9	WNL	18	-
80	CF 5mt		6/60	6/24	6/18	6/12	6/12	6/9	6/9	WNL	16	Pp Cap on day 15 on lens
81	CF 5mt		6/60	6/36	6/24	6/18	6/18	6/12	6/9	WNL	16	-
82	CF 3mt		6/60	6/36	6/24	6/12	6/12	6/9	6/9	WNL	18	PD on lens from Day 7
83	6/60	6/36	6/36	6/24	6/24	6/12	6/12	6/9	6/9	WNL	16	PD on lens from Day 7
84	6/36	6/24	6/36	6/12	6/24	6/12	6/12	6/9	6/9	WNL	14	-
85	CF 3mt		6/60	6/36	6/36	6/18	6/18	6/12	6/9	WNL	20	-
86	6/60	6/24	6/24	6/12	6/18	6/9	6/18	6/9	6/9	WNL	16	-
87	CF 5mt		6/60	6/36	6/36	6/18	6/18	6/12	6/9(p)	WNL	18	PD on lens from Day 7
88	6/24	6/18	6/18	6/12	6/18	6/12	6/12	6/9	6/6	WNL	16	-
89	CF 5mt	6/60	6/36	6/24	6/24	6/12	6/24	6/12	6/9(p)	WNL	18	PD on lens from Day 7
90	6/36	6/24	6/36	6/18	6/12	6/9	6/12	6/9	6/9	WNL	16	-
91	6/60	6/36	6/36	6/24	6/24	6/12	6/18	6/9	6/9	WNL	14	-
92	CF 5mt		6/60	6/36	6/36	6/18	6/18	6/12	6/9	WNL	16	PD on lens from Day 7
93	CF 4mt		6/36	6/24	6/24	6/18	6/18	6/9	6/9	WNL	18	
94	CF 4mt	6/60	6/60	6/36	6/36	6/24	6/24	6/18	6/12	WNL	18	PCO +
95	6/36	6/24	6/24	6/18	6/18	6/12	6/12	6/9	6/9	WNL	16	PD on lens from Day 7
96	CF 4mt		6/36	6/18	6/12	6/9	6/12	6/9	6/6(p)	WNL	16	-
97	6/60	6/24	6/24	6/12	6/24	6/18	6/18	6/9	6/9	WNL	16	-
98	6/60	6/36	6/36	6/24	6/24	6/12	6/12	6/9	6/9	WNL	16	PD on lens from Day 7
99	CF 5mt	6/60	6/36	6/24	6/24	6/12	6/24	6/12	6/12(p)	WNL	18	-
100	6/36	6/24	6/24	6/18	6/18	6/12	6/12	6/9	6/6	WNL	16	-