"COMPARISON OF PREOPERATIVE TOPICAL DEXAMETHASONE PHOSPHATE VERSUS KETOROLAC TROMETHAMINE IN MAINTAINING INTRAOPERATIVE MYDRIASIS DURING SMALL INCISION CATARACT SURGERY – A ONE YEAR RANDOMIZED CONTROL TRIAL AT KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI."

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ABSTRACT

BACKGROUND:

Cataract is the leading cause of impaired vision in the elderly population all over the world next only to refractive error. It is caused due to the opacification of the transparent lens of the eye. Senile cataracts can be a nuclear cataract, a cortical cataract or a posterior subcapsular cataract. Based on the cause, they can be complicated cataracts or uncomplicated cataracts.

Surgical treatment for senile cataracts includes an Extra Capsular Cataract Extraction(ECCE) in its modified advanced form called Small Incision Cataract Surgery(SICS) or Phacoemulsification surgery. Due to its various advantages like incision size, easy availability, technical compliance, cheaper cost etc , SICS is used more comfortably in treating senile cataracts especially in developing countries like India. In the procedure of SICS the delivery of the nucleus and cortical matter removal are the most significant steps that requires an adequate pupillary size for their uninterrupted complete removal. Maintaining adequate pupillary size is of utmost importance for a successful surgery.

Intraoperative miosisis thought to be caused by the Prostaglandins (PGs), Leukotrienes (LTs) and other inflammatory mediators released during surgical trauma. Currently, Topical Non Steroidal Anti-Inflammatory Drugs(NSAIDs) and Steroids are being used preoperatively in order to prevent the release of PGs and LTs and to maintain intraoperative mydriasis. The efficiency of these two classes of drugs needs to be compared so that the best drug can be given preoperatively.

AIM AND OBJECTIVE: To compare the efficacy of preoperative topical Dexamethasone phosphate(0.1%) and Ketorolac tromethamine (0.5%) in maintaining intraoperative mydriasis during Small Incision Cataract Surgery.

MATERIALS AND METHODS: Itwas a Randomized Control Trial of patients with uncomplicated senile cataract undergoing Small Incision Cataract Surgery. Age and sex matched patients who were eligible for the study were randomized into two groups- Group A had 90 subjects who received Dexamethasone phosphate 0.1% eye drops preoperatively and Group B had 90 subjects who received Ketorolac tromethamine 0.5% eye drops preoperatively. The pupillary diameters were measured at four intervals intraoperatively. The study population was selected from the patients attending Ophthalmology OPD at KLES Dr.Prabhakar Kore Hospital and Medical Research Centre. The study period was from January 2019 to December 2019.

RESULTS: A total of 180 patients were studied in two groups. The mean age of distribution in group A is 64.16 with a standard deviation of 8.05 and that of Group B is 64.47 with a standard deviation of 8.74. On comparing the interpupillary diameter(IPD) at different stages of the surgery between Group A and Group B, it was found that the mean IPD was almost equal. At the start of surgery(stage I), the mean IPD was 7.49 with SD of 0.55 in Group A and the mean IPD was 7.46 with SD of 0.61 in Group B. The p value was 0.6526. After the removal of lens nucleus (stage II) , the mean IPD was 6.65 with SD of 0.68 in Group A and the mean IPD was6.73 with SD of 0.77 in Group B. The p value was 0.4423. After cortical matter removal(stage III) , the mean IPD was 6.47 and SD was 0.73 in Group A and the mean IPD was 6.58 with SD of 0.83 in Group B. The p value was 0.3417. After implantation of Intra Ocular Lens(IOL)(stage IV) , the mean IPD was 6.38 with SD of 0.82 in Group A and the mean IPD was 6.69 with SD 0.91 in Group B. The p value was 0.0173. At stage 4

the p value was statistically significant as it is less than 0.05%. It was found that Ketorolac tromethamine 0.5% is maintaining intraoperative mydriasis better than Dexamethasone phosphate 0.1% especially after IOL implantation.

CONCLUSION: The study showed that both Dexamethasone phosphate 0.1% and Ketorolac tromethamine 0.5% are equivalent in maintaining mydrasis during SICS but Ketorolac tromethamine 0.5% is definitely more efficient in maintaining the interpupillary diameter at all stages of the surgery. Ketorolac tromethamine 0.5% can be safely and effectively used in order to prevent miosis during cataract surgery.

Key words: Cataract, Intraoperative miosis, Mydriasis, Topical NSAIDs, Topical Steroids, Ketorolac tromethamine 0.5%, Dexamethasone phosphate 0.1%.

LIST OF ABBREVIATIONS

AA	–	Arachdonic Acid
ACTH	–	AdrenoCortico Tropic Hormone
BSS	–	Balanced Salt Solution
CME	–	Cystoid Macular Edema
CO	–	Corneal Opacity
CSR	–	Cataract Surgical Rate
ECCE	–	Extra Capsular Cataract Extraction
E/D	–	Eye Drops
FDA	–	Food and Drug Administration
ICCE	–	Intra Capsular Cataract Extraction
IFIS	–	Intraoperative Floppy Iris Syndrome
IOL	–	Intra Ocular lens
IOP	–	Intra Ocular Pressure
IPD	–	Inter Pupillary Diameter
KPE	–	Kelman Phacoemulsification Technique
LTs	–	Leukotrienes
NSAID	–	Non Steroidal Anti- Inflammatory Drugs
PAF	–	Platelet Activating Factor
PCIOL	–	Posterior Capsule Intraocular Lens
PGs	–	Prostaglandins
PMMA	–	Poly Methyl Meth Acrylate
RAAB	–	Rapid Assessment of Avoidable Blindness
SICS	–	Small Incision Cataract Surgery
SD	–	Standard Deviation

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INTRODUCTION

A cataract is the loss of transparency of the crystalline lens of the eye, thereby preventing a clear image from forming on the retina. It is one of the leading causes of preventable blindness in the world today, accounting for 50% of loss of vision worldwide [1]. Age-related cataract is a progressive increase in lens opacification which deteriorates the quality of the retinal image thereby reducing the visual acuity in the senile age group. Three principal types of age-related cataract exist, depending on the area of the opacity of the lens, namely nuclear cataract, cortical cataract, and posterior subcapsular cataract.

Small-incision cataract surgery (SICS) is a variant of Extra Capsular Cataract Extraction (ECCE) which uses a small sclero-corneal incision to remove the cataractous lens with minimal manipulation. It is the most commonly used technique in all types of cataract. It involves the creation of a small self-sealing sclera-corneal tunnel, removal of the anterior capsule, manual onepiece expression of the nucleus, manual aspiration of the cortex and Intra Ocular Lens(IOL) implantation. Visual rehabilitation is comparable to phacoemulsification but SICS is faster and avoids the need for expensive technology.

There occur many kinds of complications at different situations in Small Incision Cataract Surgery. In the majority of cases, these occur in relation to capsule damage with consequential anterior segment vitreous and iris involvement, occasionally involving the incision as well. Poor technique may lead to inflammatory conditions when lens material has not been completely removed or incision has not been closed properly.

The important steps in cataract surgery are the delivery of the lens nucleus and cortical matter removal. The lens nucleus is the hardest part of a cataractous lens and

it requires a wide field for its easy delivery. During cataract surgery, intraoperative miosis may occur due to a number of reasons that hampers the delivery of nucleus. It increases the intraoperative and postoperative complications [2]. Maintaining adequate size of the pupil by medical techniques is of utmost importance in Small Incision Cataract Surgery.

Even in normal patients in whom there are no associated local or systemic diseases, surgical trauma can trigger a cascade of events like productions of Prostaglandins (PGs), Leukotrienes(LTs) and inflammatory mediators. Surgical incision is an iatrogenic trauma that will lead to activation of enzyme phospholipase-A2 that acts on the membrane phospholipids to produce Arachidonic acid (AA) and Platelet Activating Factors (PAFs). Cyclo-oxygenase is the enzyme that converts arachidonic acid into endoperoxidase and then to PGs[3]. Endogenous PGs produce multiple deleterious effects in the eye like miosis, postoperative uveitis, conjunctival congestion and change in intraocular pressure[4].

The miosis caused by PGs can be prevented by topical pharmacological agents or mechanical mydriasis. In uncomplicated cataract cases in order to maintain intraoperative mydriasis, pharmacological agents can be used either topically or intracamerally. Drugs that inhibit either phospholipase-A2 enzyme or cyclo-oxygenase enzyme, thereby interfering with endogenous PGs production at its various stages, can be used topically to prevent intraoperative miosis. Phospholipase-A2 is inhibited by corticosteroids and cyclo-oxygenase enzyme is inhibited by the class of drugs called Non Steroidal Anti- Inflammatory Drugs (NSAIDs). The mechanism of action of corticosteroid is important in that it indirectly inhibit the synthesis of PGs at an earlier stage by preventing the release of AA from membrane phospholipids by inhibiting the enzyme phospholipase-A2[5,6].

The NSAIDs which were initially approved for maintaining intraoperative mydriasis during cataract surgery are Flurbiprofen 0.03%[7] and Suprofen 1%[8]. Many NSAIDs have this tendency to prevent the release of PGs and thereby preventing intraoperative miosis. They are commercially available as eye drops that can be used before cataract surgery[9].

Corticosteroids commonly used to prevent post-operative complications are Prednisolone acetate 0.1% and Dexamethasone phosphate 0.1%. The efficacy of these drugs in maintaining intraoperative mydriasis is described by very few studies. Since corticosteroids are stronger anti-inflammatory drugs and they can inhibit the release of prostaglandins at a much earlier step, they can be better at preventing intraoperative miosis.

The efficacy of NSAIDs and Corticosteroids should be analyzed in detail so that the choice of preoperative drug to prevent the complications of miosis can be made in cases of uncomplicated cataract surgeries.

AIM AND OBJECTIVE

1. To study the efficacy of topical Dexamethasone Phosphate (0.1%) and topical Ketorolac Tromethamine(0.5%) in maintaining intraoperative mydriasis during Small Incision Cataract Surgery.

REVIEW OF LITERATURE

Definition:

Cataract is caused by the degeneration and opacification of the lens fibres already formed, the formation of aberrant lens fibres or deposition of other material in their place. The loss of transparency occurs because of abnormalities of the lens proteins and consequent disorganization of the lens fibres.

History of cataract surgery:

Anciently the white pupillary reflex caused by the mature cataract was called a suffusion which means a coagulation of humours formed behind the iris. The term cataract was first used by an Arabic Oculist called Constantinus Africanus in 1018 AD[10]. He introduced the Latin 'cataracta', meaning 'something poured underneath something', the 'waterfall'[11].

History of cataract surgery dates back for about 20 centuries. Early writings (Celsus, 25 BC–AD 50) allude to the fact that practitioners (e.g., Philoxenes, 300 BC) of that period did treat surgically for cataracts, but records and descriptions of their work and techniques have been lost to antiquity. For more than 20 centuries, couching was the primary method for dislodging the cataract away from the pupil. The first written description of couching came from Sushruta , an ancient Indian surgeon (c. 600 BC).In the method of couching, blunt needle and lancet were used to push the lens posteriorly and inferiorly into the vitreous cavity. A number of countries including some European nations adopted this technique in the 18th and 19th centuries. Rhazes (AD 865–925) wrote about Antyllos (AD 150), who followed a technique to remove the cataract by using a glass tube[12]. Ammar (AD 996–1020), an Iraqi

described the suction of the cataract through a hollow needle[10]. Constantinus Africanus (AD 1010-1087) first introduced the cataract procedure.

The father of modern cataract surgery, Jacques Daviel, invented the incisional extraction of the cataract in the year 1753. But couching was still popular among surgeons for about 150 years. Daviel executed a planned extracapsular cataract extraction. He made an incision at the lower limbus with a keratome. The incision was extended with scissors right and left above the level of the pupil. The lens capsule was incised with a sharp needle. The lens contents were loosened by a spatula. The cataract (nucleus) was expressed by gentle pressure. Lens material (cortex) was removed by curette.

Between 1753 and 1862, some milestones took place that affected the direction of cataract surgery. Carl Himly, a German oculist, did a great deal to the surgeon's view during operation by introducing pharmacologic mydriasis. Albert Mooren of Düsseldorf added a effect by creating a preliminary iridectomy to counteract the complication of pupillary block[12]. Daviel's technique was subsequently refined by others e.g. Albrecht von Graefe (1828-1870)[13].

The technique of Daviel had many postoperative complications. To overcome this a newer technique was invented wherein the entire lens along with the capsules was removed. This is called Intracapsular Cataract Extraction (ICCE) and it was introduced by George de la Faye (1752) and Samuel Sharp (1753) almost at the same time. Following section and iridectomy, and with the pupil dilated, the inferior section of the anterior capsule was held by the forceps and by a number of rocking manoeuvres the zonules were debilitated and disconnected from the ciliary muscle (most frequently without a breach of the capsule). Following this, the lens was

inverted by an action known as tumbling. The procedure of ICCE was enhanced during the 19th and 20th centuries with the utilization of other instruments e.g. curette, spoon and strabismus hook.

In 1867, Boston's Henry W Williams introduced suture closure of the incision site in a cataract surgery. In 1895, Colonel Henry Smith advocated performing the intracapsular cataract extraction without much internal anterior chamber manipulation.Smith would describe a modification of his mechanical zonulolysis that would cause a linear sliding of the whole lens without tumbling[14].

Mechanical zonular destruction was first used by Christiaen (1845) and Luca (1866). Joaquin Barraquer in 1957, launched Zonulysin, which could be introduced into the posterior chamber prior to either of the methods resulting in zonulolysis, thereby simplifying removal of the lens. Cryoextraction of the lens was launched by Tadeusz Krwawicz, in 1961[15]. ICCE was the most popular method. However, due to a higher occurrence of cystoid macular edema, retinal detachment, vitreous loss, astigmatism and use of anterior chamber intraocular lens, there was a switch from the technique of ICCE to the newer technique of ECCE. This was in an effort to bring down the complications and to make easier the placement of intraocular lenses[13]

Post-modern era:

The Intraocular lens (IOL) was developed between 1940-1970. Harold Ridley performed his first artificial lens implant at St Thomas' Hospital in London on Nov 29, 1949,18 but the approach was not correct for the popular ICCE movement as the IOL implantation was difficult with no anchor[16]. The year 1949 witnessed newer developments in the intraocular lenses (IOLs). During the battle of Britain in World War II many plastic canopies of spitfire air planes were shattered by enemy gun fire.

This plastic material (poly methyl methacrylate- PMMA) occasionally lodged inside the eyes of pilots. It was noted that it resulted in little response to the plastic material, only if it did not move about inside the eye. Thus, Ridley realized that such a plastic material can be used to substitute the human lens. Subsequently, he began placing the disc form PMMA lenses in a biconvex design posterior to the iris following ECCE[15] .The various advancements in the operative techniques included permitting the lens removal while leaving behind the lens capsule .The unbroken capsule represents a barrier, preventing the lens material from dropping into the vitreous cavity. This permitted less advanced cataracts to be treated, considering that any leftover fragments would be eliminated at the time of surgery with aspiration and would not be kept in the vitreous, where they would provoke inflammation.

Between 1965 and 1972, Cornelius Binkhorst of Holland was modifying the IOL concept. He believed that an intact posterior capsule would provide better anatomic support for the IOL. He first set out to refine the Extra Capsular Cataract Extraction (ECCE) so that all cataracts, hard and soft, could be removed.By 1977, Worst and colleagues worked on the concept of ECCE and reported on a large series of 2000 cases. They developed a new lens. This IOL required suture fixation to the iris as well as two loops protruding through the pupil to rest on the posterior capsule[17].

Kelman impressed the ophthalmic community by publishing his description of the ultrasonic breakup of the nucleus coupled with the Scheie concept of irrigation–aspiration of the cortex in 1967. He made the Kelman phacoemulsification technique (KPE)[18].William Simcoe (1977) introduced his Simcoe curved 23-gauge cannula connected to a small irrigating bulb[19].A new product was discovered which was the

AC -maintaining viscoelastic gel, hyaluronic acid.David Miller and Robert Stegman, demonstrated the viscoelastic substance[20].

Phacoemulsification has now become the technique of choice for all surgeons due to a smaller size of the incision and greater postoperative outcome. The most meaningful change made in the current times was the launch of phacoemulsification surgery by Dr.Charles Kelman in 1967[21]. Over the decades many changes have been made by many incredulous surgeons in the phacoemulsification technique that has resulted in the polished recent day procedure.

Summary of Major Developments in Cataract Surgery

800 BC Couching performed by Indian surgeons

1750 Daviel carries out the first ECCE on humans

1753 Sharp performs the first successful ICCE

1949 Ridley implants the first IOL

1967 Kelman introduces phacoemulsification

1980 Miller and Stegman use Healon to stabilize AC

1993 Nagahara demonstrates the ‘’phaco chop’’ technique

1998 Agarwal presents the bimanual microincisional phacoemulsification

History of mydriatics :

Around 500 years ago, an anticholinergic drug was extracted from leaves of the fatal nightshade plant and instilled into the eye to induce mydriasis. Since large

and dilated pupils were considered beautiful the term “belladonna” was introduced in the 1500s.The main active component of belladonna is atropine. Later anticholinergic drugs with shorter lifespan like tropicamide and cyclopentolate were introduced in and around 1950s. Later in 1897, a new group of mydriatic drugs was introduced, the adrenergic agonists consisting of epinephrine and phenylephrine[22].

Epidemiology of cataract:

Cataracts causes blindness (visual acuity in the better eye of less than 3/60) in 10.6 million people and moderate to severe visual impairment (visual acuity of between 6/18 and 3/60) in 34.4 million people according to a study in 2010. The cataract prevalence is influenced by the number of cataract surgeries performed per million people per year called as Cataract Surgical Rate (CSR). It varies from less than 200 to over 6000 in different regions. The CSR is determined by the effectiveness of strategies and how well easily accessible cataract surgery is delivered. So a high cataract prevalence rate in some developing countries is not because of lack of treatment but due to lack of surgeons and ineffective governmental strategies .

According to a national survey in India (1999-2001) 62.6% of blindness in age groups >50 years is due to cataract. During 2006-2007, the Rapid Assessment of Avoidable Blindness(RAAB) was conducted and reported 72.2% of blindness due to cataract in ages >50 years.

Need for sustained intraoperative pupillary mydriasis:

A well sustained mydriasis is crucial for an uneventful cataract surgery. Many times although initially good mydriasis is attained, the mydriatic effect tends to diminish during surgery [23], chiefly in patients with either rigid pupils as in diabetes

mellitus or in patients with intraoperative floppy iris syndrome (IFIS)[24,25]. Intraoperative pupillary constriction multiplies the risk for complications, such as incomplete cortex removal, iris damage, and posterior capsule rupture[26] with the nucleus dropping into the vitreous and vitreous loss. Different complementary measures proposed to sustain a good mydriasis intraoperatively include; mechanically, e.g. with iris retractors, pharmacologically, e.g. with topical non-steroidal anti-inflammatory drugs (NSAIDs)[27], viscous metaoxedrine 10%, or with intraoperative intracameral irrigation with epinephrine[23].

The amount of mydriasis induced by any pharmacological agent can be classified as; weak pupil size(<4.0 mm), moderate (4.0 to 6.0mm), large (6.0 to 8.0mm), or very large (>8.0mm)[28].

Applied Anatomy and physiology:

Cornea

The cornea is composed of six layers :

Epithelium-The corneal epithelium is stratified, squamous, non keratinized and lacks goblet cells. At the limbus it is continuous with that of the conjunctiva. The epithelium accounts for about 5-10 % of the overall corneal thickness; 540 microns[29] . It is a multilayered structure. The basal cells are held together by desmosomes and to the underlying basal lamina by hemidesmosomes. Similarly, the epithelial cells at the surface are held together by desmosomes and zonulae occludentes. These tight junctions make the corneal epithelium semipermeable. The epithelial cells possess surface microvilli that helps stabilize the precorneal tear film.

Bowman's layer- It is a narrow, cellular homogeneous zone, 8-14 microns thick. The anterior limit of the bowman's layer is sharply defined from the overlying epithelium though it is infiltrated by the lamina densa anteriorly and posteriorly it merges with the underlying stroma. Anteriorly the periphery of the Bowman's layer forms the junction between the cornea and the limbus, it is marked clinically by summits of the marginal arcades of the limbal capillaries. Trauma, both mechanical and infective is resisted by the Bowman's layer; once destroyed it cannot regenerate and is substituted by coarse scar tissue. The unmyelinated nerves perforate the bowman's layer to enter the corneal epithelium.

Stroma (substantia propria) – The stroma is around 500 microns in thickness and collagen fibril lamellae constitute it (approximately 200-300 centrally and 500 in the periphery). The lamellae lie in a proteoglycan ground substance together with keratocytes. The lamellae in the anterior third run forwards obliquely to be inserted into Bowman's layer. The arrangement of the collagen fibrils is the reason behind the transparent nature of the cornea. The corneal stroma also contains keratocytes, lymphocytes, macrophages and rarely polymorphonuclear leucocytes.

Dua's layer- It is the pre descemet's layer.

Descemet's membrane – Corneal endothelium uses the descemet's membrane as the basal lamina. Its thickness increases with age, it is around 3-4 µm thick at birth, 5 µm thick in childhood and around 10-12 µm in adulthood. The descemet's membrane primarily consists of type IV collagen. In adults the anterior third has an irregular banded pattern in cross-section while the posterior two-thirds of the membrane, consists of a homogeneous fibrillogranular material. Posteriorly, the Descemet's

membrane is attached to the underlying endothelium by hemidesmosomes. It is inelastic.

Endothelium – The endothelium comprises of a single row of cuboidal cells. During early development the endothelial cells differentiate from cells that migrate from the limbal area. They are avascular in origin. With advancing age there occurs a gradual decrease in density and increase in shape (polymegathism) of the endothelial cells. The endothelial density is about 6000 cells at birth. It becomes lesser in adulthood reaches a total count of 1500-2500 cells/ mm^{30}. A reduction in the endothelial cell density may occur due to trauma, intraocular surgery and eye diseases in addition to that seen due to advancing age.

The endothelial cells contribute to maintaining the stroma in a dehydrated state by controlling the ion transport across the endothelium and thus the term "fluid pump" has been used to describe the potential of the endothelium to dehydrate the stroma. A break in the endothelial cell layer that decreases the capability of the fluid pump, can produce corneal oedema or decompensation. The anatomy of the corneal endothelium can be illustrated by a specular microscope and assessed by imaging programs[31].

Iris:

The iris is the anterior most part of the uveal tract which is the pigmented middle layer of the eye. It is continuous with the pia-arachnoid coverings of the optic nerve. The iris acts as a diaphragm with an aperture, the pupil. The stroma of the iris is composed of fibroblasts, melanocytes and loose collagenous material that contain its nerves and blood vessels. The main function of the iris is to control the aperture of the pupil. Larger the pupillary diameter, more is the amount of light entering the eye,

while, smaller the diameter the lesser the amount of light entering the eye. The pupil size is maintained and altered by the muscles -sphincter pupillae and the dilator pupillae[32]. The sphincter pupillae is formed by circularly arranged smooth muscle fibres at the rim of the pupillary margin. The pupillary aperture decreases upon contraction of these fibers. The dilator pupillae muscle fibers are arranged radially and on contraction increase the pupillary aperture. The anterior surface of the iris has no epithelial covering. The posterior surface of the iris has a bilayered epithelium, its deeper anterior layer is pigmented to absorb light. The more superficial posterior layer is non-pigmented and is in continuity with the pigmented layer of the retina. The muscles of the iris are supplied by autonomic nerves. The constrictor pupillae muscle is innervated by parasympathetic fibers arising from the ciliary ganglia, which also innervate ciliary body and lacrimal glands. Therefore, parasympathetic activation causes not only the contraction of sphincter muscle leading to pupillary constriction but also causes ciliary muscle contraction leading to spasm of accommodation and stimulation of tear secretion.

When stimulated by light, the pupil constricts, reducing its diameter and causing a five-fold decrease in the amount of light entering the eye. The dilator pupillae muscle is innervated by sympathetic autonomic fibers that originate in the T1 segment of the spinal cord. Preganglionic fibers pass to the superior sympathetic cervical ganglion. Postganglionic fibers pass along with the blood vessels such as the internal carotid artery and supplies the dilator pupillae and mullers muscle. When stimulated, the radial fibers of the dilator pupillae constrict and the pupillary diameter increases. The adrenergic receptors in the dilator pupillae are mainly α and a few β (β2-receptor subtype). Sympathetic control of ciliary muscle action is not well established[33].

Cholinergic receptors in sphincter and ciliary muscle are of muscarinic type with predominance of M3 subtype. Sympathetic input to eye is relatively small as compared to parasympathetic input. The sympathetic responses have a slower onset and takes about 30-40 seconds to reach the peak effect. In comparison parasympathetic responses reach the peak effect within 1–2 seconds.

Sympathomimetic drugs have a relatively little effect on accomodation. Parasympatholytic drugs act by blocking of the muscarinic receptors, which are present on both the sphincter pupillae and ciliary muscle, thereby, producing pupillary dilatation and paralysis of accommodation. Sympathomimetics are comparatively weak mydriatic agents and in subjects having an iris difficult to dilate, such as diabetics or blacks, stronger antimuscarinic agents are used[33].

Lens:

The lens is suspended in the eye by zonules which are inserted on the anterior and equatorial regions. It is attached to the ciliary body on the other end.The lens is composed entirely of epithelium surrounded by a capsule. The lens capsule is a thick, collagenousbasement membrane which is transparent, isthickest at the anterior pre equatorial region and thinnest atthe posterior pole.

The lens epithelium is a single layer ofcells enclosing the capsule which has two types of cells: the cells in central zone that are not actively dividing, and cells inthe pre-equatorial germinative zone that are actively dividing and give rise to the lensfibres. The cells are interconnected by gap junctions anddesmosomes. As the lens fibres elongate and new ones form, the older ones are pushed towards the depth of the lens so that the youngest lens fibres are the most superficially located.The nucleus includes an embryonic nucleus consisting of primary lens fibres surrounded by the

foetal nucleus. The yellow-brown, dense central zone of the lens is termed the nucleus. The region of the lens surrounding the nucleus is called the cortex.

Pathophysiology of cataract formation:

The loss of transparency that occurs in cataract is because of the abnormalities occurring in the lens proteins and the resultant disorganization of the lens fibres. Physical or chemical factors which disturbs the intracellular and extracellular equilibrium of water and electrolytes or disturbs the colloid system within the lens fibres might end up in the opacification of lens. When the germinal epithelium of the lens loses its ability to form normal fibres, aberrant lens fibres are formed. This is seen in posterior subcapsular cataract. In the early stages of cataract hydration of the lens may occur and frequently fluid lacunae are seen below the capsule and between the fibres resulting in the swelling of the entire tissue leading to intumescent cataract. Hydration may be due to osmotic changes within the lens or to changes in the semipermeability of the capsule. The second factor is denaturation of lens proteins. Denaturation of proteins is another important factor in which there is an increase in the insoluble proteins that are irreversible. Such an alteration occurs typically in the young lens or the cortex of the adult lens where metabolism is relatively active. In older fibres slow sclerosis occurs. Clinically, when the hydration of lens or denaturation is predominant the condition is called a ‘soft cataract’. When the sclerosis of lens occurs it is described as a ‘hard cataract’.

Pharmacology and drug transport:

Ideal properties of mydriatics as given by Havener (1975)[33].

- Quick in onset.
- Adequate duration.
- Fast recovery after examination.
- Light reflex abolished.
- No cycloplegia.
- Capable of quick reversal in an emergency.
- Intraocular pressure remains constant.
- No other pharmacological effect.
- No local toxic reaction.
- No systemic toxic reaction.
- No adverse subjective complaints such as 'stinging'.

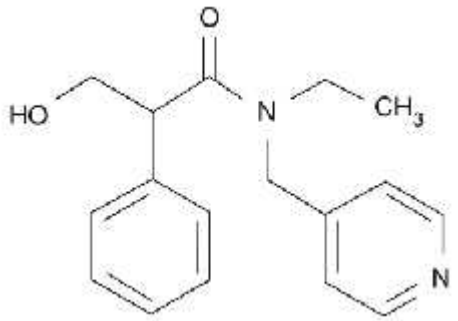
The mydriatics commonly used are:

1. Sympathomimetics: Non selective: epinephrine/adrenaline

   Selective: Phenylephrine, hydroxyamphetamine.

2. Antimuscarinic: Atropine, homatropine, cyclopentolate, tropicamide.

Tropicamide:



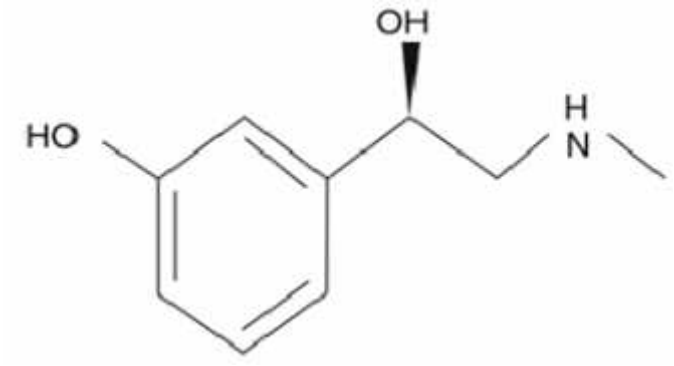
Tropicamide is a synthetic derivative of tropic acid. It is a non-selective antimuscarinic agent. Its penetration through corneal epithelium is better than atropine, homatropine and cyclopentolate and therefore, it has a quick onset and shorter duration of mydriasis. It is available in two concentrations, 0.5% and 1%. Maximum mydriasis occurs in 20–40 minutes and pupil reaches pre-instillation size in 6 hours[34]. Mydriatic effect of tropicamide is not dependent on concentration and 1% concentration produces only slightly larger pupil. Cycloplegia appears in 30–35 minutes and is dose-related. The pupillary dilatation produced by tropicamide is stronger than its cycloplegic effect. For cycloplegic refraction tropicamide is not the drug that is most preferred[35–37].

Tropicaimde is free from vasopressor effects. Tropicamide 1% was also reported to cause greater mydriasis as compared to phenylephrine 2.5% and a combination of tropicamide 1% and phenylephrine 2.5% more effective than either of them used alone[38–40]. The combination has an equivalent mydriatic efficacy and greater cycloplegic efficacy as compared to phenylephrine 2.5% followed by tropicamide 0.5% instillation. Its effect does not vary with age or iris pigmentation. A combination of tropicamide 0.5% and phenylephrine 0.5%, injected intracamerally has proved to be effective and safe in dilating pupils in subjects with poor mydriasis following preoperative instillation[41].

Adverse effects and Contraindications: Adverse effects include stinging and burning sensation. In patients with narrow angle, intraocular pressure may rise. It is, therefore, avoided in patients with angle-closure glaucoma. Tropicamide is significantly absorbed in systemic circulation but it has poor affinity for systemic muscarinic

receptors and, therefore, its systemic adverse effects are rare. Hypersensitivity to tropicamide has been reported.

Phenylephrine:



Phenylephrine is an α 1-adrenergic agonist[42]. It causes contraction of dilator pupillae causing pupillary dilatation, constriction of conjunctival vessels causing blanching and contraction of Muller's muscle causing widening of palpebral aperture. The effect of phenylephrine on accommodation is relatively weak. It is available in single-use units in concentrations of 0.12, 2.5 and 10%[43].

Onset of mydriasis begins in about 10 minutes and reaches peak in 45–60 minutes[24].The pupil returns to pre-instillation size in 6–7 hours. Diabetics dilate slowly and less widely as compared to non-diabetics. Phenylephrine 10% has significantly higher efficacy as compared to 2.5% concentration in diabetics[44]. People with dark iris tend to develop mydriasis slowly but for a longer duration as the drug binds to pigment in iris[38]. It produces less effect on accommodation as compared to muscarinic antagonists.

In addition to its uses as a mydriatic agent, phenylephrine is also used for the following: It causes blanching of superficial conjunctival blood vessels and in very

low concentrations (0.125%), it is employed as an ocular decongestant. In a concentration of 10% phenylephrine is applied topically for breaking synechiae.

Phenylephrine also causes widening of the palpebral fissure by stimulation of muller's muscle therefore ptosis caused by sympathetic denervation such as in Horner's syndrome may be recovered. Phenylephrine 1% is used for the diagnosis of Horner's syndrome. It causes marked pupillary dilatation in the eye with sympathetic denervation in the postganglionic fibres. If the lesion is central or preganglionic, the pupil has minimal or no dilatation[45].

Adverse Effects:

Ocular adverse effects: Local adverse effects of phenylephrine include stinging, pain, lacrimation and keratitis. It can cause allergic dermatoconjunctivitis. In elderly patients, phenylephrine causes rebound miosis; long-term repeated use results in slow and less intense mydriasis. Long-term use as an ocular decongestant causes rebound conjunctival congestion.

Systemic adverse effects: Phenylephrine 10% is known to cause a rise in mean arterial blood pressure therefore elderly patients especially those with cardiovascular disease, those patients receiving tricyclic antidepressants and monoamine oxidase inhibitors are prone to develop acute rise in blood pressure following topical application of 10% phenylephrine. Other systemic adverse effects of 10% phenylephrine include occipital headache, ventricular arrhythmias, tachycardia, reflex bradycardia, subarachnoid hemorrhage, ruptured aneurysm, skin blanching[38,43] . Phenylephrine 2.5% is rarely associated with systemic adverse effects[39,40].

Behndig and Lundberg in 2010 conducted a study to evaluate the mydriatic response to concentrations of phenylephrine injected intracamerally from 0.15 mg/mL to 30.00 mg/mL (0.015% to 3.000%). The results showed that phenylephrine when injected intracamerally does not show a linear mydriatic dose-response relationship in humans. The mydriatic response obtained was almost the same at all the four lower phenylephrine concentrations (0.015% to 0.500%),with ultimate sizes of the pupils of approximately 4.3 mm and greater for the two higher concentrations (mean 5.80 mm)[33].

John A. Hovanesian et al conducted a randomized clinical trial in the United States and the Netherlands in twenty centers to study the efficacy and safety of phenylephrine 1.0%-ketorolac 0.3% for sustaining mydriasis during the procedure, and for the reduction of ocular pain after, cataract surgery in which they concluded that phenylephrine 1.0%–ketorolac 0.3% given intracamerally with irrigation solution during cataract surgery was safe and effective for sustaining mydriasis through the procedure and decreasing postoperative ocular pain[27].

Lignocaine (Lidocaine, Xylocaine)

Lignocaine is currently most commonly employed anesthetic agent. It is the prototypical amide local anesthetic. As local injection, it is available as 1% to 2% and for topical use it is available as 4% with preservative and 1% as preservative free for intracameral use. Lidocaine at 1% concentration is prepared from 2% solution by diluting in balanced salt solution (BSS) or BSS plus or directly as a 1% unpreserved lidocaine for intracameral irrigation. Lidocaine produces more intense, rapid, longer sustaining, and more extensive anesthesia than does an equal concentration of procaine. Although it is effective when used without any vasoconstrictor, when used

with epinephrine, epinephrine reduces the rate of absorption such that the toxicity is reduced and the duration of action usually is prolonged. The various routes of administration include injection, topical, mucosal, and transdermal. Lidocaine crosses the corneal epithelium and stroma causing sodium channel blockade thus resulting in local anesthesia. Since lidocaine is not degraded within the eye, it has long period of action of almost 20 minutes[42]. Intracameral lidocaine dilates the pupil due to the anesthetic action of the lidocaine on the nerves in the iris stroma[42,46]. Inhibiting the action of both the iris sphincter and the dilator results in the dilatation of the pupil because the sphincter has a more powerful tone than the dilator and it has no cycloplegic effect[33,47].

Lincoff et al reported the consequence of the use of lidocaine on iris paralysis and mydriasis. They found that accidental intraocular injection of lidocaine without injection of a mydriatic drug dilated the pupil[48].

Lidocaine is metabolized in the liver by dealkylation carried out by cytochrome P450. It is converted to monoethylglycine xylidide and glycine xylidide, which can further be metabolized to monoethylglycine and xylidide. Both, the products monoethylglycine xylidide and glycine xylidide hold back their local anesthetic activity. In humans, approximately 75% of the xylidide is eliminated in the urine as a metabolite 4-hydroxy-2, 6-dimethylaniline.

Adverse effects: The most severe adverse reactions of lidocaine include seizures, coma, cardiac arrest and respiratory paralysis. The most common side effects seen are tinnitus, dizziness, twitching and drowsiness.

Cionni, Barros et al in 2003 introduced a technique with the use of preservative free lidocaine 1% to cause pupillary mydriasis without the use of any preoperative

dilating eye drops. The study showed that lidocaine causes paralysis of the sphincter pupillae, achieving adequate mydriasis in about 90 seconds which was sustained or enhanced at the end of surgery[47].

Nikeghbali, Falavarjani et al conducted a study and concluded that preservative-free lidocaine 1% administered intracamerally provided rapid, effective mydriasis comparable to that of topical mydriatics[46].

A research was conducted by Rajesh Subhash Joshi to study the effect of intracameral injection of preservative-free lignocaine to induce pupil dilatation, without using any preoperative dilating eye drops or intraoperative mydriatics, on 32 patients patients with age-related cataract associated with type 2 diabetes mellitus. It was concluded that intracameral lignocaine 1% delivers adequate mydriasis for a secure phacoemulsification of the cataract in patients having type 2 diabetes for a variable duration[49].

The various routes of delivery of the mydriatic drugs

The iris is a porous structure, allowing a rapid access of solutes present in the anterior chamber irrespective of size which in turn control the sphincter and dilator muscles. Thus a drug in the anterior chamber can reach the biophase of these muscles in a very short time, and their response will correspond to that in the incubation bath of an in vitro system.

Topical route

Primary routes of drug delivery following topical administration of ocular drugs include cornea and conjunctiva.

Transcorneal drug Absorption- Cornea is the major site of drug absorption intothe intraocular tissue following topical administration.The tight junctions in thesuperficial layer of corneal epitheliumserve as selective barrier for the small moleculesand completely prevent the diffusion ofmacromolecules. The corneal epithelium is hydrophobic but lipophilic in nature. High extracellular and lowintracellular calcium levels are required formaintaining the normal permeability of tightjunctions. Hypertonic solutions showedan increase in the leakiness of tight junctions. The corneal stroma is a barrier to the lipophilic molecules as it is hydrophilic in nature, but also permits its easy passage. The endothelium offers little resistance to the movement of drug molecules as it is sandwiched by gap junctions. Due to its biphasic solubility characteristics, cornea functions as a barrier as well as depot for the topically applied drugs. Most of the drugs diffuse through corneal epithelium the intracellular pathways but some through the intercellular pathway. Passive diffusion along the concentration gradient is the main permeation mechanism for most topically applied drugs by both inter and intracellular routes. The anatomical structure of the cornea thus delays the onset of dilatation owing to the slow penetration of active ingredients through the cornea.

Transconjunctival drug Absorption - The conjunctiva consists of stratified columnarepithelium and lamina propria. The cells of which have tight junctions forming the main barrier for drugpenetration. Lipophilic drugs diffuse through theintracellular pathway but hydrophilic drugsrequire passage through intercellular pathway. However, the intercellular spacesin conjunctival epithelium are larger than thosein corneal epithelium. Thus, the conjunctiva is more permeable to hydrophilic drugs, thanthe cornea and molecules upto the molecular weight of 20000–40000 kilo Dalton canmove through the conjunctiva. The carrier mediated mechanisms help in

transferring the drugs to the inferior of the eye. Therefore, due to a significantly larger surface area than that of the cornea and it being highly vascular in nature the conjunctiva is a major route for the entry of topically applied drugs into the systemic circulation.

Mydriatic agents in eye drops are depot preparations; a wick soaked in standard mydriatic agents is applied on to the ocular surface[50], but the desired site of action is at the iris. The conjunctival sac has a capacity of approximately 15–30 μL and the natural tear film volume is 7–8 μL. When there is a normal blink rate of 15–20 blinks/min, the tear turnover rate is approximately 16 % per minute. Therefore, as a result of the overspill, a significant portion of the solution is lost. The remainder approximately 80% is drained through the nasolacrimal duct until the normal tear volume is restored[51].Approximtely, around 20% of a drop is preserved in the cul-de-sac because there is only a minimal increase in the volume of the lacrimal fluid [32]. Thus the mydriatic drug given topically has the disadvantages of delayed effect and low bioavailability[33].

Systemic absorption of topically delivered drugs is mainly through the open punctum into the nasolacrimal duct, followed by absorption through the lining mucosa. Absorption via the conjunctival vasculature is also an important route for systemic absorption of topical drugs. Instillation of multiple drops at a time increases the risk of adverse effects due to enhanced systemic absorption rather than increasing the ocular bioavailability.

In the mydriatic eye drop regimen usually given in patients planned for cataract surgery, a sympathomimetic like phenylephrine 2.5% is combined with an anticholinergic agent like tropicamide 1% in order to get maximum results. It has a

stronger effect than giving individual drugs. When given along with tropicamide, phenylephrine produces a significant increase in pupillary mydrasis. Decreased concentration of tropicamide 0.25% and phenylephrine 1.25% can give a long sustaining pupillary dilatation[41,52].

In the succeeding part of a study conducted by Lundberg and Behnding in 2007, to evaluate the possibility of removing epinephrine from the irrigating solution in phacoemulsification surgery, 50 patients were randomly distributed into 2 groups, all of whom were administered with topical mydriatics. Only in group 1, epinephrine was mixed to the irrigating balanced salt solution. It was seen that the size of the pupil decreased more rapidly in the group which had no epinephrine($12 \pm 7\%$)[53].

Intracameral route

Intracameral route of drug administration is mainly used for procedures such as for pupillary dilatation**,** anesthesia for surgical procedures, preventionof intraocular infection and inflammation. This method provides immediate and easydelivery of required concentration of drug intothe aqueous humor and, therefore, provideshigh efficacy. In the intracameral route of drug delivery the concentration of drugs used are less in comparison with those used topically, the permanence time in the anterior chamber is less than 1 minute, and no nasal mucosa absorption occurs thus decreasing the systemic absorption[33]. The need for repeatedadministration, as is necessary with the use of topical route, is avoided, which is the main concern in non-compliant patients.

Drugs for maintaining intraoperative mydriasis:

Corticosteroids:

Corticosteroids (glucocorticoids and mineralocorticoids) are 21-carbon structures that are synthesized by adrenocorticotropic hormone (ACTH)-controlled conversion of cholesterol in the adrenal cortex. They can also exist in synthetic forms such as prednisone, methylprednisolone, dexamethasone, triamcinolone, betamethasone, medrysone, fluorometholone (FML), and others.

Dexamethasone phosphate 0.1%:

Dexamethasone (dexamethasone disodium phosphate 0.1%) is more potent and longer acting, butits corneal penetration is less than that of prednisolone acetate. Dexamethasone achieves its peak concentration in aqueous humour between91 and 120 minutes following instillation when given in a mean concentration of 31ng/ml and is still detectablein the aqueous 12 hours after instillation[54]. Dexamethasone when given alone can increase the intraocular pressure. However, it can be in combination with antibiotics etc as it is effective in treating moderate inflammation and post-operative complications. Dexamethasone is approved by FDA for the treatment of inflammatory conditions of the palpebral and bulbar conjunctiva, cornea, and anterior segment of the globe and corneal injury. Dexamethasone is FDA approved for treatment of macular edema following branch retinal vein occlusion or central retinal vein occlusion. Its property of antagonizing inflammatory mediators can be used to prevent intraoperative miosis. It has the tendency to inhibit both the cyclo-oxygenase and lipoxygenase pathways thereby preventing the release of prostaglandins, leukotrienes and other mediators.

Adverse effects : Blue conjunctivitis, diplopia, stromal opacities, mydriasis, posterior subcapsular cataract, increase in intraocular pressure (secondary open-angle glaucoma), myopia, retinopathy, impaired color vision, pseudotumor cerebri, visual acuity/ visual field loss, blurred vision

NSAIDs:

NSAIDs affect the cyclooxygenase pathway of the arachidonic acid cascade, and offer varying degrees of anti-inflammatory and analgesic effects through inhibition of prostaglandins. Ophthalmic NSAIDs tend to be associated with fewer adverse events than systemic NSAIDs. The mechanisms through which the undesired effects appear are uncertain, and may be linked to concurrent conditions, rather than directly to the NSAID. Topical NSAIDs can reduce intraoperative miosis during ocular surgery, thereby increasing the surgeon's visualization and decreasing the risk of complications. Pre-operative use is key to achieving the NSAID's full effect. Given the more favorable side effect profile of NSAIDs, they are being increasingly used over corticosteroids to control inflammation after cataract surgery. NSAIDs are gaining off-label attention for their ability to prevent and treat cystoid macular edema, which can arise as a complication of cataract surgery.

Ketorolac tromethamine:

The chemical name of ketorolac tromethamine is (±)-5-benzoyl-2,3-dihydro-1H-pyrrolizine-1-carboxylic acid, compound with 2-amino-2-(hydroxymethyl)-1,3-propanediol (1:1).Ketorolac tromethamine is a NSAID that, when administered systemically, has demonstrated analgesic, anti-inflammatory and antipyretic activity which, characteristically, is used for ≤ 5 – 7 days. The mechanism of its action is thought to be due to its ability to inhibit prostaglandin biosynthesis. The detectable

amount of ketorolac tromethamine in the plasma is 10.7 – 22.5 ng/ml at day 10 during topical ocular treatment. When ketorolac tromethamine 10 mg is administered systemically every 6 h, peak plasma levels at steady-state are ~ 960 ng/ml.

Bucci *et al* recently compared the efficacy of Ketorolac tromethamine 0.5%, ketorolac tromethamine 0.4% and Diclofenac sodium 0.1%, used concomitantly with prednisolone 0.1% for controlling mild to severe inflammation following cataract surgery. "There were no significant differences in the efficacy of Ketorolac tromethamine 0.5% versus 0.4% when used in combination with prednisolone"[55,56].

A clinical study compared the efficacy, safety and patient comfort of two topical steroids (prednisolone 1% and rimexolone 1%) with ketorolac tromethamine 0.5% after extracapsular cataract extraction in a prospective, randomised, double-masked study of 45 patients. Patients were assigned to receive topical treatment with prednisolone, rimexolone or ketorolac tromethamine 0.5% ophthalmic solution after phacoemulsification for cataract extraction. Although there were no significant between-group differences in inflammatory cell counts ($p = 0.165$), flare readings in the anterior chamber were lowest in the ketorolac tromethamine 0.5% group ($p = 0.008$). One patient in the prednisolone group experienced elevated intraocular pressure (IOP) and had to be excluded. The authors concluded that ketorolac tromethamine 0.5% provides good control of intraocular inflammation after cataract extraction without the risk of a steroidal IOP increase[57].

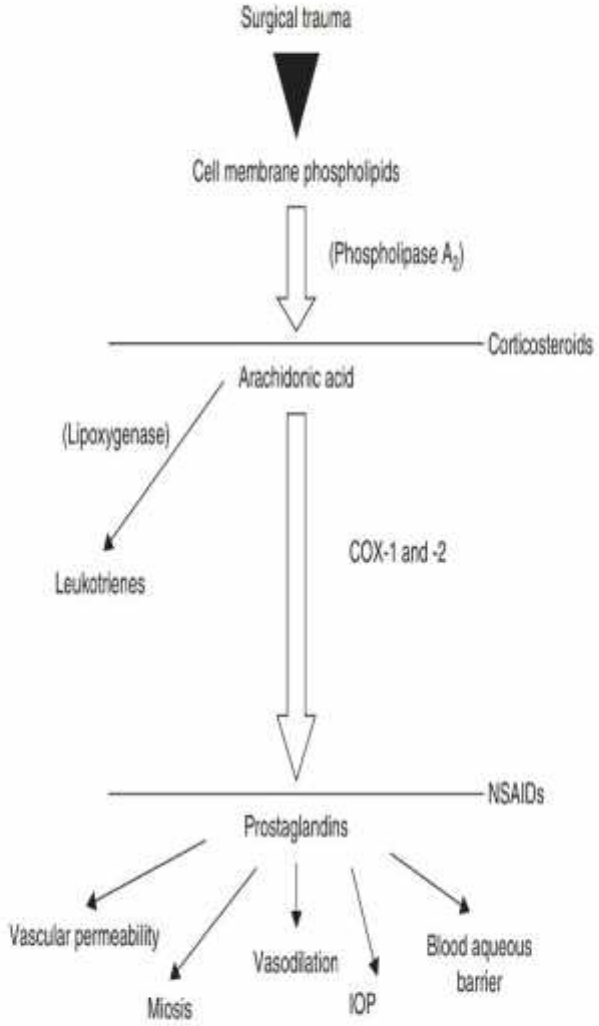
Ketorolac in the inhibition of miosis:

Ketorolac tromethamine 0.5% is effective in preventing miosis in Cataract surgery as it has the propensity to prevent the release of prostaglandins and other inflammatory mediators.

Srinivisin *et al.* [58] reported that "topical Ketorolac tromethamine 0.5% was a more effective inhibitor of miosis than topical diclofenac during extracapsular cataract extraction and IOL implantation. Ketorolac tromethamine 0.5% also provided a more stable mydriatic effect throughout surgery".

Similarly, Snyder *et al.* [59] reported that "the use of Ketorolac tromethamine as a single agent negated the need for use of a combination of preoperative NSAID (Flurbiprofen) and postoperative corticosteroid for the prevention of intraoperative miosis and postoperative inflammation in cataract surgery".

In their study of 26 patients, there were no statistically significant differences in dilation (preoperative versus postoperative) or cell and flare postoperatively. The authors concluded that "the use of ketorolac tromethamine as a single agent could eliminate the expense of using separate anti-inflammatory and antimiotic preparations preoperatively and postoperatively, thereby enhancing surgeon convenience and patient convenience and compliance".



Prostaglandin synthesis.

IOP: Intraocular pressure.

METHODOLOGY

The present study was conducted in the North Western part of the state of Karnataka in the Department of Ophthalmology, KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi to compare the efficacy ofpreoperative topical Dexamethasone phosphate 0.1% versus Ketorolac tromethamine 0.5% in maintaining intraoperative mydriasis during small incision cataract surgery. The study was done for a period of one year between 1st January 2019 and 31st December 2019. The research project was approved as ethical and justifiable by the Jawaharlal Nehru Medical College Institutional Ethical Committee on Human Subjects Research, Belagavi.

Source of Data:

Subjects attending the Ophthalmology OPD with uncomplicated cataract.

Method of collection of data:

Study Design: A one yearRandomized Control Trial.

Study period: January 2019 – December 2019.

Study population: Patients >40 years of age attending the Ophthalmology OPD with uncomplicated cataract and who were scheduled to undergo Small Incision Cataract Surgery(SICS) with Posterior Capsule Intraocular Lens (PCIOL) implantation at KLES Dr Prabhakar Kore Hospital and MRC, Belagavi.

Sample size: Sample of 180 patients were divided into two groups of 90 patients each namely Group A and Group B using Simple Random Sampling method.

Sample size:

The minimum sample size formula based on mean and standard deviation is

$$n = \frac{(z_\alpha + z_\beta)^2 (s_1^2 + s_2^2)}{(\bar{X}_1 - \bar{X}_2)^2}$$

where z_α was linked with the level of significance and z_β was linked with the power of the test. For 5% level of the significance z_α =1.96 and z_β =0.84 for 80% power of the test.

$\bar{X}_1$ was the mean of the pupillary diameter for the first group (5.75) and $\bar{X}_2$ was the mean of pupillary diameter of the second group (6.16).s_1 was the standard deviation of the first group (0.73) and s_2 was the standard deviation of the second group (0.97). With these values the sample size obtained was 69.

To make the study more confirmative, the sample size was raised to 90.

Statistical Analysis:

For the continuous quantitative variables mean and standard deviation was calculated. The inter group continuous variables was compared using suitable tools of statistics like unpaired student's t test. Two quantitative variables, within a group, was compared using student's paired t test.The association between the outcome, clinical and demographic characteristics was tested using Chi-square test or Fisher's exact test.For all the tests the value of p less than 5% (0.05) was considered significant.

Instruments used for data collection-

1. Proforma and Consent form.

Group A : Dexamethasone group: n= 90

Group B : Ketorolac group: n=90

SELECTION CRITERIA:

Inclusion criteria:

1) Patients of either sex > 40 years of age with uncomplicated Senile Cataract.
2) Patients who were scheduled to undergo Small Incision Cataract Surgery with PCIOL implantation.

Exclusion criteria:

1) Secondary, congenital and complicated cataracts.
2) Previous anterior segment surgery, ocular trauma or pre-existing ocular disease.
3) Local pupil abnormalities like iris atrophy, Marfan's syndrome,synechiae.
4) Pseudoexfoliation.
5) Patients allergic to Lignocaine, topical NSAIDs , preservative or any other component of study medications.
6) Pupil size <6 mm at the start of surgery.

Methodology proper

The patients who satisfied the inclusion criteria were enrolled into the study after taking written and informed consent. The patients were explained about the pharmacological treatment they will be receiving and the possible side effects of the drugs. They were explained about the procedure of the surgery and that pupillary measurements will be taken intraoperatively.

On first OPD visit, the inclusion criteria matched patients were selected and their demographic data like age, sex, address, phone number etc were noted on the predesigned proforma.

HISTORY TAKING:

- H/O Diminution of vision RE/LE
    A. Duration
    B. Gradual/Sudden
    C. Progression/static
    D. Distant/Near vision
- Diplopia/Polyopia
- Photophobia
- Flashes of light
- Coloured halos
- Floaters
- Watering
- Redness
- Discharge
- Black spots in front of the eye
- H/O Curtain falling in front of the eyes
- H/O spectacle use
- H/O Diabetes Mellitus, Hypertension or any other systemic illness

OCULAR EXAMINATION:

- Visual acuity testing using Snellen's distant chart and Jaeger's near vision chart was taken. Best corrected visual acuity was assessed and refraction readings were taken.
- External ocular examination was done

- Slit lamp biomicroscopic examination was done for evidencing the following findings:
  - Eyelids, conjunctiva and sclera were examined to check for any infection or inflammation
  - Cornea was examined for any ulcer, opacity, pseudoexfoliation or vascularization
  - Anterior chamber depth was assessed using van herrick's method
  - Pupil was assessed for any Pseudoexfoliation material at the margin, Presence of posterior synechiae and Pupillary reaction
- IOP was measured by Non Contact Tonometer
- Maximum pupillary dilatation was measured preoperatively after instilling topical mydriatic eye drops containing tropicamide 0.8% and 5% phenylephrine hydrochloride.
- Slit lamp examination post mydriatics:
  - Pupil size was measured after complete dilatation
  - The type of cataract was assessed and grading was done
  - Lens capsule was examined for deposition of pseudoexfoliation materials
- Posterior segment evaluation was done using direct ophthalmoscope and looked for any diabetic or hypertensive retinopathy changes. The disc and macula were evaluated thoroughly.
- Keratometry readings were taken

- Intraocular lens power calculation was done by A Scan Biomicroscopy.
- Lacrimal duct patency testing was done
- Vitals like Pulse rate and Blood pressure were measured. Random blood sugar was tested.
- Xylocaine sensitivity testing was done.

PREOPERATIVE TOPICAL EYE DROPS ADMINISTRATION:

Group A- Dexamethasone Phosphate (0.1%) Moxifloxacin(0.5%) combination eye drops

Group B- Ketorolac Tromethamine (0.5 %) and Moxifloxacin(0.5%) combination eye drops

These eye drops were started 24 hours before sugery and given 6 th hourly for 4 times. It was administered as one drop at 24 hours before surgery, 18 hours before surgery, 12 hours before surgery and 6 hours before surgery.

INSTILLATION OF MYDRIATIC EYE DROPS BEFORE SURGERY:

Tropicamide 0.8% + Phenylephrine 5% eye drops was given preoperatively in the eye to be operated. It was administered as one drop in every 15 minutes interval at 60 mins before surgery, 45 minutes before surgery and 30 minutes before surgery.

OPERATIVE PROCEDURE:

All patients underwent Small Incision Cataract Surgery with Posterior Chamber Intra Ocular Lens (PCIOL) implantation. Peribulbar block was given with 2% lignocaine mixed with 1500 units/ml of hyaluronidase injection for local anaesthesia.

Surgical technique:

- Under strict and aseptic precautions peribulbar block was given.
- Universal eye speculum was put. Superior rectus bridle suture was taken.
- Conjunctival flap was raised and hemostasis was achieved using bipolar wet field cautery.
- A 6.5 mmsclero-corneal incision was given about 1-2 mm from the limbus and the sclera-corneal tunnel was dissected with crescent knife.
- Anterior chamber entry was done with keratome. Tryphan blue dye was injected and washed with normal saline after 30 seconds. Viscoelastic substance injected into AC. Continuous curvilinear capsulorrhexis was done using modified cystitome. Anterior capsule was removed using forceps.
- Hydrodissection was done by injecting saline between the nucleus and posterior capsule of the lens. Viscoelastic substance was injected into the anterior chamber. Anterior chamber entry was extended using a keratome. The lens nucleus was dialled into AC. Nucleus was delivered by phacosandwich technique using wire vectis and lens dialer.
- Thorough cortical wash was given using a two way irrigation aspiration cannula.
- A single piece polymethyl methacrylate IOL was implanted in the posterior chamber in all the cases. The IOL was dialled into position using Sinskey's hook.

- Thorough wash was given to remove the viscoelastic substance and remaining cortical matter.
- Anterior chamber was formed with normal saline
- Subconjunctival injection dexamethasone and gentamicin was given at the end of surgery

Measurement of pupillary diameter:

- Pupillary dilation was not supplemented by the use of adrenaline in the irrigating ocular solutions.
- The horizontal interpupillary diameters were measured using a Castroviejo-calliper which had markings upto 20 mm.
- Measurements were made by placing the calliper in front of the cornea under operating microscope.
- Readings were taken at four intervals - before making the incision, after nucleus delivery, following cortical lens matter removal and after IOL implantation.

- The interpupillary diameter is measured at 4 intervals:

    1. At the start of surgery
    2. After delivery of the lens nucleus
    3. After cortical matter removal
    4. After IOL implantation

- Care was taken that pupillary diameter was not affected by any of the external factors. Measurements were taken before the instillation of viscoelastics or after their removal.

- Intraoperative complications were avoided by best possible means. Injuries to the iris or the cornea were avoided.

RESULTS

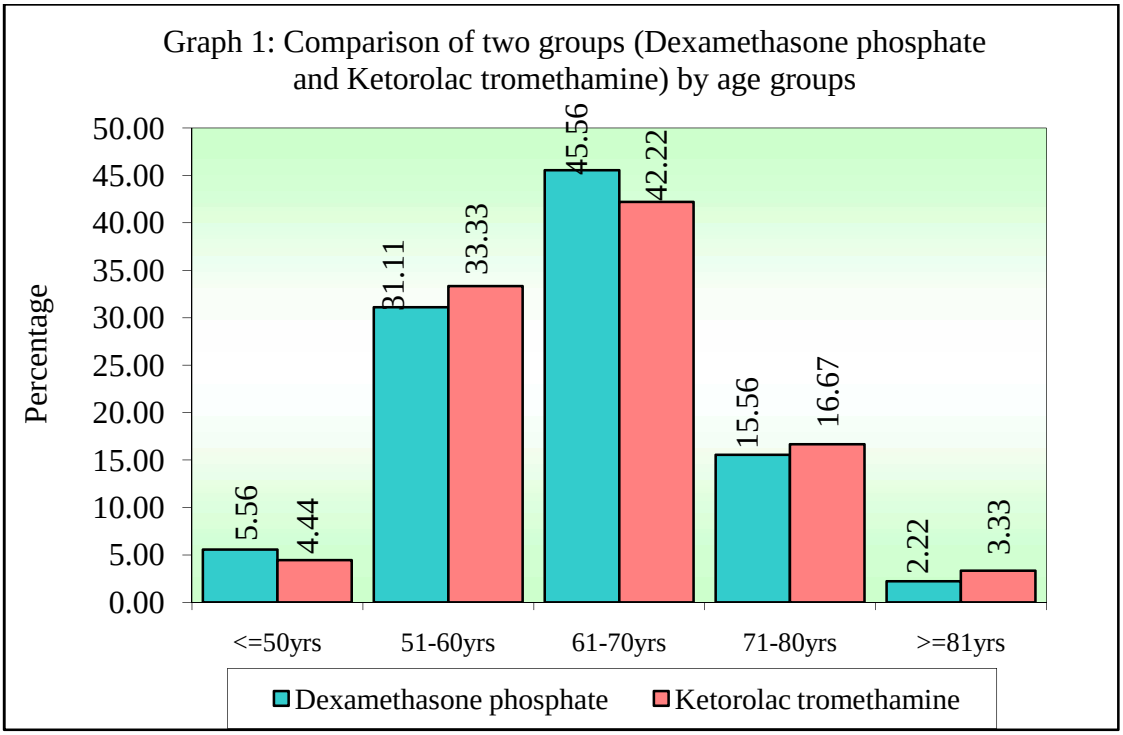
The present study was conducted in the department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi on subjects with uncomplicated cataract undergoing Small Incision Cataract Surgery. The patients were divided into two groups that is Group A (Topical Dexamethasone phosphate 0.1%) and Group B(Topical Ketorolac Tromethamine 0.5%) with 90 subjects each.

The data was tabulated on Microsoft excel spread sheet. The data was analyzed using SPSS version 20.0. For the continuous quantitative variables mean and standard deviation was calculated. The inter group continuous variables were compared using suitable tools of statistics like unpaired student's t test. Two quantitative variables, within a group, were compared using student's paired t test. Discrete variables were represented by median. Suitable graphs were used to depict the comparison. The categorical data were expressed in terms of rates, ratios and percentages. The association between the outcome, clinical and demographic characteristics were tested using Chi-square test or Fisher's exact test. For all the tests the value of p less than 5% (0.05) was considered significant.

Table 1: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by age.

Age groups	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
<=50yrs	5	5.56	4	4.44	9	5.00
51-60yrs	28	31.11	30	33.33	58	32.22
61-70yrs	41	45.56	38	42.22	79	43.89
71-80yrs	14	15.56	15	16.67	29	16.11
>=81yrs	2	2.22	3	3.33	5	2.78
Total	90	100.00	90	100.00	180	100.00
Mean age	64.16		64.47		64.31	
SD age	8.05		8.74		8.38	

The mean age of distribution in group A was 64.16 with a standard deviation of 8.05 and that of Group B was 64.47 with a standard deviation of 8.74. Most of the patients were within 61-70 years of age.



This is a graph that compares the percentage of subjects within each decade between group A and B.

Table2: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean age by independent t test

Groups	n	Mean	SD	SE	t-value	P-value
Dexamethasone phosphate	90	64.16	8.05	0.85	-0.2485	0.8040
Ketorolac tromethamine	90	64.47	8.74	0.92		

Graph 2: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean age

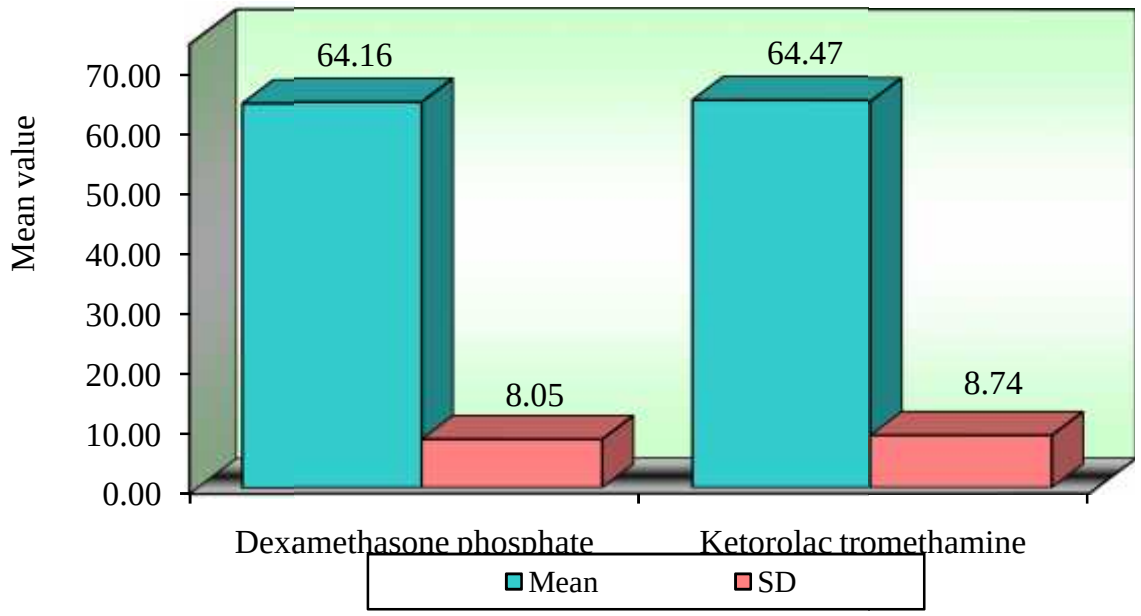
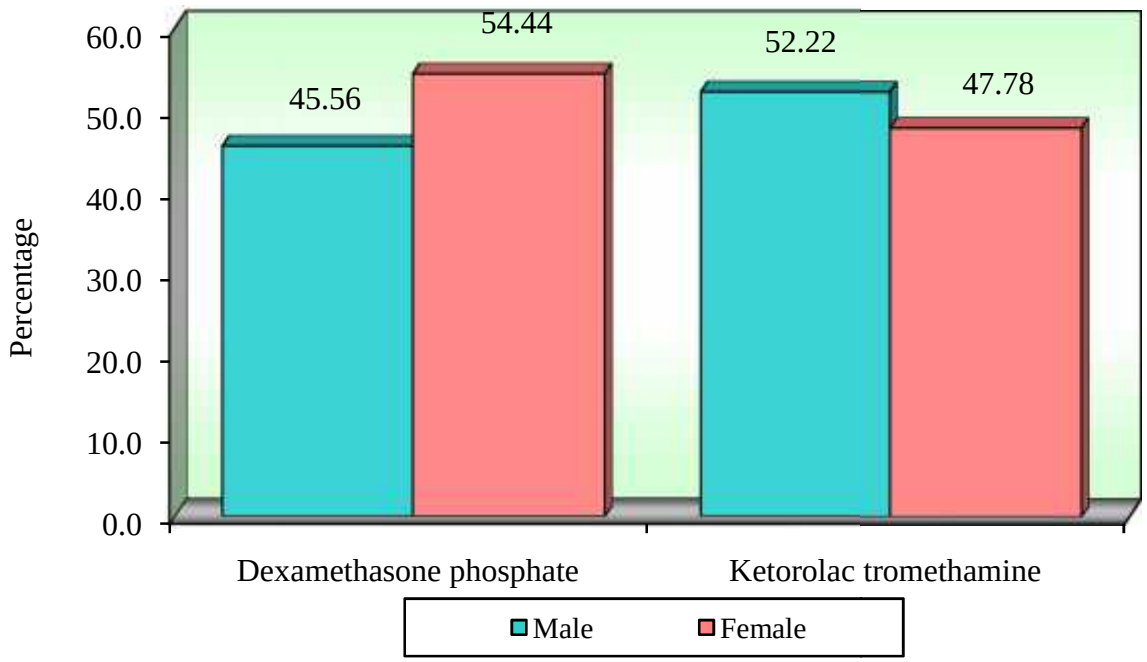


Table 3: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by gender

Gender	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
Male	41	45.56	47	52.22	88	48.89
Female	49	54.44	43	47.78	92	51.11
Total	90	100.00	90	100.00	180	100.00
Chi-square= 0.8001 P = 0.3712						

Among the 90 cases taken in group A, 41 were males and 49 were females and in group B, 47 were males and 43 were females. Predominantly 54.44 % were females on Group A and 52.22% were males in Group B.

Graph 3: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by gender



This figure shows that 45.56 % of subjects in group A were males and 54.44% of individuals were females. In group B 52.22% were males and 47.78% females.

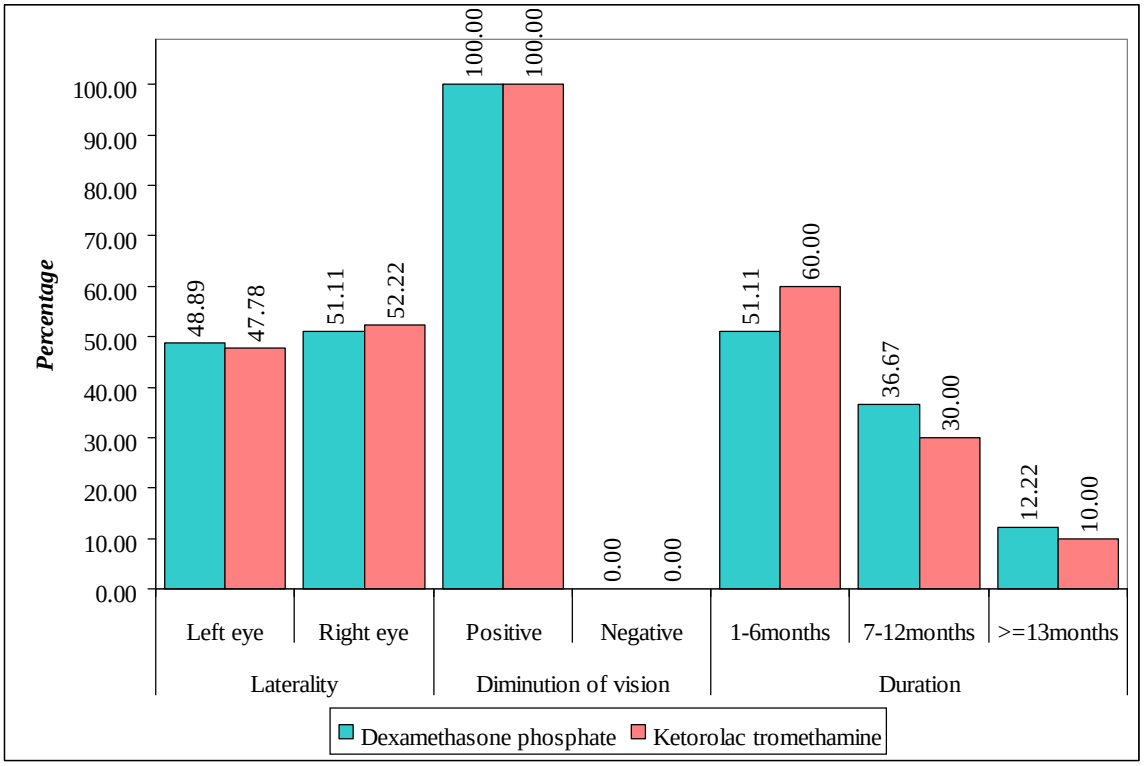
Table 4: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by chief complaints

Chief complaints	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
Laterality						
Left eye	44	48.89	43	47.78	87	48.33
Right eye	46	51.11	47	52.22	93	51.67
Total	90	100.00	90	100.00	180	100.00
Diminution of vision						
Positive	90	100.00	90	100.00	180	100.00
Negative	0	0.00	0	0.00	0	0.00
Total	90	100.00	90	100.00	180	100.00
Duration						
1-6months	46	51.11	54	60.00	100	55.56
7-12months	33	36.67	27	30.00	60	33.33
>=13months	11	12.22	9	10.00	20	11.11
Mean	9.38		8.64		9.01	
SD	7.58		7.51		7.54	
Total	90	100.00	90	100.00	180	100.00

The laterality predominant in group A and group B was Right eye. The percentage distribution was 51.11% in group A and 52.22 % in group B.

Diminution of vision was the chief complaint in all the patients. The average duration of the symptom was 9.38 months in group A and 8.64 months in group B.

Graph 4: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by chief complaints



Most patients had sypmtoms for less than 6 months. 51.11 % in Group A and 60% in Group B had symptoms less than 6 months.

Table 5: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean duration in months by independent t test

Groups	N	Mean	SD	SE	t-value	P-value
Dexamethasone phosphate	90	9.38	7.58	0.80	0.6518	0.5154
Ketorolac tromethamine	90	8.64	7.51	0.79		

Graph 5: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean duration in months

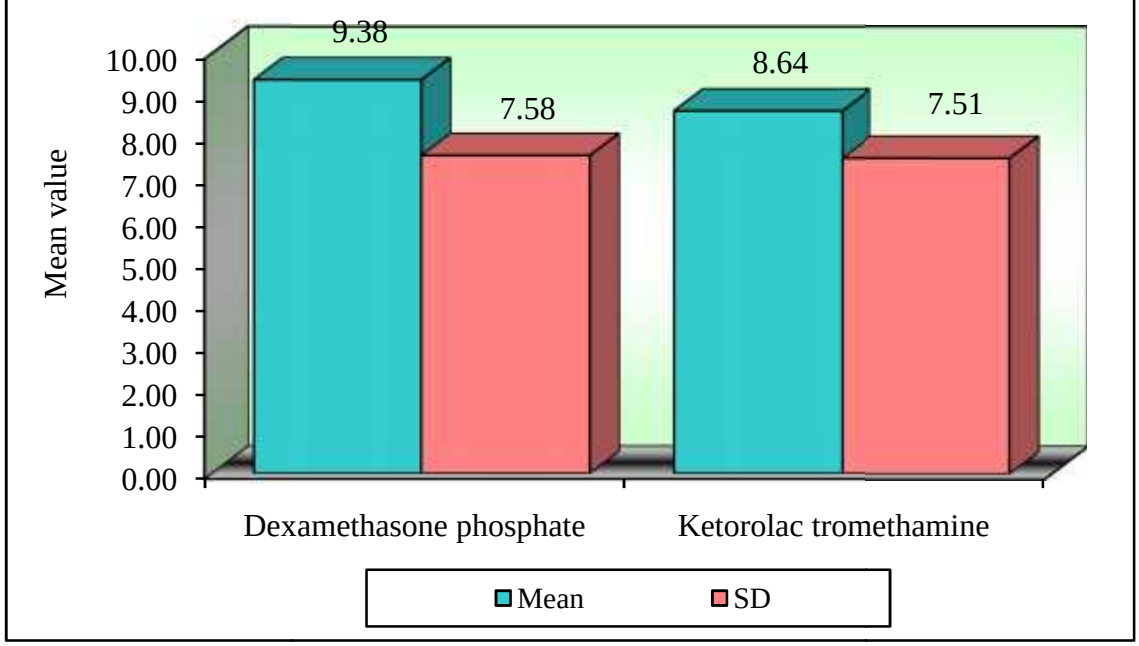


Table 6: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by past history

Fast history	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
Spectacles use						
Negative	56	62.22	50	55.56	106	58.89
Positive	34	37.78	40	44.44	74	41.11
Total	90	100.00	90	100.00	180	100.00
Chi-square= 0.8262 P = 0.3633						
Intraocular Surgery						
Negative	66	73.33	57	63.33	123	68.33
Positive	24	26.67	33	36.67	57	31.67
Total	90	100.00	90	100.00	180	100.00
Chi-square= 2.0806 P = 0.1497						
Trauma						
No	90	100.00	90	100.00	180	100.00
Yes	0	0.00	0	0.00	0	0.00
Total	90	100.00	90	100.00	180	100.00
Chi-square=0.0000,p=1.0000						
Medical History						
Asthma	0	0.00	1	1.11	1	0.56
Hypertension	19	21.11	17	18.89	36	20.00
Nil	71	78.89	72	80.00	143	79.44
Total	90	100.00	90	100.00	180	100.00
Chi-square= 0.0342 P = 0.8543						

It was found that 24(26.67%) patients in Group A and 33(36.67%) patients in Group B had H/o previous intraocular surgery. Mostly they had underwent cataract surgery of the other eye.

Group A had 19 hypertensive patients and group B had 17 hypertensive patients. The percentage distribution was 21.11 % in group A and 18.89 % in group B. Chi-square value was 0.0342 and p value was 0.8543 which was insignificant.

Graph 6: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by Past history

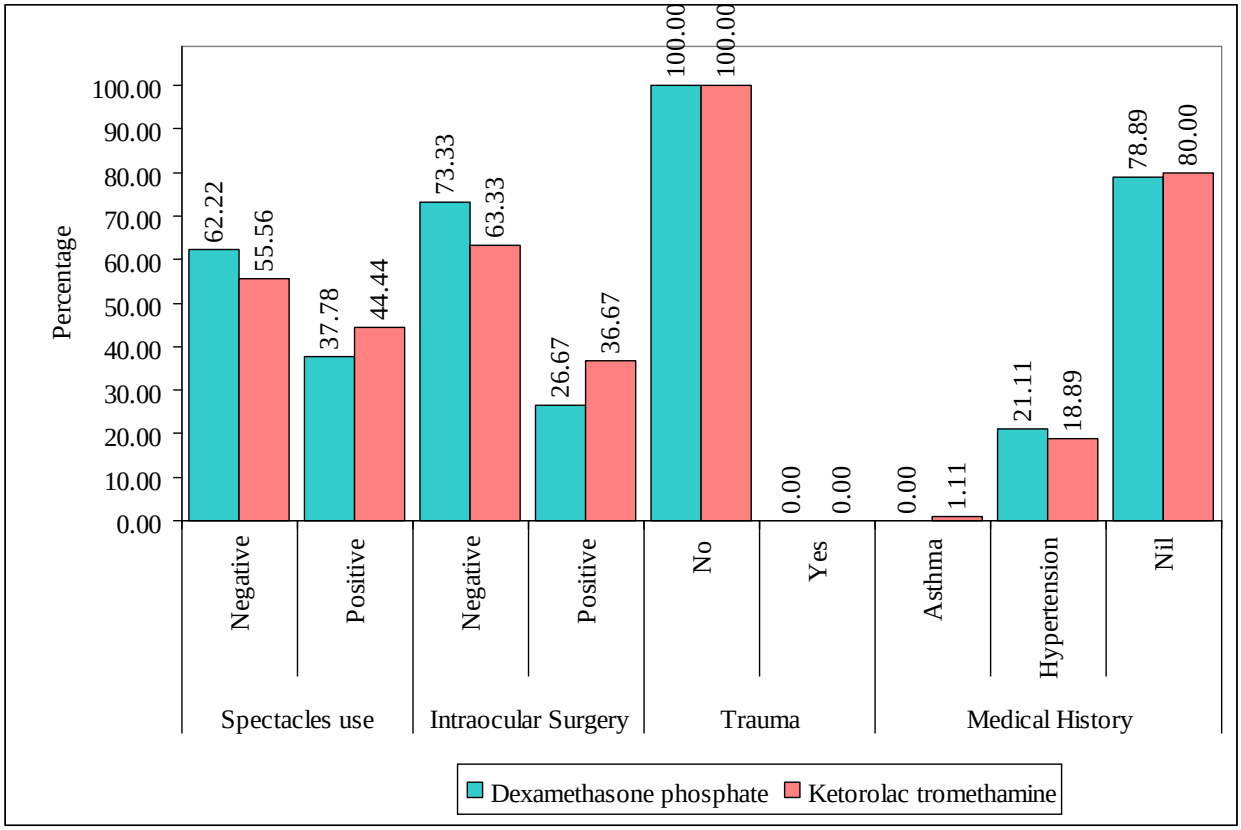


Table 7: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by Visual acuity

Visual acuity	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
6/12	1	1.11	3	3.33	4	2.22
6/18	3	3.33	4	4.44	7	3.89
6/36	10	11.11	12	13.33	22	12.22
6/60	13	14.44	15	16.67	28	15.56
CF 1 MT	19	21.11	12	13.33	31	17.22
CF 2 MT	9	10.00	9	10.00	18	10.00
CF 3 MT	9	10.00	6	6.67	15	8.33
CFCF	2	2.22	3	3.33	5	2.78
HMCF	2	2.22	10	11.11	12	6.67
PL +	15	16.67	2	2.22	17	9.44
Total	90	100.00	90	100.00	180	100.00
Chi-square= 18.8512 P = 0.0260*						

The visual acuity was defective in all patients with moderate to severe visual impairment in snellen's visual acuity testing. In Group A, most of the patients had an visual acuity of CF 1 mt (21.1 %) and group B they had an visual acuity of 6/60(16.67%).

Graph 7: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by Visual acuity

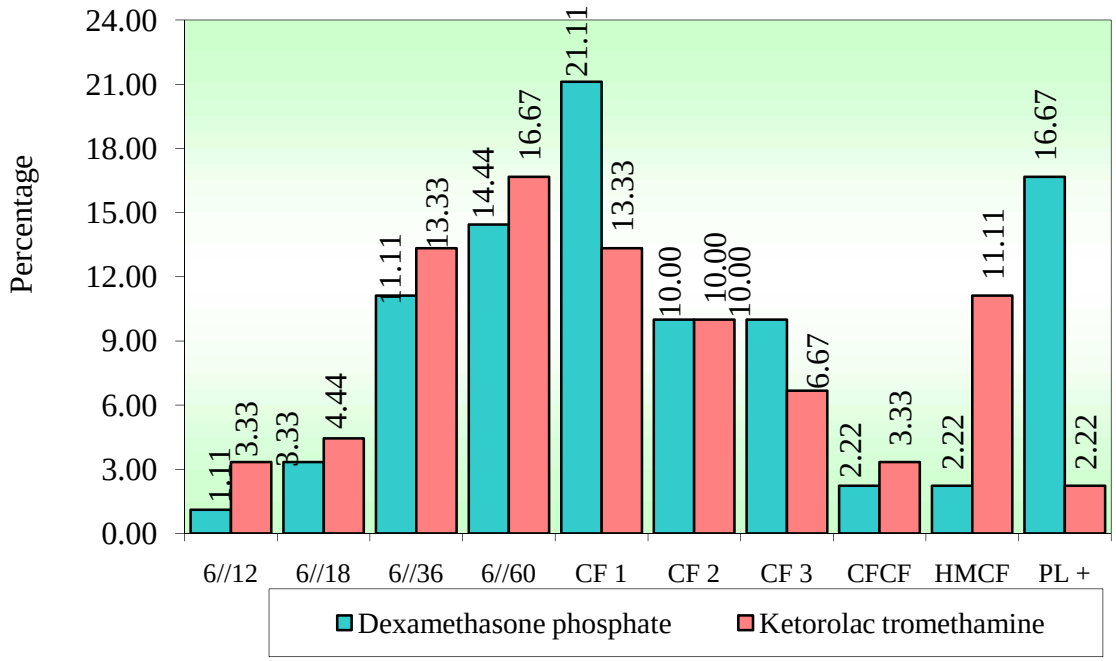


Table 8: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by other examinations

Examinations	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
Anterior Segment						
Normal	83	92.22	81	89.90	164	91.12
CO	0	0.00	2	2.22	2	1.11
Grade I/II Nasal Pterygium	7	7.78	7	7.78	14	7.78
Total	90	100.00	90	100.00	180	100.00
Posterior Segment						
Hypertensive Retinopathy	1	1.11	3	3.33	4	2.22
Normal	89	98.89	87	96.67	176	97.78
Total	90	100.00	90	100.00	180	100.00

Anterior segment examination of the patients shows that nasal pterygium was found in 7 (7.78 %) patients in Group A and B respectively. Corneal opacity was seen in 2 patients i.e 2.22% patients in Group B. Posterior segment findings were insignificant in most of the patients except in one patient in Group A and 3 patients in Group B had Hypertensive Retinopathy changes in the fundus.

Graph 8: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by other examinations

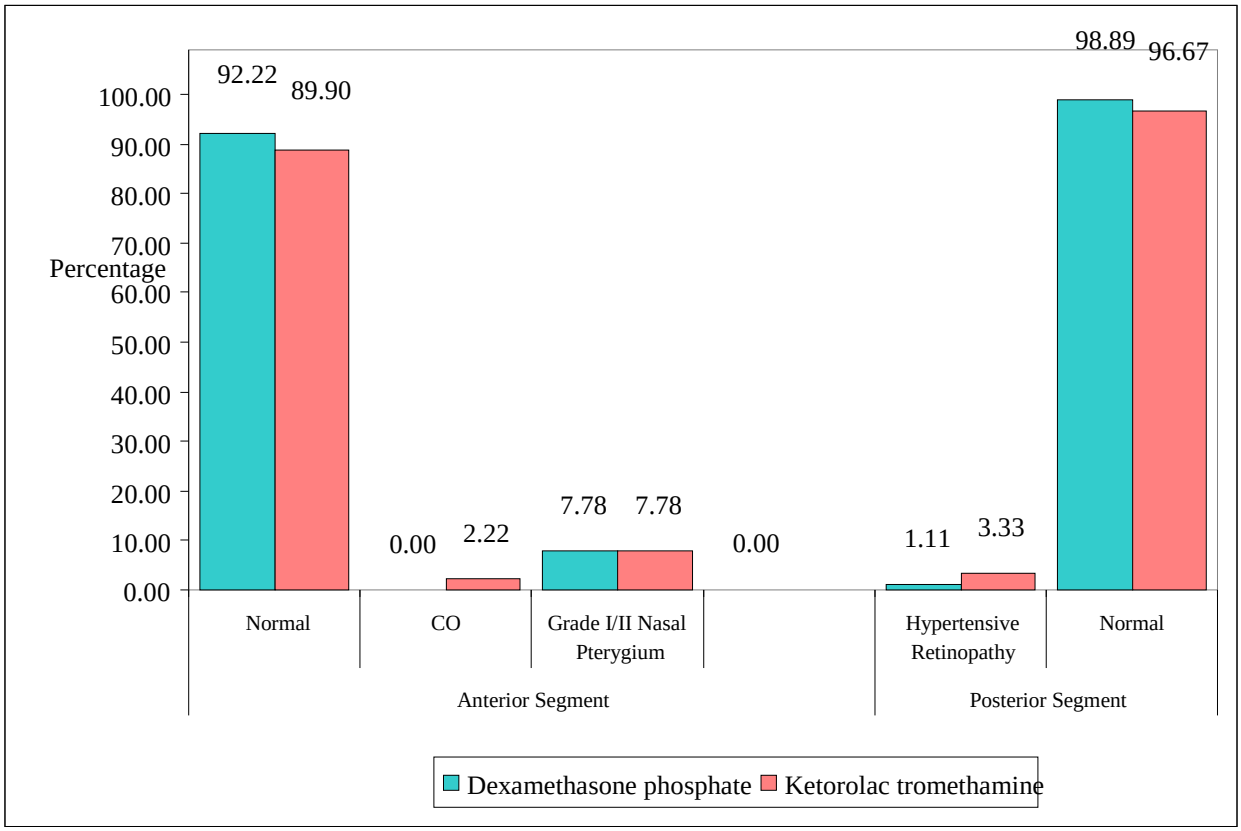
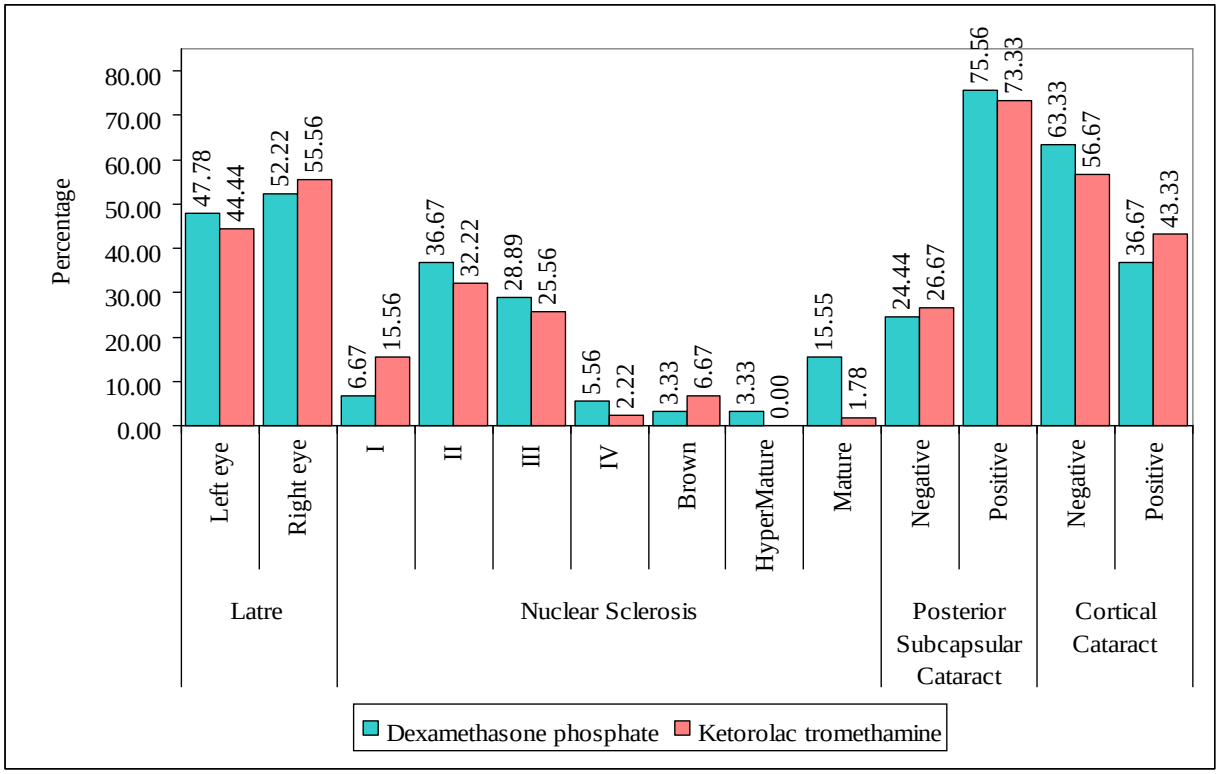


Table 9: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by Grades of cataract

Diagnosis	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
Laterality						
Left eye	43	47.78	40	44.44	83	46.11
Right eye	47	52.22	50	55.56	97	53.89
Total	90	100.00	90	100.00	180	100.00
Nuclear Sclerosis						
I	6	6.67	14	15.56	20	11.11
II	33	36.67	29	32.22	62	34.44
III	26	28.89	23	25.56	49	27.22
IV	5	5.56	2	2.22	7	3.89
Brown	3	3.33	6	6.67	9	5.00
Mature	14	15.55	16	1.78	28	15.56
Hypermature	3	3.33	0	0.00	3	1.67
Total	90	100.00	90	100.00	180	100.00
Chi-square= 9.0611 P = 0.1704						
Posterior Subcapsular Cataract						
Negative	22	24.44	24	26.67	46	25.56
Positive	68	75.56	66	73.33	134	74.44
Total	90	100.00	90	100.00	180	100.00
Chi-square= 0.1172 P = 0.7333						
Cortical Cataract						
Negative	57	63.33	51	56.67	108	60.00
Positive	33	36.67	39	43.33	72	40.00
Total	90	100.00	90	100.00	180	100.00
Chi-square= 0.8332 P = 0.3610						

It was seen that 33 (36.67 %) and 29(32.22%) were of grade II nuclear sclerosis in Group A and Group B respectively.

Graph 9: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine)by diagnosis



This graph depicts the grades of cataract and their distribution among Group A and Group B.

Table 10: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by Lacrimal Patency

Lacrimal Patency	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
Patent	90	100.00	90	100.00	180	100.00
Total	90	100.00	90	100.00	180	100.00
Chi-square=0.0000, p=1.0000						

Lacrimal patency testing was done and was found that it was patent in all patients(100%) in both Group A and B.

Table 11: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean IOP by independent t test

Groups	n	Mean	SD	SE	t-value	P-value
Dexamethasone phosphate	90	13.23	3.40	0.36	1.2721	0.2050
Ketorolac tromethamine	90	12.58	3.50	0.37		

The mean IOP in Group A was 13.23mmHg with a standard deviation of 3.40mmHg and that in Group B was 12.58 mmHg with a standard deviation of 3.50 mmHg

Graph 10: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean IOP

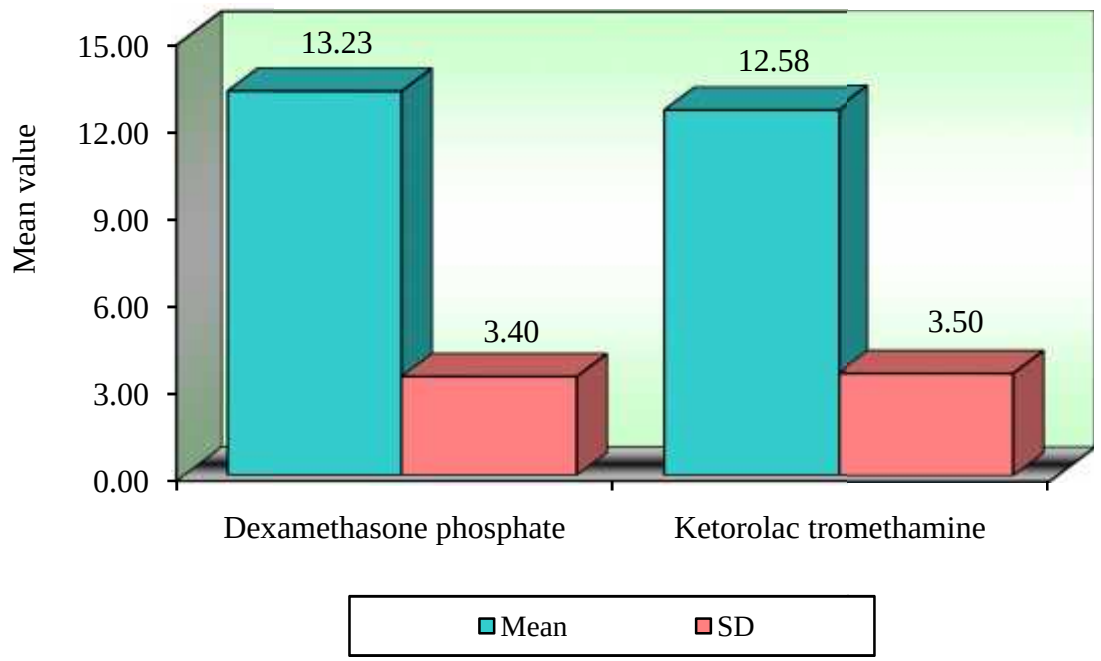


Table 12: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by Pre Op drug started one day before Surgery

Pre Op drug started one day before Sx	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
24 hrs						
Yes	90	100.00	90	100.00	180	100.00
18 hrs						
Yes	90	100.00	90	100.00	180	100.00
12 hrs						
Yes	90	100.00	90	100.00	180	100.00
6 hrs						
Yes	90	100.00	90	100.00	180	100.00

All the patients were started on preoperative eye drop either Dexamethasone phosphate or Ketorolac tromethamine eye drops depending on the group they belong to. Starting 24 hours prior to surgery they were asked to put one drop in the eye planned for surgery and every 6^{th} hourly thereafter i.e 24 hours, 18 hours ,12 hours and 6 hour before surgery.

Table13: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) by Mydriatic E/D given one hr before Sx

Mydriatic E/D one hr before Sx	Dexamethasone phosphate	%	Ketorolac tromethamine	%	Total	%
60 mins						
Yes	90	100.00	90	100.00	180	100.00
45 mins						
Yes	90	100.00	90	100.00	180	100.00
30 mins						
Yes	90	100.00	90	100.00	180	100.00

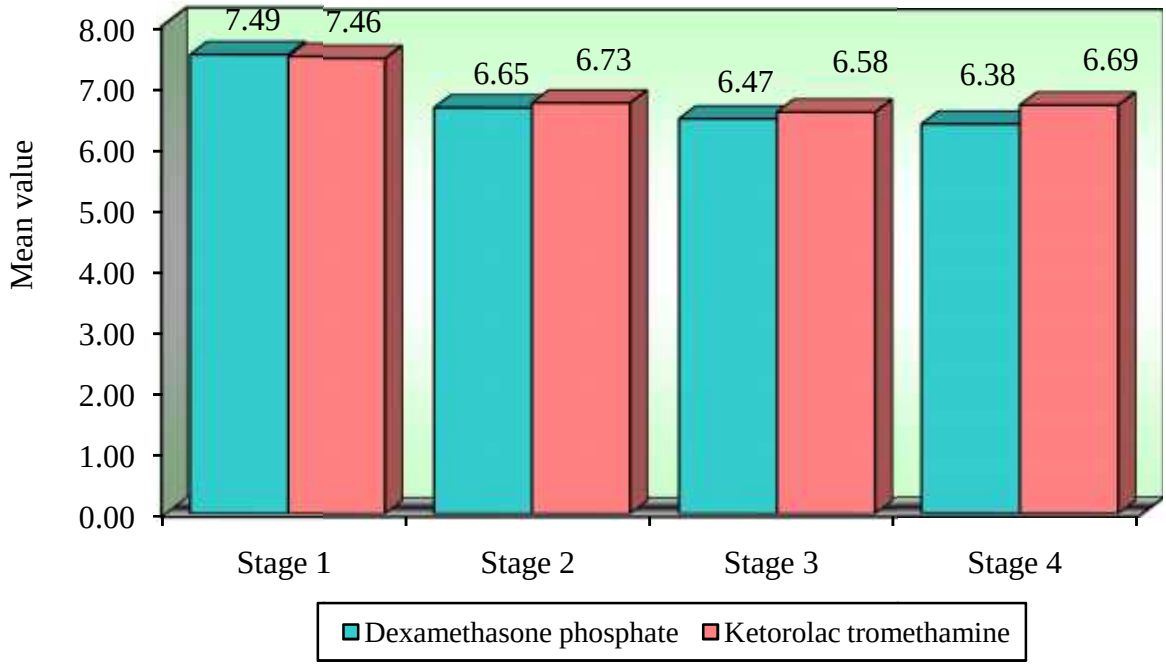
All the patients were started on mydriatic eye drops that had a combination of tropicamide 0.8% and phenylephrine 5% at about one hour before surgery. They were asked to instil one drop every 15 minutes for three times. Most of the patients were well dilated by this. Few patients required an additional one drop to attain full dilatation.

Table 14: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean Pupillary Diameter at different stages by independent t test

Variable	Groups	Mean	SD	SE	t-value	P-value
I	Dexamethasone phosphate	7.49	0.55	0.06		
	Ketorolac tromethamine	7.46	0.61	0.06	0.4510	0.6526
II	Dexamethasone phosphate	6.65	0.68	0.07		
	Ketorolac tromethamine	6.73	0.77	0.08	-0.7700	0.4423
III	Dexamethasone phosphate	6.47	0.73	0.08		
	Ketorolac tromethamine	6.58	0.83	0.09	-0.9533	0.3417
IV	Dexamethasone phosphate	6.38	0.82	0.09		
	Ketorolac tromethamine	6.69	0.91	0.10	-2.4020	0.0173*

This is the important parameter that was studied. On comparing the interpupillary diameter(IPD) at different stages of the surgery between group A and group B, it was found that the mean IPD was almost equal. At the start of surgery (stage I) , the mean IPD was 7.49 with SD of 0.55 in Group A and the mean IPD was 7.46 with SD of 0.61 in Group B. The p value was 0.6526. After lens nucleus delivery (stage II) , the mean IPD was 6.65 with SD of 0.68 in Group A and the mean IPD was 6.73 with SD of 0.77 in Group B. The p value was 0.4423. After cortical matter wash (stage III) , the mean IPD was 6.47 with SD of 0.73 in Group A and the mean IPD was 6.58 with SD of 0.83 in Group B. The p value was 0.3417. At stage IV, the mean IPD was 6.38 with SD of 0.82 in Group A and the mean IPD was 6.69 with SD of 0.91 in Group B. The p value was 0.0173. At stage 4 the p value was statistically significant.

Graph11: Comparison of two groups (Dexamethasone phosphate and Ketorolac tromethamine) with mean Pupillary Diameter at different stages



DISCUSSION

Miosis occurring during cataract removal will result in a small anterior capsulorrhexis or capsulotomy that causes difficulty in nucleus manipulation and delivery, loss of vitreous, high risk iridodialysis, retained cortical matter of lens, excessive trauma to the iris which may further result in prostaglandins release and cystoid macular oedema[60]. The reasons for small pupil can be chronic iritis, prior glaucoma, pilocarpine therapy or trabeculectomy, pseudoexfoliation syndrome, Diabetes mellitus, Post Traumatic synechiae, Tamsulosin therapy for benign prostatic hypertrophy etc. In these conditions, special technique and precautions are taken in order to deliver the nucleus.

Trauma to the ocular tissue that occurs during surgery leads to activation of phospholipase A2[61] which further liberates Arachidonic acid and Platelet Activating Factors. Arachidonic acid is metabolized through cyclo-oxygenase pathway to form PGs and lipooxygenase pathway to form leukotrienes (LTs). In case of trauma to the eye, PGs are released and induces miosis not controlled by cholinergic mechanisms[62]. In many patients prostaglandins are released immediately after the anterior chamber entry is made and so pupillary constriction occurs at an early phase. This reaction is followed by a breakdown of blood-aqueous barrier caused by PGs and other inflammatory mediators[63,64]. The inflammatory mediators are acute phase reactants that leads to postoperative complications as well.

Non Steroidal Anti Inflammatory drugs (NSAIDs) inhibit the cyclo-oxygenase enzyme, thereby inhibiting the biosynthesis of PGs but not LTs. The NSAID Ketorolac tromethamine has demonstrated efficacy in the prevention of surgically induced miosis, in the treatment of postoperative ocular pain[65], in the treatment of chronic aphakic and pseudophakic CME and in the prevention and suppression of

ocular inflammation after cataract surgery. Flurbiprofen , suprofen, ketorolac, bromfenac, Indomethacin are the drugs that are widely used to prevent intraoperative miosis.

Glucocorticoids inhibit the phospholipase A2 enzyme and consequently inhibit the biosynthesis of both platelet-activating factors and arachidonic acid. This results in the inhibition of the biosynthesis of both PGs and LTs. Prednisolone acetate 0.1% and Dexamethasone phosphate 0.1% were rarely used for the purpose preventing intraoperative miosis.

These two groups of drugs are also used postoperatively to reduce vascular permeability of the blood-aqueous barrier and thereby reducing inflammatory reactions and to control pain[66–69].

Various studies are done to study the mechanism of action and therapeutic effect of different NSAIDs[70,71]. The studies compare the efficacy of different drugs and some studies have even compared more than two drugs[72]. But the efficacy of corticosteroids in maintaining mydriasis is least studied. The mechanism of action of corticosteroid in preventing postoperative complication is done by few researchers[6]. Very few studies are available regarding the action of Prednisolone acetate 0.1%[73] and Dexamethasone phosphate 0.1% [74] and only some authors have compared the efficacy of a NSAID and Corticosteroid[69]. It is therefore vital to make an attempt in analyzing the action of a strong corticosteroid like Dexamethasone phosphate in preventing intraoperative miosis.

The present study was performed on a total of 180 eyes of 180 consecutive patients with senile cataract as selected by the inclusion criteria after taking an informed and written consent. Thedemographic data of patients were noted in a predesigned proforma.

Demographic data:

In the present study the mean age of distribution in group A was 64.16 with a standard deviation of 8.05 and that of Group B was 64.47 with a standard deviation of 8.74. The age distribution was comparable between two groups with not much difference. In Group A,45.56 % and in Group B 42.22% belonged to age 61-70 years. Among the 90 cases taken in group A, 41 were males and 49 were females and in group B, 47 were males and 43 were females. The laterality predominant in Group A and Group B was the Right eye. The percentage distribution was 51.11% in group A and 52.22 % in group B.

Diminution of vision was the chief complaint in all the patients. The average duration of the symptom was 9.38 months in group A and 8.64 months in group B. Most patients had symptoms for less than 6 months. 51.11 % in Group A and 60% in Group B have symptoms less than 6 months.

Past history details:

It is found that 24(26.67%) patients in Group A and 33(36.67%) patients in Group B have H/o previous intraocular surgery. Most of them history of cataract surgery of the other eye.

Group A had 19 hypertensive patients and group B had 17 hypertensive patients. The percentage distribution was 21.11 % in group A and 18.89 % in group B. History of hypertension did not have any bias between the two groups.

Examination :

Anterior segment examination of the patients shows that nasal pterygium was found in 7 (7.78 %) patients in Group A and B respectively. Corneal opacity was seen in 2 patients i.e 2.22% patients in Group B. Posterior segment findings were insignificant

in most of the patients except in one patient in Group A and 3 patients in Group B had Hypertensive Retinopathy changes in the fundus.

The intraoperative interpupillary diameter:

This is the important parameter that is studied in this test. On comparing the interpupillarydiameter(IPD) at different stages of the surgery between Group A and Group B, it is found that the mean IPD is almost equal. At stage I , the mean IPD is 7.49 with SD is 0.55 in Group A and the mean IPD is 7.46 with SD 0.61 in Group B. The p value is 0.6526. At stage II , the mean IPD is 6.65 with SD is 0.68 in Group A and the mean IPD is 6.73 with SD 0.77 in Group B. The p value is 0.4423. At stage III , the mean IPD is 6.47 with SD is 0.73 in Group A and the mean IPD is 6.58 with SD 0.83 in Group B. The p value is 0.3417. At stage IV , the mean IPD is 6.38 with SD is 0.82 in Group A and the mean IPD is 6.69 with SD 0.91 in Group B. The p value is 0.0173.

There was no statistically significant difference in the mean pupillary diameter between the Dexamethasone and Ketorolac treated eyes at the start of the surgery, after nucleus delivery and after cortical matter removal. But after IOL implantation the measurement of the interpupillary diameter showed a statistically significant result. Ketorolac seems to maintain mydriasis better than Dexamethasone as there is a mean difference of 0.31 mm in the interpupillary diameter.

In a study done by Zanetti F R, et al. the use of preoperative topical NSAID was compared with a placebo. All the patients achieved pupil size ≥ 6mm at the beginning of the surgery. The number of patients in the prednisolone (29/35), nepafenac (31/35) and ketorolac (30/35) groups with pupil ≥ 6mm was greater than in the placebo group in the maintenance of intraoperative mydriasis (19/35 - *P* =0.003). There was no statistical difference among the prednisolone, nepafenac and ketorolac

groups in the maintenance of intraoperative mydriasis (*P* =.791). There were no complications during surgery or related to the preoperative use of the eye drops. It was found that preoperative use of topical Ketorolac, Prednisolone and Nepafenac was effective in maintaining intraoperative mydriasis when compared to placebo [75].

In a study by Simone JN et al, to compare the anti-inflammatory and analgesic efficacy and safety of ketorolac tromethamine 0.5% ophthalmic solution with those of prednisolone acetate 1% in patients having cataract surgery it was seen that both treatments produced comparable reductions in intraocular inflammation and pain after cataract surgery and were well tolerated by patients. No adverse events were reported, and there were no significant changes in intraocular pressure in either group. Improvements in visual acuity were also similar in both groups[76].

Stewart R, et al. study on the efficacy and safety of 0.5% Ketorolac Tromethamine ophthalmic solution in the prevention of surgically induced miosis during cataract surgery. The mean change in horizontal and vertical pupil diameter from the time of the first incision to after cortical irrigation and aspiration was significantly less with active ketorolac than with vehicle (P < or = 0.014). Consequently, mean pupil diameter after cortical irrigation and aspiration was significantly greater with ketorolac than with vehicle (P < or = 0.030). It shows that Ketorolac provides effective, well tolerated and convenient inhibition of surgically induced miosis and has documented efficacy in the treatment of postoperative ocular inflammation[77].

Suleiman Y.M, et al study on the comparison of Ketorolac Tromethamine and Prednisolone Acetate in preventing surgically induced miosis during Cataract Surgery,it was seen that the mean pupil diameter change from the time of the pre-incision until after cortical irrigation and aspiration and lens implantation was

significantly less with ketorolac than with prednisolone (P = 0.003). Consequently, mean pupil diameter after cortical irrigation and aspiration and lens implantation was significantly greater with ketorolac than with prednisolone (P <0.0001). No significant differences between groups were observed in the pupil diameter before the first incision (P = 0.244), nor after administration of a miotic agent (P = 0.505). Safety variables were comparable and no drug-related adverse events were reported. It was concluded that Ketorolac tromethamine 0.5% and prednisolone acetate 1% solutions were equally well tolerated without related adverse events, but Ketorolac was better in maintaining intraoperative mydriasis.[78]

In a study by V P Gupta et al,Ketorolac tromethamine in the maintenance of intraoperative mydriasis was studied. “Sixty-three patients, undergoing extracapsular cataract extraction with posterior chamber intraocular lens implantation, were randomly divided into three equal groups. Group 1 received 20 mg of oral Ketorolac tromethamine 2 hours prior to surgery, group 2 received 0.03% Flurbiprofen sodium topical solution at the recommended dosage, and group 3 received topical normal saline in a regimen identical to that of group 2. Identical mydriatics were used in both groups. The patients pupils were measured at five stages of surgery. It was seen that the mean pupillary diameters at each surgical stage were greater in group 1 than in group 2; however, the difference was not statistically significant. The pupils in group 3 were significantly smaller than those of group 1 or group 2 (P < .05). No systemic toxicity to Ketorolac was observed. Moreover, the need for additional postoperative analgesics was significantly greater in groups 2 and 3. Oral Ketorolac is safe, convenient, and as effective as topical Flurbiprofen, and has the added advantage of reducing postoperative pain[79].

Srinivasan et al, studied on topical ketorolac tromethamine 0.5% versus diclofenac sodium 0.1% to inhibit miosis during cataract surgery it was noted that the mean horizontal pupil diameter was 7.40 mm at the start of surgery in both groups. The ketorolac group showed a consistent trend toward larger pupil diameters at subsequent surgical intervals. Changes from baseline also indicated more significant inhibition of miosis in the ketorolac group. Topical ketorolac was a more effective inhibitor of miosis than topical diclofenac during extracapsular cataract extraction and IOL implantation. It also provided a more stable mydriatic effect throughout surgery[58].

George L. Spaeth studied the effect of autonomic agents on the pupil and the intraocular pressure of eyes treated with Dexamethasone. It was found that Dexamethasone produced a slight increase in pupil size which was transient[80].

Another study conducted by Solomon KD et al, on comparison of the effects of topical 0.5% ketorolac tromethamine ophthalmic solution with topical 0.03% flurbiprofen sodium ophthalmic solution it was shown that the mean horizontal pupillary diameter measurements for both medications were similar at the start of surgery. However, a consistent trend of larger pupillary diameter was seen in all subsequent surgical intervals in the ketorolac-treated group. Changes from baseline measurements also indicated a more significant inhibition of miosis at all subsequent intervals, and a more stable mydriasis throughout the procedure in the ketorolac-treated group.

In a study by CMahdy MA-MS on "Effect of flurbiprofen and dexamethasone acetate in prevention of surgically induced miosis during cataract surgery" the role of dexamethasone in maintaining mydriasis was demonstrated[81].

A research done by Sethi S, Chalia D, Chalia M, Kaur M on "A study on effect of pre-operative topical prednisolone acetate, nepafenac and placebo, on intraoperative mydriasis and its sustenance during cataract surgery" the role of prednisolone in maintaining mydrasis was elaborated more. All the patients in the study achieved pupil ≥6 mm at the beginning of the surgery. The number of patients in the prednisolone (16/20) and nepafenac (17/20) groups with pupil ≥6 mm was greater than in the placebo group in the maintenance of intraoperative mydriasis (7/20 – p=0.003). There was no statistically significant difference among the prednisolone and nepafenac groups in the maintenance of intraoperative mydriasis (p=0.791). There were no complications during surgery or related to the pre-operative use of the eye drops[82].

In a study by Sarkar S, Mondal KK, Roy SS, Gayen S, Ghosh A, De RR. Comparison of preoperative nepafenac (0.1%) and flurbiprofen (0.03%) eye drops in maintaining mydriasis during small incision cataract surgery in patients with senile cataract.A total of 70 eyes of cataract surgery patients, 33 males and 37 females, with a mean age of 58.5 ± 11.24 years, were included in the study. The mean horizontal and vertical diameters of the two groups were similar at the start of surgery. Significant differences were seen after IOL implantation, with the nepafenac group having the larger mean diameters in both horizontal (P = 0.03) and vertical (P = 0.04) pupillary measurements. Topical nepafenac has been shown to be a more effective inhibitor of miosis during SICS and provides a more stable mydriatic effect compared to topical flurbiprofen[83] .

Sharma AK, et al.did a study on the "Comparison of Dexamethasone Phosphate and Ketorolac Tromethamine in maintaining intraoperative mydriasis during Small Incision Cataract Surgery". The two drugs showed no statistically

significant difference in pupillary diameter at the commencement of surgery (p=0.435). The difference between the two drugs was statistically significant, for the mean pupillary diameter which changed from the start of surgery to after cortical clean-up. At this stage, ketorolac group showed a tendency towards larger mean pupillary diameter than dexamethasone group (6.70 ± 0.85mm and 6.32 ± 0.84mm, respectively, p=0.002)whereas in the present study the pupillary diameter was remaining equivalent between the two groups at this stage. Again, ketorolac group patients had larger pupillary diameter after IOL implantation than dexamethasone group patients (the mean was 6.16± 0.97mm and 5.75 ± 0.73mm, respectively, p=0.001) which was consistent with the current study. It was concluded that both Ketorolac tromethamine (0.4%) and Dexamethasone phosphate (0.1%) are effective in maintaining adequate mydriasis during cataract surgery, but the comparative analysis of the two drugs concludes that, ketorolac is definitely a better option in preventing surgically induced miosis. concluded that both the drugs are effective in maintaining adequate mydriasis during cataract surgery but Ketorolac is definitely better in preventing surgically induced miosis[84].

The number of time that the drug should be instilled and the number of drops to be instilled was given by O'Brien TP in his "Emerging guidelines for use of NSAID therapy to optimize cataract surgery patient care" article. They have mainly discussed on the dosage of drugs to prevent postoperative inflammation especially Cystoid Macular Edema.

Many studies are conducted in patients undergoing Phacoemulsification surgery and the effect of the NSAIDs or Steroids in maintaining mydriasis during the various steps have been elaborated.

Atanis et al did a research on the "Effect of topical Ketorolac tromethamine and topical Nepafenac on maintaining pupillary dilation during phacoemulsification". A total of 47 eyes of 44 cataract surgery patients, 13 males and 34 females, with a mean age of 66.04 ± 8.87 years, were included in the study. The mean horizontal and vertical diameters of the three groups were similar at the start of surgery. Significant differences were seen after IOL implantation, with the Nepafenac group having the largest mean diameters in both horizontal ($p = 0.012$) and vertical ($p = 0.012$) pupil measurements. It concluded that topical Nepafenac has been shown to be a more effective inhibitor of miosis during phacoemulsification and provides a more stable mydriatic effect throughout the surgical procedure compared to topical ketorolac and placebo[85].

Thus the present study has proved that Ketorolac tromethamine 0.5% is the better drug in maintaining mydriasis during Small Incision Cataract Surgery at all stages of surgery. We had mainly analyzed the effect of the drug in uncomplicated cataract with no systemic diseases except hypertension. The effect of these drugs needs to be studied in patients with complicated cataract as well. The study corroborate well with the above quoted studies that both the corticosteroids and the NSAID group of drugs are efficacious in inhibiting surgically induced miosis during cataract surgery since both the drugs prevent PGs release caused by ocular trauma either by inhibition of phospholipase-A2 enzyme (Dexamethasone) or cyclo-oxygenase enzyme (Ketorolac tromethamine), but NSAIDs are superior to corticosteroids in terms of maintaining a consistently larger pupil size and having a more prolonged effect. The hypothesis given in support of this states that Dexamethasone inhibit platelet activating factors (PAFs) which probably are

beneficial in maintaining mydriasis during surgery, while this mechanism of action is lacking in Ketorolac tromethamine.

CONCLUSION

The present study has documented the efficacy of both Dexamethasone and Ketorolac eye drops in maintaining intraoperative mydriasis during Small Incision Cataract Surgery. Ketorolac tromethamine 0.5% is effective in preventing miosis during cataract surgery. Dexamethasone being a potent steroid has almost equivalent effect as compared to ketorolac. However, Dexamethasone eye drops are known to cause more side effects as compared to Ketorolac, like increased IOP, posterior subcapsular opacification etc. Both these drugs can be given as a preoperative medication along with an antibiotic like Moxifloxacin in patients planned for cataract surgery. These drugs also help cataract surgeons in controlling postoperative pain and inflammation.

Therefore, we may conclude that both Ketorolac tromethamine (0.5%) and Dexamethasone phosphate (0.1%) are effective in maintaining adequate mydriasis during cataract surgery, but the comparative analysis of two drugs have shown that Ketorolac is definitely a better option in preventing surgically induced miosis. Further studies with a larger sample size is required for a conclusive evidence.

SUMMARY

Small Incision Cataract Surgery (SICS) is a most common procedure used to treat cataracts in elderly population especially in a developing country like India. It is the technique which uses cheaper instruments and machines but gives equivalent results comparable to Phacoemulsification technique. Delivering the cataractous nucleus without much complications, removal of cortical matter and placing an IOL are the most challenging step in SICS. Though the incision made is small compared to conventional Extra Capsular Cataract Extraction, other steps are similar and so the surgical trauma produced by the procedure is almost the same. Surgical trauma produced on the ocular tissues like cornea, iris or the lens will trigger a cascade of reactions releasing Prostaglandins, Leukotrienes and other inflammatory mediators. These are the substances that causes intraoperative miosis.

The release of PGs can be inhibited by NSAIDs which acts on the cyclo-oxygenase pathway. Drugs like Ketorolac 0.4% or 0.5%, Flurbiprofen0.03%, Nepafenac 0.1%, Bromfenac 0.07% etc have been tested and used for the purpose of preventing intraoperative mydriasis and thereby maintaining mydriasis.The NSAIDs are not effective against other inflammatory mediators like leukotrienes. Steroids are also useful for this purpose and even more they are expected to be more efficacious in maintaining mydriasis as they act at an earlier step of cyclo-oxygenase and lipooxygenase pathway. They may be more potent as they inhibit both PGs and Leukotrienes production and also inhibitors of other inflammatory mediators. They have a very important role in preventing post-operative complications.

In the present study, a total of 180 subjects were selected and were randomized into two groups. Group A was the Dexamethasone phosphate 0.1% and Group B was the Ketorolac tromethamine 0.5%. Written and informed consent was

got from all the patients who were enrolled for the study. A detailed history was taken and the study subjects were evaluated for visual acuity, anterior segment examination under slit lamp, posterior segment examination using ophthalmoscope and grading of the cataract was done. Preoperative assessment of lacrimal gland patency, IOP measurement, A scan biometry and keratometry for IOL power calculation were done.

All the patients participating were given preoperative eye drops 24 hours prior to surgery every 6^{th} hourly for 4 times. They were given either Dexamethasone phosphate 0.1% or Ketorolac tromethamine 0.5% eye drops depending on the group they are in.On the day of surgery, Tropicamide 0.8% with phenylephrine 5% eye drops were instilled one hour prior to surgery for three times. Intraoperatively interpupillary diameter were measured using a Castroviejo caliper at four stages that is at the start of the surgery, after nucleus delivery, after cortical matter removal and after the placement of IntraOcular Lens.

The results were tabulated and analysed. The study was focused on comparison of two groups. The association between the outcome, clinical and demographic characteristics are tested using Chi-square test or Fisher's exact test. For all the tests the value of p less than 5% (0.05) is considered significant.

The present study showed that the age, sex, laterality, anterior and posterior segment evaluation, preoperative examinations were are comparable between the two groups. The interpupillary diameter measured was statistically significant (p= 0.0173) in the Ketorolac tromethamine 0.5% group (Group B) after IOL implantation. Thus, Ketorolac serves as a better drug in maintaining intraoperative mydriasis during Small Incision Cataract Surgery when given preoperatively. The action of Dexamethasone is also as comparable to that of Ketorolac in all the other stages of the surgery. So, it can be concluded in terms of the results that both Dexamethasone and Ketorolac are as

effective as each other in maintaining intraoperative mydriasis in SICS , however Ketorolac is marginally better acting. Further studies with larger sample size is therefore needed in order to draw a stern conclusion.

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ANNEXURE- I

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
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Ref: MDC/DOME/ 13 **Date: 24/11/2018**

To,

REG. NO.BK0118004
PG student in Ophthalmology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"COMPARISON OF PREOPERATIVE TOPICAL DEXAMETHASONE PHOSPHATE VERSUS KETOROLAC TROMETHAMINE IN MAINTAINING INTRAOPERATIVE MYDRIASIS DURING SMALL INCISION CATARACT SURGERY – A ONE YEAR RANDOMIZED CONTROL TRIAL AT KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE II

INFORMED CONSENT

Title Of Research Study:COMPARISON OF PREOPERATIVE TOPICAL DEXAMETHASONE PHOSPHATE AND KETOROLAC TROMETHAMINE IN MAINTAINING MYDRASIS DURING SMALL INCISION CATARACT SURGERY- A ONE YEAR RANDOMISED CONTROLLED STUDY AT DR PRABHAKAR KORE CHARITABLE HOSPITAL AND MRC, BELAGAVI.

Principal Investigator:-

REG. NO.BK0118004

Post Graduate Student,

Department Of Ophthalmology,

JNMC, Belgaum.

Guide:

Dr. _____

MBBS ,MS

Professor

Department of Ophthalmology,

JNMC, Belagavi.

Introduction and Purpose:-

The following randomised controlled study is being undertaken to compare dexamethasone phosphate and ketorolac tromethamine in maintaining intraoperative mydrasis during small incision cataract surgery. In order to maintain mydrasis topical eye drops either dexamethasone and moxifloxacine combination or ketorolac and

moxifloxacine combination will be instilled 4 times starting from 24 hours before surgery every 6 hours. In addition topical mydriatic eye drops will be given three times from 1 hour before surgery every 15 minutes.

Procedure: If you agree to be part of the research study, you will be asked about the history related to your disease and will be subjected to assessment of visual acuity, refraction, intraocular pressure measurement, sac-syringing and anterior and posterior segment assessment of eye.

Risk and Benefits:

The risks associated with small incision cataract surgery are applicable here. You will have the best possible corrected vision post cataract surgery and complications occuring due to intraoperative miosis like difficult nucleus manipulation and deliver, vitreous loss, retained lens matter, high risk of iridodialysis, excessive iris tissue handling with subsequent prostaglandin release and cystoid macular edema can be prevented.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study. If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy: Does not apply to this research

Financial incentives for participation: You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MS degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

1**REG. NO.BK0118004**
Investigator,
PG in Ophthalmology,
J.N.M.C.,Belagavi

2. _____
Professor
Dept.of Ophthalmology
J.N.M.C.,Belagavi

3. Dr. Roopa Bellad MBBS MD DCH
Chairman
J.N.M.C. Ethical Committee
for Human Research
J.N.M.C.,Belagavi

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, and I have had all the questions answered.

Signature / Left Thumb print of the Participant or legally authorized representative:

.....

Participant's name:

…….....

Signature / Left thumb impression of the participant:

.....…

Name of the legally authorized representative/guardian:

…….....

Signature / Left thumb impression:

.....……...

Witness' name:

.....

Signature / Left thumb impression:

.....……

Investigator's name and signature:

.....……

Date:

Place:

ANNEXURE III

PROFORMA

GENERAL INFORMATION

IP NUMBER: PATIENT ID NUMBER:

NAME:

_____

AGE: _____ GENDER: F/M CONTACT NUMBER: _____

ADDRESS:

_____

_____

_____

DATE OF ADMISSION: _____ DATE OF DISCHARGE: _____

Is the patient eligible for the study? YES/NO

Has informed consent been given? YES/NO

Final result information:

1. Ineligible
2. Eligible –Refusal
3. Eligible – Participating

CHIEF COMPLAINTS

Diminution of vision: RE/LE/BOTH EYES

Duration: RE: _____ days/months/years

LE: _____ days/months/years

HISTORY OF PRESENTING ILLNESS

Diminution of vision: Gradual/Sudden

Progressive/Static

Painless/Painful

For distance/For near/For both distance and near

Diplopia: Present/Absent

Coloured halos: Present/Absent

Black spots before the eyes: Present/Absent

Watering: Present/Absent

Redness: Present/Absent

Discharge: Present/Absent

Clear/Whitish

Serous/Mucoid

Spectacle use: Distance/Near/Both

Duration: _____ days/months/years

Last refraction done: _____ days/months/years

back

PAST HISTORY

Ocular surgery: Yes/No Type of Surgery: _____

Duration: _____ days/months/years

Diabetes: Yes/No

Duration: _____ days/months/years

Hypertension: Yes/No

Duration: _____ days/months/years

Any other medical disorders:

_____

_____

PERSONAL HISTORY

Smoking: Yes/No

Duration: _____ days/months/years

Alcoholism: Yes/No

Duration: _____ days/months/years

Other addictions: Yes/No

Duration: _____ days/months/years

GENERAL PHYSICAL EXAMINATION

General appearance: Well-built/Moderately built/Poorly built/Emaciated

Pallor: Present/Absent If present: Mild/Moderate/Severe

Pulse: _____ beats/minute BP: _____ mmHg

Temperature: _____ ^{o}F Respiratory Rate: _____/minute

SYSTEMIC EXAMINATION

CVS: Normal/Abnormal

Specify:

_____

RS: Normal/Abnormal

Specify:

_____

CNS: Normal/Abnormal

Specify:

_____

GIT: Normal/Abnormal

Specify:

_____

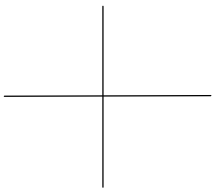
OCULAR EXAMINATION

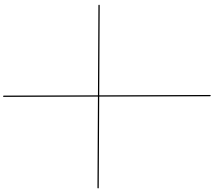
Head posture: Erect/Tilted

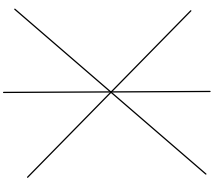
Visual axis: Parallel/Deviated

Facial symmetry: Symmetrical/Asymmetrical

Extra-ocular movements: Normal/Restricted

RE: 

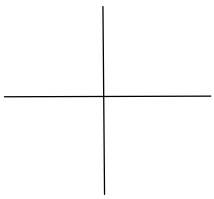
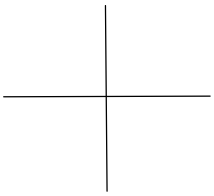
LE: 

Binocular: 

VISUAL ACUITY:

	OD	OS
UNAIDED		
PINHOLE		
WITH SPECTACLES		

RETINOSCOPY:

RE:  LE: 

REFRACTION:

RE	SPH	CYL	AXIS	SPH	CYL	AXIS	LE
V							V
NV							NV

	OD	OS
LID		
ADNEXA		

CONJUNCTIVA		
SCLERA		
CORNEA		
ANTERIOR CHAMBER		
IRIS		
PUPIL A. Size B. Shape C. Direct D. Indirect E. Near reflex	 _____ in mm Present/Absent Present/Absent Present/Absent	 _____ in mm Present/Absent Present/Absent Present/Absent
LENS	Clear/Opaque Aphakia/Pseudophakia Immature/Mature/Hypermature NS/CC/PSC Grade – I / II / III / IV	Clear/Opaque Aphakia/Pseudophakia Immature/Mature/Hypermature NS/CC/PSC Grade – I / II / III / IV

Fundus Examination	OD	OS
GLOW		
MEDIA		
DISC		

1. Size		
2. Shape		
3. Colour		
4. NRR		
5. Vessels		
6. Lamellar Dot Sign		
7. Haemorrhagic Spots		
8. Other Signs		
C:D RATIO		
BLOOD VESSELS		
BACKGROUND		
MACULA		

DIAGNOSIS:

_____

_____

INVESTIGATIONS:

1. Lacrimal Patency:

	Patent	Regurgitation		Blocked
		Clear Fluid	Mucoid/Purulent	
RE				
LE				

2. IOP:

	By NCT	By Schiotz		
		5.5g	7.5g	10.0g
RE				
LE				

3. A-Scan: RE/LE

   K_H

   K_v

   Axl:

   ACD:

   PCIOL:

4. BLOOD SUGAR:

   Random Blood Sugar(mg/dl) -

   Fasting Blood Sugar(mg/dl) -

5. Blood Pressure(mmHg) -

TREATMENT GIVEN PREOPERATIVELY:

PREOPERATIVE TOPICAL EYE DROPS ADMINISTRATION:

DRUG GIVEN- DEXAMETHASONE/KETOROLAC

INSTILLATION OF EYEDROPS FROM 24 HOURS BEFORE SURGERY:

	DOSAGE OF EYEDROP	ADMINISTERED
1.	24 HOURS BEFORE SURGERY	
2.	18 HOURS BEFORE SURGERY	
3.	12 HOURS BEFORE SURGERY	
4.	6 HOURS BEFORE SURGERY	

INSTILLATION OF MYDRIATIC EYE DROPS BEFORE SURGERY:

DRUG GIVEN-

	DOSAGE OF EYEDROP	ADMINISTERED
1.	60 MINUTES BEFORE SURGERY	
2.	45 MINUTES BEFORE SURGERY	
3.	30 MINUTES BEFORE SURGERY	

OPERATIVE PROCEDURE:

Surgery: Small Incision Cataract Surgery with PCIOL

Date: _____ Eye to be operated: Right/ Left

ANAESTHESIA: Peribulbar block/ Topical

INCISION: Superior/Temporal/Supero-temporal/Infero-temporal

OPERATIVE COMPLICATIONS: Present/Absent

If present, specify -

_____

INTRAOPERATIVE MEASUREMENT OF PUPILLARY DIAMETER:

	MEASUREMENT OF PUPILLARY DIAMETER	READINGS
1.	AT THE START OF SURGERY	
2.	AFTER DELIVERY OF NUCLEUS	
3.	AFTER CORTICAL LENS MATTER REMOVAL	
4.	AFTER IOL IMPLANTATION	

POST-OPERATIVE COMPLICATIONS: Present/Absent

If present, specify -

_____

_____

OPERATING SURGEON:

_____

SURGEON'S SIGNATURE:

ANNEXURE IV –PHOTOGRAPHS

Photograph: 1 Dexamethasone phosphate 0.1% Eye drops

Photograph 2: Ketorolac tromethamine 0.5% eye drops

Photograph 3: Topical Mydriatic Containing Tropicamide 0.8% AndPhenylephrine Hydrochloride 5% With Chlorbutol As A Preservative.

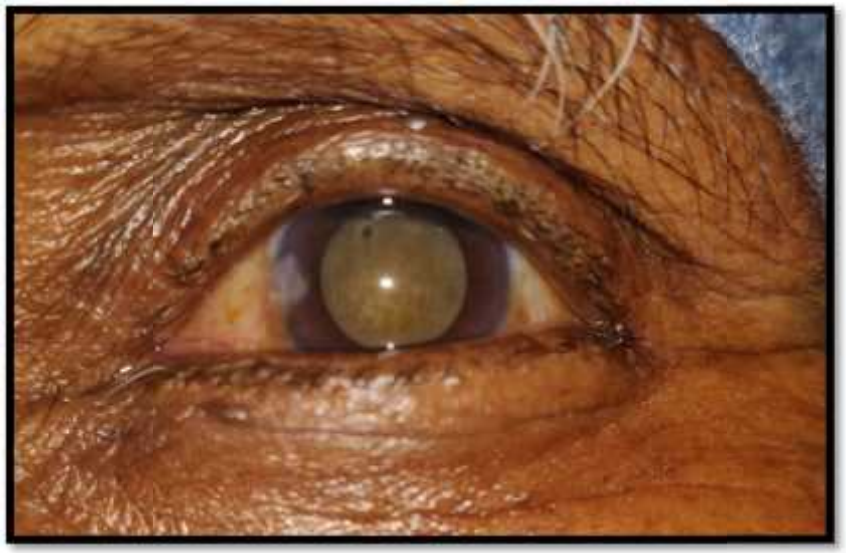
Photograph 4: Local anaesthetic – Lignocaine Hydrochloride 2 %

Photograph 5: Cataract set

Ptotograph 6: Castro- viejo calliper

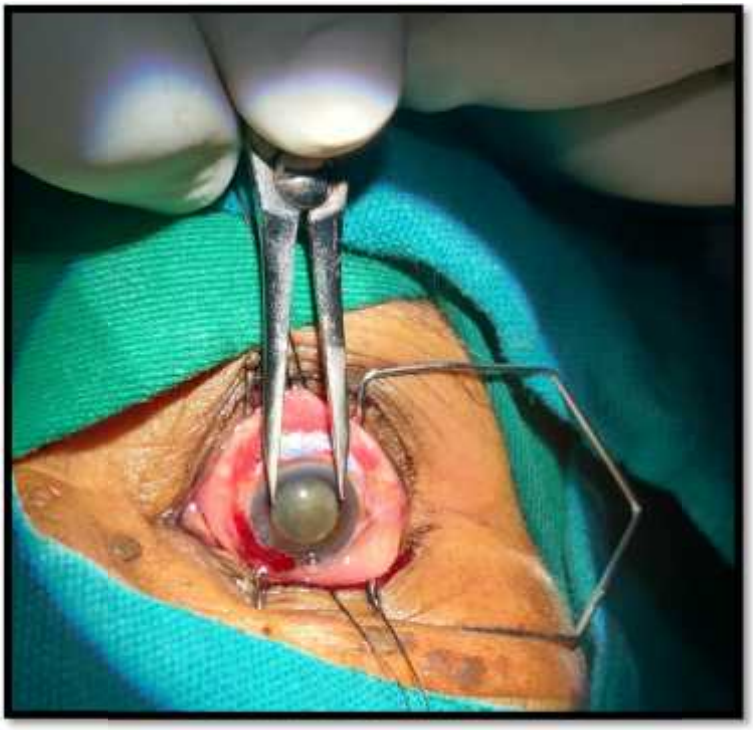
Photograph 7: Operating Surgeon

Photograph 8: Pupil at the start of surgery



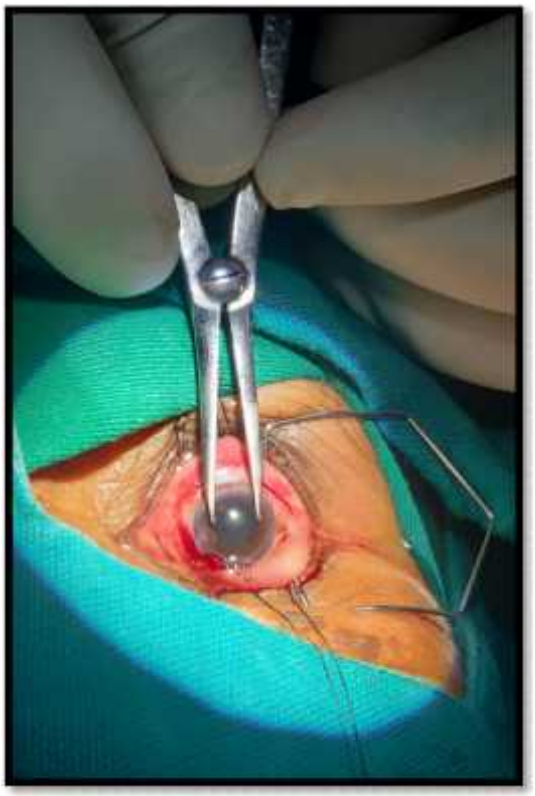
Photograph 9: Measurement of Inter Pupillary Diameter at the start of surgery

'



Photograph 10: Measurement of pupillary diameter after cortical wash

Photograph 11: Measurement of pupillary diameter after IOL implantation



ANNEXURE VI

KEY TO MASSTER CHART

1. AS- Anterior Segment
2. B – Brown
3. CC- Cortical Cataract
4. CO- Corneal Opacity
5. CF – Counting Fingers
6. D – Duration
7. DOV – Diminution of Vision
8. F – Female
9. GI/II NP- Grade I/II Nasal Pterygium
10. HM – Hypermature
11. HMCF – Hand Movements Close to Face
12. H/O – History Of
13. HTN – Hypertension
14. HR – Hypertensive Retinopathy
15. Hrs – Hours
16. IOP – Intra Ocular Pressure
17. IOS – Intra Ocular Surgery
18. IP – In Patient
19. Lat – Laterality
20. LE – Left Eye
21. LP – Lacrimal Patency
22. Ma – Male
23. M – Mature

24. MH – Medical History
25. Mm - Millimeter
26. N - Normal
27. NS – Nuclear Sclerosis
28. P – Patent
29. PD – Pupillary Diameter
30. PL – Perception of Light
31. Preop E/D – Preoperative Eye Drops
32. PS – Posterior Segment
33. PSC- Posterior Subcapsular Cataract
34. RE – Right Eye
35. SN- Serial Number
36. SP – Spectacle use
37. Stage 1 – At the start of surgery
38. Stage 2- After nucleus delivery
39. Stage 3 – After cortical matter removal
40. Stage 4 – After IOLimplantation
41. Sx- Surgery
42. Tr - Trauma
43. VA- Visual Acuity
44. Y- Yes
45. Yrs – Years

SN	Age(yrs)	Sex	Chief complaints				Past history			Examination			Diagnosis				LP	IOP	Pre Op E/D								Results			
			LAT	DOV	Duration in months	SP	IOS	Tr.	MH				LAT	GRADE						Pre Op drug started one day before Sx				Mydriatic E/D one hr before Sx			PD in mm			
										VA	AS	PS	RE/LE	NS	PSC	CC				24 hrs	18 hrs	12 hrs	6 hrs	60 mins	45 mins	30 mins	1	2	3	4
1	65	F	LE	+	12	-	+	-	HTN	HMCF	N	HR	LE	M	-	-	P	10.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	7.5	7
2	54	M	LE	+	1	-	-	-	NIL	6/36	N	N	LE	II	+	-	P	11.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
3	68	F	RE	+	12	-	-	-	NIL	PL+	N	N	RE	M	-	-	P	7.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6	5.5	5.5	5
4	65	M	LE	+	12	-	-	-	NIL	CF 2 MT	N	N	LE	III	+	+	P	7.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	5	5	5
5	80	M	RE	+	24	-	-	-	NIL	6/60	N	N	RE	III	+	+	P	7.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	5.5	5.5	5.5
6	61	F	LE	+	12	-	+	-	NIL	CF1MT	N	N	LE	IV	+	-	P	11.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8.5	7	7	7
7	65	M	RE	+	3	-	+	-	NIL	6/18	N	N	RE	II	+	+	P	10.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	6.5	7	7.5
8	59	M	RE	+	1	-	-	-	HTN	CF 3 MT	N	N	RE	III	+	-	P	14.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	6.5
9	60	F	LE	+	6	-	-	-	NIL	6/60	N	N	LE	II	+	-	P	11.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	7.5	7.5	7.5
10	75	F	LE	+	2	-	-	-	NIL	6/60	N	N	LE	IV	+	-	P	12.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6.5	5.5	5.5
11	55	M	LE	+	3	-	-	-	NIL	HMCF	N	N	LE	M	-	-	P	7.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
12	58	F	LE	+	6	+	+	-	HTN	CF 1 MT	N	N	LE	B	-	-	P	10.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	6	5.5	5.5
13	70	M	RE	+	6	-	-	-	NIL	PL +	N	N	RE	M	-	-	P	7.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	6.5
14	65	M	LE	+	6	-	-	-	NIL	6/36	N	N	LE	III	+	-	P	15.3	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8.5	7	7	7
15	57	M	LE	+	6	-	+	-	NIL	CF 1 MT	N	N	LE	III	+	-	P	14	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6.5	6.5	7
16	50	F	LE	+	12	-	+	-	NIL	CF 1 MT	N	N	LE	III	+	-	P	14	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	6	5	5
17	80	M	LE	+	12	-	-	-	NIL	CF 3 MT	N	N	LE	III	+	-	P	13	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	5	5	5
18	58	F	LE	+	6	-	-	-	NIL	6/24	N	N	LE	II	+	-	P	16.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7.5	7.5
19	65	M	RE	+	4	-	-	-	NIL	6/24	N	N	RE	III	+	+	P	11.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6.5	6	7
20	57	F	RE	+	4	-	-	-	NIL	HMCF	N	N	RE	M	-	-	P	13.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	8	7.5	7.5
21	55	F	RE	+	3	-	-	-	NIL	CF 1 MT	N	N	RE	II	+	+	P	16.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6	5.5	5.5
22	70	M	RE	+	2	-	-	-	NIL	6/36	N	N	RE	I	+	-	P	8.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
23	65	F	RE	+	4	-	-	-	NIL	CF 3 MT	N	N	RE	I	+	-	P	12.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	6.5	6.5
24	55	F	RE	+	4	+	-	-	NIL	6/60	N	N	RE	II	+	+	P	16.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7	6	5.5
25	55	F	RE	+	24	+	+	-	NIL	6/60	N	N	RE	II	+	+	P	13.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7.5	7.5	7.5
26	72	M	RE	+	3	-	-	-	NIL	CF 3 MT	N	N	RE	II	+	-	P	11.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	7	7
27	80	F	RE	+	12	-	+	-	HTN	PL +	GIINP	N	RE	M	-	-	P	7.3	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6	6
28	79	M	RE	+	3	-	-	-	NIL	6/36	N	N	RE	II	+	+	P	8.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	8	8	8
29	82	F	LE	+	6	+	-	-	HTN	6/60	N	N	LE	II	+	+	P	21	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	6	6
30	80	M	RE	+	3	-	-	-	NIL	6/24	N	N	RE	II	+	+	P	16.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	8	8	8.5
31	62	F	RE	+	7	-	-	-	HTN	PL +	N	N	RE	III	+	-	P	11.4	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	6.5
32	55	M	LE	+	12	+	-	-	NIL	CF 3 MT	N	N	LE	II	+	+	P	14	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	7	7
33	74	M	LE	+	12	-	-	-	NIL	CF 3 MT	N	N	LE	II	+	+	P	7.8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6	6
34	88	M	RE	+	3	-	+	-	NIL	6/18	N	N	RE	I	+	-	P	14.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	6	6	5.5
35	60	F	RE	+	12	-	-	-	NIL	CF 2 MTS	N	N	RE	III	+	-	p	10.6	DEXAMETHASONE	y	y	y	y	y	y	y	7.5	6.5	6.5	6.5
36	63	M	RE	+	18	-	+	-	NIL	6/12	N	N	RE	II	+	-	P	8.8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7.5	7	7
37	77	M	LE	+	12	-	-	-	NIL	CF 2 MTS	N	N	LE	III	+	+	P	10.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
38	80	M	RE	+	12	-	-	-	NIL	6/24	N	N	RE	III	-	-	P	7.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	6	6	6
39	65	F	LE	+	6	-	-	-	NIL	CF 2 MTS	N	N	LE	II	+	-	P	12.3	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	5.5	5.5
40	59	M	RE	+	3	-	-	-	NIL	6/12	N	N	RE	I	+	+	P	11.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	8.5	8.5	7.5
41	54	M	LE	+	3	+	-	-	NIL	CF 3 MT	N	N	LE	II	-	-	P	10.8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	5.5	5	5
42	59	M	RE	+	7	-	-	-	NIL	6/18	GIINP	N	RE	II	+	-	P	15.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	6.5
43	68	M	RE	+	3	+	+	-	HTN	6/60	N	N	RE	III	+	+	P	11.4	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	6.5	6.5
44	75	M	RE	+	12	+	+	-	NIL	HMCF	N	N	RE	B	-	-	P	7.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	6.5
45	80	M	LE	+	2	+	+	-	NIL	CF 2 MT	N	N	LE	III	+	-	P	11.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	7
46	65	F	RE	+	5	+	+	-	NIL	6/18	N	N	RE	II	+	-	P	12.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6.5	6	5.5
47	65	F	RE	+	6	-	-	-	NIL	CF 1 MT	GINP	N	RE	III	+	-	P	13.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	6.5	6	6	6
48	67	F	RE	+	6	+	+	-	NIL	6/60	N	N	RE	IV	+	-	P	10.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
49	74	M	LE	+	6	+	+	-	NIL	6/60	CO	N	LE	II	+	+	P	15.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	5	5	5
50	50	M	RE	+	12	+	+	-	NIL	6/60	N	N	RE	I	+	-	P	12.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	8.5	8.5	8.5

55	70	F	LE	+	25	-	-	-	NIL	CF1MT	N	N	LE	III	-	-	P	10.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	6.5	6	5.5	5.5
56	53	F	LE	+	36	-	+	-	HTN	CF1MT	N	N	RE	III	+	-	P	15.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
57	65	M	LE	+	12	-	-	-	NIL	CF3MT	N	N	LE	II	+	-	P	15.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6	6
58	59	M	LE	+	12	+	+	-	HTN	6/36	N	N	LE	III	+	+	P	17.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6	5.5	5.5
59	51	F	RE	+	12	-	+	-	NIL	6/60	N	N	RE	III	+	+	P	12	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	7
60	65	F	LE	+	8	-	-	-	NIL	CF1MT	N	N	LE	III	+	+	P	18.8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6.5	6.5	6.5
61	66	M	LE	+	12	-	-	-	NIL	CF1MT	N	N	LE	II	+	+	P	18.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6	6
62	65	F	LE	+	12	-	+	-	HTN	6/60	N	N	LE	II	+	+	P	11.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	7.5	7.5	7.5
63	60	F	RE	+	24	-	-	-	HTN	6/36	N	N	RE	II	+	+	P	9.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	6.5	6.5	6.5
64	52	F	RE	+	10	+	-	-	NIL	6/18	N	N	RE	I	+	-	P	16.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	7.5	7	7.5
65	66	F	LE	+	1	+	-	-	HTN	6/60	N	HR	LE	II	+	+	P	19.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	7	7
66	66	M	RE	+	6	-	+	-	ASTHMA	CF I MT	GIINP	N	RE	IV	+	-	P	9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	6.5	6.5
67	53	M	RE	+	2	-	+	-	NIL	HMCF	GINP	N	RE	M	-	-	P	14.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
68	69	M	RE	+	2	+	+	-	NIL	6/24	N	N	RE	III	+	+	P	9.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	9	8	7.5	7.5
69	45	F	RE	+	24	+	-	-	NIL	PL+	GINP	N	RE	M	-	-	P	8.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	6.5	5.5	5.5	5
70	70	F	RE	+	12	-	-	-	NIL	6/24	N	N	RE	I	+	+	P	12.3	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7.5	7.5	8
71	53	M	RE	+	2	-	-	-	NIL	HMCF	N	N	RE	IV	+	+	P	17.3	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	6.5	6.5	6.5
72	59	M	LE	+	12	-	-	-	NIL	CF3MT	GINP	N	LE	II	+	+	P	15.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	8	8	7.5
73	63	M	LE	+	36	-	-	-	NIL	6/60	N	N	LE	II	+	+	P	15.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8.5	8	8	8.5
74	66	M	RE	+	3	+	-	-	NIL	CF2MT	PXF	N	RE	III	+	+	P	11.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	6	6	6
75	70	F	LE	+	5	+	-	-	NIL	CF 2 MT	N	N	LE	III	+	+	P	10.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	7.5
76	62	F	RE	+	24	+	+	-	NIL	6/60	N	N	RE	B	-	-	P	8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	6.5	6	6.5
77	55	F	LE	+	12	+	-	-	NIL	6/18	N	N	LE	II	+	+	P	16.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
78	81	M	RE	+	12	-	+	-	NIL	CF 2MT	N	N	RE	II	+	+	P	13.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6	5.5	6
79	70	F	RE	+	12	-	-	-	NIL	6/36	N	N	RE	B	-	-	P	10.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	5.5	5.5	5.5
80	72	F	LE	+	4	-	+	-	NIL	PL+	N	N	LE	M	-	-	P	16.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6.5	6.5	6.5
81	69	F	LE	+	4	-	-	-	HTN	CF1MT	N	N	LE	B	-	-	P	18.7	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	7	6	6
82	52	M	LE	+	12	+	-	-	NIL	6/24	N	N	LE	III	+	+	P	18.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6	6	5.5
83	70	F	LE	+	12	-	-	-	NIL	6/24	N	N	LE	I	+	-	P	10.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	6.5
84	75	M	RE	+	24	-	-	-	HTN	CF2MT	N	N	RE	II	+	+	P	8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	7	7
85	58	F	RE	+	12	+	+	-	NIL	6/36	N	N	RE	II	+	+	P	14.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6	6
86	62	M	LE	+	6	-	-	-	NIL	PL+	N	N	LE	HM	-	-	P	17.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5
87	57	M	LE	+	12	+	+	-	NIL	6/24	GIINP	N	LE	II	+	+	P	13.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6	5	4.5
88	75	M	LE	+	12	+	-	-	NIL	CF2MT	N	N	LE	III	+	-	P	12.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	6.5	6.5	6.5
89	61	M	RE	+	3	+	-	-	NIL	6/60	CO	N	RE	III	+	+	P	8.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	6.5
90	65	F	LE	+	3	-	-	-	NIL	HMCF	N	N	LE	M	-	-	P	18	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7	6.5	6
91	60	F	RE	+	3	-	-	-	HTN	6/60	N	N	RE	II	+	-	P	16.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	6.5	6.5
92	75	M	LE	+	3	+	+	-	NIL	CF 2MT	N	N	LE	III	-	-	P	11.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	7	8
93	42	F	RE	+	4	+	-	-	NIL	6/24	N	N	RE	II	+	+	P	18.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
94	65	M	RE	+	6	-	-	-	HTN	6/24	N	N	RE	II	+	-	P	8.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	7	7	7
95	58	F	LE	+	12	-	-	-	HTN	6/36	N	N	LE	II	+	+	P	15.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	5.5	4.5	5
96	81	M	LE	+	6	-	-	-	HTN	CF 1 MT	N	N	LE	III	+	+	P	8.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6.5	6.5	7.5
97	68	M	RE	+	12	-	-	-	NIL	6/36	N	N	RE	B	-	-	P	9.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6	6.5	7
98	54	F	RE	+	3	+	+	-	NIL	CF 1 MT	N	N	RE	M	-	-	P	18.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	5.5	6	6.5
99	55	M	LE	+	36	-	-	-	NIL	HMCF	N	N	LE	M	-	-	P	12.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	7.5
100	68	F	LE	+	5	-	+	-	HTN	CF1MT	N	N	LE	B	-	-	P	12.8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	5.5	5.5	5.5
101	68	F	RE	+	12	+	-	-	NIL	PL+	N	N	RE	M	-	-	P	11.4	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6	7
102	52	M	LE	+	10	-	-	-	NIL	HMCF	N	N	LE	I	+	-	P	18.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	6.5	6.5	7
103	54	F	RE	+	6	-	+	-	HTN	HMCF	N	N	RE	M	-	-	P	18.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	7
104	61	M	RE	+	12	-	-	-	HTN	CFCF	N	N	RE	M	-	-	P	11.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7.5	6.5	6.5
105	67	M	LE	+	3	+	+	-	NIL	6/60	N	N	LE	III	+	+	P	19.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	5.5
106	60	M	RE	+	3	-	-	-	NIL	6/12	N	N	RE	I	+	-	P	10.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	7	6.5
107	80	M	LE	+	6	-	+	-	NIL	CFCF	GINP	N	LE	M	-	-	P	8.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	5.5	5.5	5.5
108	70	F	LE	+	12	+	+	-	HTN	6/36	N	HR	LE	III	+	-	P	18.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	5	5.5	6
109	58	F	LE	+	4	+	-	-	NIL	6/24	N	N	LE	II	+	-	P	17.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7.5	7.5
110	65	M	RE	+	4	+	-	-	NIL	CF1MT	N	N	RE	II	+	-	P	12.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	7.5
111	63	F	RE	+	4	-	-	-	NIL	CF2MT	N	N	RE	I	+	-	P	9.5	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	7.5	7.5
112	65	M	LE	+	6	+	-	-	NIL	CF 1 MT	N	N	LE	II	+	+	P	13.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	7.5	7
113	60	F	LE	+	12	-	-	-	NIL	6/24	N	N	LE	II	+	-	P	8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	7	7	7
114	60	M	LE	+	12	+	-	-	NIL	6/24	N	N	LE	III	+	+	P	10.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	7.5
115	67	M	LE	+	3	+	-	-	NIL	CF3MT	GIINP	N	LE	II	+	+	P	17	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	6.5
116	71	F	LE	+	4	-	+	-	HTN	PL +	N	N	LE	M	-	-	P	18.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7.5	7.5	8
117	70	F	RE	+	2	+	-	-	NIL	CF 1 MT	N	N	RE	M	-	-	P	11.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5
118	71	M	LE	+	5	-	+	-	NIL	CF 2 MT	GIINP	N	LE	II	+	+	P	17.8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	6.5
119	65	F	RE	+	12	-	-	-	NIL	HMCF	N	N	RE	M	-	-	P	11.3	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5
120	65	F	LE	+	3	-	-	-	NIL	6/36	N	N	LE	B	-	-	P	11.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5
121	68	M	RE	+	12	+	-	-	HTN	6/36	N	N	RE	II	+	+	P	15.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
122	71	F	RE	+	6	+	+	-	NIL	6/60	N	N	RE	II	+	+	P	14.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7	5.5	5.5	5.5
123	68	M	RE	+	3	-	-	-	HTN	6/36	N	N	RE	I	+	+	P	6.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5

128	59	F	LE	+	2	+	+	-	NIL	HMCF	N	N	LE	M	-	-	P	15.7	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	6.5	7	7.5
129	60	F	LE	+	2	-	-	-	NIL	6/36	N	N	LE	II	+	-	P	11.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8.5	6.5	7	7.5
130	78	M	RE	+	12	-	-	-	NIL	CF1MT	N	N	RE	I	+	+	P	12.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6.5	7	7
131	65	M	LE	+	2	+	-	-	NIL	CF2MT	N	N	LE	IV	+	+	P	13	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	5	4.5	5
132	58	F	LE	+	6	+	-	-	NIL	CF2MT	N	N	LE	II	+	-	P	18	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6.5	7
133	55	F	RE	+	12	-	-	-	NIL	CF1MT	N	N	RE	I	+	+	P	18.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7.5	8	8.5
134	60	F	LE	+	6	+	-	-	NIL	6/36	N	N	LE	III	+	-	P	8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	7	7	7.5
135	60	F	RE	+	7	+	-	-	NIL	CFCF	N	N	RE	M	-	-	P	18.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7.5	7	7
136	50	F	RE	+	12	-	-	-	HTN	CF1MT	N	N	RE	II	+	-	P	12.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	6.5	6	6	5.5
137	75	F	LE	+	6	-	+	-	NIL	PL+	N	N	LE	HM	-	-	P	12.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	6.5
138	57	F	RE	+	12	-	-	-	NIL	6/24	N	N	RE	III	+	+	P	16.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	6.5	6	6
139	63	F	RE	+	3	+	-	-	NIL	6/24	N	N	RE	II	+	-	P	12.3	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	5.5	5	5
140	66	M	RE	+	24	-	-	-	NIL	6/60	N	N	RE	II	+	-	P	10.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	6.5	5.5
141	69	M	RE	+	6	+	-	-	HTN	6/36	N	N	RE	III	+	-	P	14.4	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	6.5	6.5
142	66	F	RE	+	6	+	-	-	NIL	CF1MT	N	HR	RE	III	+	-	P	13.7	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	5.5	5.5
143	65	F	RE	+	24	-	-	-	HTN	PL+	GIINP	N	RE	M	-	-	P	11.4	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	7	7
144	65	M	RE	+	2	+	-	-	NIL	6/36	N	N	RE	II	+	-	P	16	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	6	6
145	67	M	LE	+	36	+	-	-	HTN	6/24	N	N	RE	I	+	+	P	6.7	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	7.5	7.5	7
146	63	M	RE	+	6	-	-	-	NIL	CF2MT	N	N	RE	II	+	+	P	12.9	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	6.5	6	5.5	5.5
147	60	F	RE	+	3	-	-	-	NIL	CF2MT	N	N	RE	IV	+	-	P	9.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	6.5	6.5	6
148	65	M	RE	+	6	-	-	-	NIL	CF1MT	N	N	RE	III	+	-	P	14.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6	6
149	67	F	LE	+	12	+	+	-	NIL	CF3MT	N	N	LE	III	+	-	P	9.3	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	6.5	6
150	52	F	LE	+	1	-	+	-	NIL	PL+	N	N	LE	M	-	-	P	9.1	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	6
151	62	F	RE	+	6	+	+	-	NIL	6/18	N	N	RE	II	+	+	P	12.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6	6
152	60	F	LE	+	12	-	+	-	NIL	CF1MT	N	N	LE	II	+	-	P	10.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6	6
153	75	M	LE	+	24	-	+	-	NIL	PL+	N	N	LE	M	-	-	P	15.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7.5	7	7
154	60	F	RE	+	4	-	-	-	NIL	CF3MT	N	N	RE	I	+	+	P	15.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7.5	7	6.5
155	63	F	RE	+	12	+	-	-	HTN	6/12	N	N	RE	I	+	+	P	14.4	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	6.5	6.5	6
156	68	M	LE	+	4	+	+	-	NIL	CF1MT	GINP	N	LE	III	+	+	P	10.1	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5
157	65	F	RE	+	12	-	+	-	HTN	6/36	N	N	RE	II	+	-	P	10.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	6.5
158	70	F	LE	+	2	-	-	-	NIL	PL+	N	N	LE	M	-	-	P	13.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	6.5	6	6	6
159	60	M	LE	+	2	+	-	-	NIL	PL+	N	N	LE	M	-	-	P	10.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	5.5	5.5	5.5
160	65	M	LE	+	12	-	-	-	NIL	CF1MT	N	N	LE	II	+	+	P	20.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6.5	6.5	6
161	58	F	RE	+	12	-	-	-	NIL	PL+	N	N	RE	M	-	-	P	13.9	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	6.5	6	6	6
162	60	M	RE	+	4	-	+	-	NIL	PL+	N	N	RE	HM	-	-	P	15.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	6.5	6.5	6.5	6
163	60	F	RE	+	6	-	-	-	NIL	6/36	N	N	RE	II	+	-	P	16.5	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6.5	6.5
164	60	M	RE	+	12	+	-	-	NIL	CF1MT	N	N	RE	III	+	+	P	9.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8.5	6	5.5	5.5
165	66	F	RE	+	6	+	+	-	NIL	6/24	N	N	RE	I	+	+	P	13.8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5
166	68	F	LE	+	12	+	+	-	NIL	CF3MT	N	N	LE	II	+	+	P	12.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7.5
167	75	F	LE	+	2	-	-	-	NIL	CF3MT	N	N	LE	III	+	+	P	10.2	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	7	5
168	86	M	LE	+	2	-	-	-	NIL	CF1MT	N	N	LE	B	-	-	P	7.8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
169	47	M	RE	+	4	-	-	-	NIL	CF1MT	N	N	RE	III	+	-	P	9.4	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	9	9	9	9
170	50	F	RE	+	24	+	-	-	NIL	CF3MT	N	N	RE	II	+	-	P	8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	6.5
171	72	M	RE	+	24	-	+	-	NIL	6/24	N	N	RE	II	+	+	P	8.4	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7	6	6	5
172	65	F	LE	+	4	+	-	-	NIL	6/36	N	N	LE	II	+	+	P	12.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	6.5	6
173	68	F	RE	+	6	+	+	-	HTN	6/60	N	N	RE	II	+	+	P	14.7	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	8	7	7
174	50	M	LE	+	8	+	-	-	NIL	6/60	N	N	LE	II	+	-	P	13.8	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
175	50	F	RE	+	36	+	+	-	NIL	CF2MT	N	N	RE	III	+	+	P	14.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8.5	7	7	7
176	70	M	RE	+	24	+	-	-	NIL	6/60	N	N	RE	III	+	-	P	8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	6	6.5
177	73	M	RE	+	6	+	+	-	NIL	6/60	N	N	RE	M	-	-	P	14.6	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	8	6.5	5.5	5
178	65	F	LE	+	6	+	+	-	HTN	CFCF	N	N	LE	M	-	-	P	8.8	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	6.5	6.5	6.5
179	66	M	RE	+	5	+	-	-	NIL	CF2MT	N	N	RE	III	+	-	P	11.2	KETOROLAC	Y	Y	Y	Y	Y	Y	Y	7.5	7	7	7
180	60	F	RE	+	9	+	-	-	HTN	CF1MT	N	N	RE	III	+	+	P	16.6	DEXAMETHASONE	Y	Y	Y	Y	Y	Y	Y	8	7	6.5	6.5