PREVALENCE OF DRY EYES FOLLOWING PHACOEMULSIFICATION SURGERY AND ITS RELATION TO INTRAOPERATIVE RISK FACTOR - A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI

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ENDORSEMENT BY HEAD OF THE DEPARTMENT, PRINCIPAL/ HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"PREVALENCE OF DRY EYES FOLLOWING PHACOEMULSIFICATION SURGERY AND ITS RELATION TO INTRAOPERATIVE RISK FACTOR - A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR.PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"** is a bonafide research work done by REG. NO. BK0116002.

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LIST OF ABBREVIATIONS USED

ADDE	Aqueous tear deficient dry eye
BCVA	Best corrected visual acuity
DED	Dry Eye Disease
ECCE	Extra capsular cataract extraction
EDE	Evaporative dry eye
ICCE	Intra capsular cataract extraction
IOL	Intraocular Lens
LE	Left Eye
LFU	Lacrimal Functional Unit
LG	Lissamine green
PMMA	Polymethylmethacrylate
RE	Right Eye
SS	Sjogren syndrome
SSDE	Sjogren syndrome dry eye
ST-I	Schirmers test-I
TBUT	Tear film breakup time
TFLL	Tear film lipid layer
TMH	Tear meniscus height
UCVA	Uncorrected visual acuity

ABSTRACT

Background and objectives

Dry eye is no longer a trivial problem ignored by the eye care professionals. Dry eye is a multi-factorial disease that affects the quality and quantity of tears that alter the ocular surface. After cataract surgery many patients complain of foreign body sensation, irritation, redness, blurring of vision which are considered as unwanted effects of surgery. For patients who experience dry eye after phacoemulsification surgery, vision and quality of life can significantly deteriorate. Increased ocular discomfort after cataract surgery may be a result of failure to diagnose dry eye before or after surgery and/or subsequent inadequate treatment after cataract surgery.

The objectives of our study are

1. To study the prevalence of dry eye in patients undergoing phacoemulsification surgery at KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi.
2. To analyze dry eye in relation to microscopic light exposure time.

Methodology

The present study was conducted in the Department of Ophthalmology, KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi to study the prevalence of dry eye in patients undergoing phacoemulsification surgery and to analyze dry eye in relation to microscopic light exposure time during the period of 1stJanuary 2017 to 31st December 2017. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belagavi.

Results

In our study, out of 100 cases who underwent phacoemulsification, studied for dry eye evaluation, 42% were males and 58% were females, with a M:F ratio of 1:1.4. 68% of the patients were in the age group of 45-59 years and 32% were 60 years and above.

In our study, on analysis of Schirmers test-1 at 2 months post-operatively, in the age group of 45-59 years, dry eyes persisted in 13 cases (19.12%) out of the total 68 cases in that group and in the age group 60 years and above, 8 cases (25%) had dry eyes out of the total 32 cases in that group and also on analysis of Tear film breakup time at 2 months post-operatively, in the age group of 45-59 years, dry eyes persisted in 7 cases (21.88%) out of the total 32 cases (32%) in that group and in the age group 60 years and above, 28 cases (41.18%) had dry eyes out of the total 68 cases(68%) in that group.

On analysis of Schirmers test-1 , 32% of the cases had dry eye at 1 week post phacoemulsification surgery, 28% at 1 month and 21% at 2 months. Pre-operatively, the mean of the schirmers test values was 19.55 $\pm$ 8.20 which reduced to 15.70 ± 9.44 at one week post-op which eventually stabilized to 16.71 ± 8.05 at 2 months follow-up.

On analysis of Tear film breakup time, 43% of the cases had dry eye at 1 week post phacoemulsification surgery, 47% at 1 month and 35% at 2 months. Pre-operatively, the mean of the TBUT test values was 15.14 ± 4.66 which reduced to 10.76 ± 5.26 at one week post-op, which stabilized till one month follow-up with a

mean of 10.97 ± 5.48 and eventually increasing slowly at two months follow-up when the mean was 12.28 ± 5.05.

In our study, on analysis of Lissamine green staining, 35% of the cases had dry eye at 1 week post phacoemulsification surgery, 30% at 1 month and 23% at 2 months.

In this study on analysis of association between Schirmers test-1 and lissamine green staining at 2 months postoperatively, out of the 65 cases who showed grade-0 lissamine green staining, 6 cases(9.23%) had decreased Schirmers test values, out of the 11 cases who showed grade-I lissamine green staining, 6 cases(54.55%) had decreased Schirmers test values, out of the 8 cases who showed grade-II lissamine green staining, 7 cases(87.50%) had decreased Schirmers test values, and the 2 cases who showed grade-III lissamine green staining, showed decreased Schirmers test values at 2 months.

In this study on analysis of association between Tear film breakup time and lissamine green staining at 2 months postoperatively, out of the 77 cases who showed grade-0 lissamine green staining, 18 cases(23.28%) had decreased TBUT test values, out of the 14 cases who showed grade-I lissamine green staining, 9 cases(64.29%) had decreased TBUT test values, out of the 7 cases who showed grade-II lissamine green staining, 6 cases(85.72%) had decreased TBUT test values, and the 2 cases who showed grade-III lissamine green staining, showed decreased TBUT test values at 2 months.

In our study, the mean of the MLET values was 22.72+/-3.49 with a minimum value of 13.34 and maximum of 34.90. This study shows a negative correlation between MLET and the dry eye test values which is highly significant($p < 0.001$).

Conslusions and Interpretations

Thus we conclude from this study that Phacoemulsification surgery is indeed capable of inducing dry eye. The values were worse in the early post-operative period which gradually increased over 2 months. Therefore, prior to cataract surgery, patients must be informed about the possible increase in dry eye, however the condition might remain the same in some cases. Intra-operative exposure to microscopic light should be minimized by appropriate use of filters and shortening the exposure time.

Key words

Phacoemulsification, Dry eye, Schirmers Test-I, Tear film breakup time, Lissamine Green staining, Microscopic Light Exposure Time.

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INTRODUCTION

Dry eye is a frequently encountered ocular problem in our tropical climate[1]. It occurs when there is inadequate tear volume or function resulting in an unstable tear film and ocular surface disease[2]. Dry eye disease(DED) produces discomfort and reduced vision due to tear film instability. It is per se not a disease entity, but a symptom complex occurring as a sequelae to deficiency or abnormalities of tear film, exposing the corneal and conjunctival epithelium to evaporation. It is one of the most important factors influencing the quality of life in elderly population.

Dry eye syndrome is a multifactorial disease characterized by dryness of the ocular surface due to tear deficiency and over evaporation[3,4]. There are many causes and factors leading to dry eye, including aging, female gender, connective tissue diseases, diabetes mellitus, systemic hypertension, contact lens usage, drugs like anticholinergics, antidepressants, oral contraceptives and topical eye drops containing preservatives and ocular diseases like blepharitis, chronic conjunctivitis, meibomitis and pterygium[5-7].

Dry eye is the most frequent disorder in ophthalmology practice. The prevalence of which varies from 4% to 57%, thereby showing disparity worldwide[8-11].

The word cataract, which means both opacity of the lens and a torrent of water, comes from the Greek word kataraktes meaning the fall of water. Cataract is opacity of the natural crystalline lens Cataract is currently the main cause of avoidable blindness especially in the developing world accounting for about three quarters of blindness. Cataract surgery has undergone significant developments over the past two decades. In developed world phacoemulsification (phaco) is the primary method of performing cataract surgery. However, in many developing countries involving the

majority of cataract blindness in the world today, phacoemulsification is not viable due to density of cataract involved and high cost of the equipment[12,2].

Phacoemulsification has become the preferred method of cataract extraction over the last 15 years. The smaller incision of phacoemulsification is associated with little induced postoperative astigmatism and early stabilization of refraction[2]. In the developed world phacoemulsification is the primary method of performing cataract surgery.

There are numerous factors that might affect the ocular surface environment after cataract surgery. Topical anaesthesia and eye drops with preservatives like benzalkonium chloride are known to have effects on the corneal epithelium. Exposure to light from the operating microscope may also be associated with postoperative dry eye. The normal organization of the corneal innervation is disrupted by most corneal surgical procedures resulting in pathologic changes of the cornea and discomfort to the patient. Dry eye-associated symptoms, such as foreign body sensation and fatigue, frequently occur after cataract surgery. Affected patients may experience red or watery eyes along with constant foreign body sensation. Several published studies have documented the aggravation of dry eye symptoms and signs after LASER in situ keratomileusis and penetrating keratoplasty. Some studies have even reported aggravation of dry eye symptoms and signs after cataract surgery[13]. In one of the studies, approximately 85% of patients experienced DED-like symptoms, particularly foreign body sensation, on day 1 and the percentage decreased to 25% by day 30, but 15% of patients continued to complain of dry eye symptoms at day 90[14]. The dry eye test value (fluorescein staining) was positive in 12% of patients preoperatively and in 90% on day 1, 30% on day 30, and 8% on day 90 postoperatively and Lissamine green staining was positive in 5% of patients preoperatively and in 40% on day 1, 4% on day 30,

and about 1% on day 90 postoperatively[14]. In one of the studies, the results suggest that the microscopic light exposure might be one of the possible contributory factors for dry eye after ophthalmic surgery. The healing process was slowed down by the cell damage caused by intensity of light exposure from a clinical surgical microscope[15].

Cataract surgery has been identified as one of the risk factors for the development of DED by disruption of normal ocular homeostasis by decreasing corneal sensation and changing the contour of the ocular surface as a result of the inflammation caused by its surgical trauma[16,17].

Two kinds of dry eye were clinically observed after cataract surgery, early dry eye and chronic dry eye. Most cases of early dry eye, who usually had the normal lacrimal secretion before surgery, were reversible and involved in some of factors associated with surgery and post-surgery medication. But most cases of chronic dry eye, who have abnormal lacrimal secretion or "borderline state" of lacrimal secretion test before surgery, may suffer from the ocular surface diseases related to irreversible DED. It is significantly important to maintain the ocular surface stability and recovery of visual acuity after cataract surgery to do early diagnosis and promptly manage the dry eye syndrome. So, it is necessary that each patient undergoing cataract surgery should have dry eye evaluation to know for any symptoms and signs of dry eye and if found should be treated before undertaking the patient for the surgery, so as to minimize the symptoms and signs of dry eye and to have a good outcome.

Thus the present study was undertaken to estimate the prevalence of dry eye rates after phacoemulsification surgery and the associated risk factor involved. Hence it is necessary that each patient undergoing cataract surgery should have dry eye evaluation to detect signs of dry eye disease and if found should be treated appropriately.

AIMS AND OBJECTIVES

1) To study the prevalence of dry eye in patients undergoing phacoemulsification surgery at KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

2) To analyze dry eye in relation to microscopic light exposure time.

REVIEW OF LITERATURE

History of Cataract

The word cataract which means both opacity of the lens and a torrent of water, comes from the Greek word "kataraktes" meaning the fall of water. The oldest documented case of cataract throughout history was from the Egyptian museum in Cairo from a small famous statue from the 5th dynasty, discovered in 1860. The Latins called cataract, "suffisio" an extravasation and coagulation of humors behind the iris and the Arabas called it white water.

The old Egyptian name for the lens is not yet known and the medical literature does not let us conclude that old Egyptians were able to diagnose cataracts. The only possible reference to cataract is the disease mentioned in the Ebers Papyrus about 1525 B.C. Ebbel translated the disease as cataract. However, other distinguished linguistics such as Hirschberg and Deines interpreted it as a discharge or accumulation of water in the eyes. According to Ebers Papyrus, the old Egyptians tried to treat cases of cataract by eye ointments and magic spells. It is hardly believable that such remedies had any effect on the cataract, since the extraction of the lens is the only effective measure[18].

Without doubt, one of the oldest surgical procedures for cataract was “couching” which was lens depression. An ancient Indian surgeon, Maharshi Sushruta first described the .procedure in "Sushruta Samhita" in 800 B.C. This technique involved using a sharp instrument such as a curved needle to push the cloudy lens into the rear of the eye and out of the field of vision. The eye would later be soaked with warm clarified butter and then bandaged. The first references to cataract and its treatment in the West are found in 29 B.C. according to the work in Latin, which also

describes the couching operation. During the 18th and 19th centuries this technique was also popular in a number of the European countries. Throughout the Middle Ages Couching technique was continued to be used and is still used in some parts of Africa. Serious complications were frequent as it was an ineffective and dangerous method of cataract therapy and there was no method for optical rehabilitation. Thus, patients suffered from optical aphakia for the rest of their lives, and often resulted in patients remaining blind or with only partially restored vision[18].

During the 2nd century A.C, cataract extraction surgery was described which replaced couching technique. The lens could be removed by suction through a hollow instrument such as a bronze oral suction instrument by an assistant with an extraordinary lung capacity.

The first modern European physician to successfully extract cataracts from the eye was a French ophthalmologist Jacques Daviel. On April 8th, 1747, he introduced the first extracapsular cataract extraction (ECCE) with an intact lens capsule wherein he made an incision on the inferior part of the cornea and place a needle posterior to the lens and delivered it with some loss of vitreous. It was the first significant advance in cataract surgery since couching was invented. This led to the subsequent developments in the techniques of cataract surgery[17]. This technique was subsequently refined by people like Albrecht von Graefe[19].

In 1752, Intracapsular cataract extraction (ICCE) was introduced where the lens together with the capsule was taken out by George de la Faye and in 1753, by Samuel Sharp. Most frequently without a breach of the capsule, 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. Following this, the lens was inverted by an action known as tumbling. During the 19th and 20th

centuries with the utilization of other instruments such as curette, spoon and strabismus hook, the procedure of ICCE was enhanced. In 1957, Joaquin Barraquer launched the zonulysin which is an alpha chymotrypsin, a digestive proteolytic enzyme of bovine origin, which could be introduced into the posterior chamber prior to either of the methods resulting in zonulolysis, thereby simplifying removal of the lens. In 1961, cryoextraction of the lens was launched by Tadeusz Krwawicz. ICCE was the most popular method but due to a higher occurrence of cystoid macular edema, retinal detachment, vitreous loss, astigmatism and use of anterior chamber intra-ocular 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[19].

Sir Harold Ridley thought to use an artificial lens after observing the eye's tolerance of PMMA(poly methyl methacrylate) following eye injuries in Royal Air Force pilots. During the battle of Britain in World War II, when the pilots' plastic canopies were struck with bullets, they shattered, leaving small pieces of PMMA in their eyes. Ridley observed that the pilots' eyes were compatible with them and did not reject the inert PMMA substance. It resulted in little response to the plastic material, only if it did not move about inside the eye This inspired him to use this material in early intraocular lens implantations to correct cases of cataracts. At St. Thomas Hospital in London, Sir Harold Ridley successfully implanted the first Intraocular lens (IOL) on 29 November 1949. Subsequently, he began placing the disc form PMMA lenses in a biconvex design posterior to the iris following ECCE[20]. It was abandoned again due to the various undesirable effects, which led to the developments of IOL.

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. Nucleus removal which was performed primarily in the anterior chamber, is now performed in the posterior chamber, decreasing damage to the corneal endothelium[18].

In 1967, Charles D. Kelman, an ophthalmologist who was a pioneer of cataract surgery, introduced phacoemulsification[20]. He was inspired by his dentist's ultrasonic probe. It was one of the most remarkable change made in the current times and has now become the method of choice for all surgeons due to a smaller size of the incision. This technique utilizes an intraocular probe which is introduced and ultrasound waves are generated that emulsify the nucleus of the crystalline lens into smaller pieces, these minute fragments are then aspirated. Thus a combined ultrasonographic, aspiration and irrigation hand piece allows the expulsion of any lens through a small incision. This new method of surgery decreased the need for an extended hospital stay and made the surgery less painful[18].

Tear film

The basic representation of the interface between the eye and the external world is the ocular surface. The tear film consists of different components which are well balanced and is a very complex and dynamic structure, the intricacies of which are not yet completely understood. Over the last decade there has been tremendous growth in the understanding of tear film biology. Earlier, the tear film was viewed as a simple structure which was composed of segregated layers of mucus, water and electrolytes, and lipid. Now it is known that the tear film accomplishes its multiple functions by interaction of its different components to create a hydrated gel. Tear film

maintains the ocular surface homeostasis by its dynamicity and a constant state of flux responding to environmental conditions.The healthy ocular surface comprises of a functional unit that utilizes a variety of structures, all of which remain inter-related in anatomy, composition, and physiological function. These structures include the tear film, corneal and conjunctival epithelium, meibomian and lacrimal glands, and eyelids. A normally functioning tear film is required to maintain clarity of vision and ocular health. The tear film maintains the health of corneal and conjunctival epithelia and is responsible for providing a smooth and powerful refractive surface for clear vision and acts as the first line of defence against microbial infections[22].

Tear film constituents which are mucin, aqueous, and lipid components work in concert to provide these functions and any alterations to the quality or quantity of the tear film will disturb this fragile homeostasis. The mucin layer is produced by goblet cells in the conjunctiva while the aqueous layer is secreted by the main and accessory lacrimal glands. The mucin layer plays an important role in producing an even distribution of aqueous layer on the ocular surface. Generally, the lipids of the tear film originate at the meibomian glands, but also from other sources in the conjunctiva, cornea, and lacrimal glands. They compose of an interface between the underlying aqueous and mucin and the external environment. The tear film evaporation is affected by alterations to the quality or quantity of the lipids in the human tear film. Regulation of tear secretion is through a complex neurohormonal system[22].

Tear Film Structure and composition

The tear film was traditionally described as being composed of three separate and distinct layers which are mucin, aqueous, and lipid. However it is suggested that mixing between the mucin and aqueous layer occurs which creates a gradient of decreasing mucin concentration into the aqueous layer. This aqueous-mucin layer forms a hydrated gel which is then covered by the lipid layer to form a complex biology, which has its own highly ordered structure. For the sake of simplicity, the mucin, aqueous, and lipid layers are considered separately.

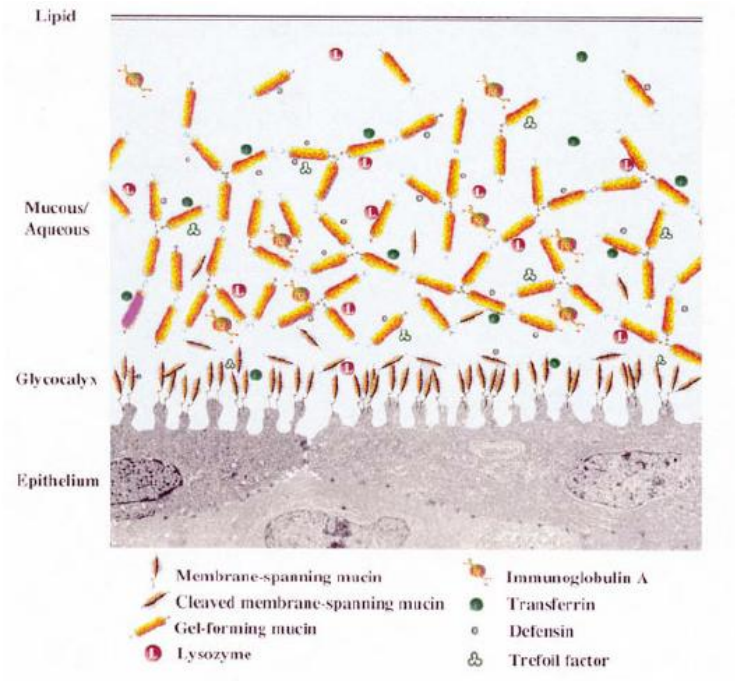


Fig.1 Tear Film Hydrated Gel[22].

Mucin Layer

Typically, the mucin which is found on the ocular surface is described as being associated with two sub-layers which are, an outer thicker and looser mucous blanket believed to be produced by the conjunctival goblet cells and an innermost tightly bound glycocalyx layer that is secreted by the microplicae of the conjunctival and corneal

epithelia and is in intimate contact with it[23]. The glycocalyx which is found along the apical surfaces of the epithelial cells is an extrinsic, carbohydrate-rich surface coat[24].

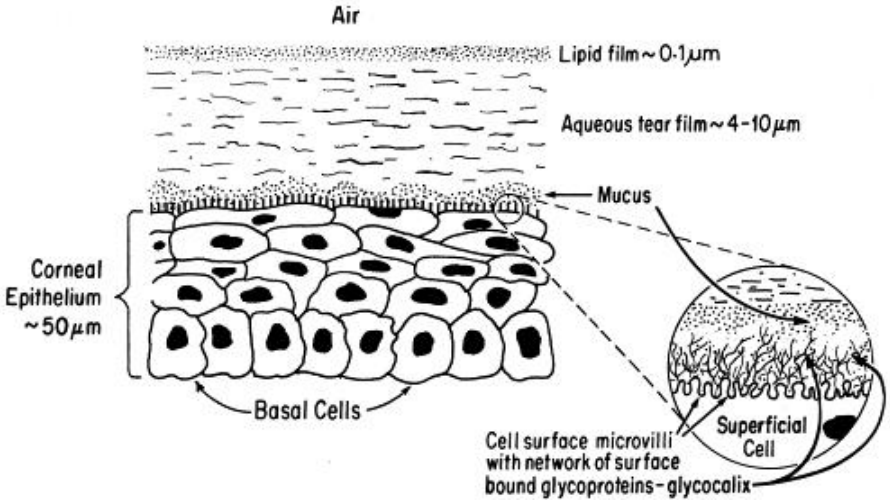


Fig 2. Precorneal tear film resting on the corneal epithelium[25].

Ocular mucus is composed of mucin, immunoglobulins, urea, salts, glucose, leukocytes, cellular debris, and enzymes[26]. Mucins can be defined as high molecular weight glycoproteinteins, hydrophilic in nature, that have at least 50-80% of their mass as carbohydrate, O-linked to serine and threonine residues present within tandem repeats of amino acids in their protein backbone[27]. An overall negative charge to the glycoprotein is imparted by the heavy glycosylation, making the mucins highly hydrophilic and able to admix with the aqueous layer and maintain water on the surface of the eye. As the technology to detect the genes has improved, the number of genes found, coding for mucin, have increased over the years. In the year 2000, 9 genes[28], in 2001, 14 genes[29], in 2004, 17 genes[25] and in the year 2007, 20 genes[27] were found out. The genes are named and indicated as MUC1, MUC2 etc. and numbered chronologically in the order of their discovery[29]. A common characteristic is the presence of a tandemly repeated nucleotide sequence located in the central part

of the gene and is found in all mucin coding genes. Each gene can result in the production of a specific mucin appropriately named MUC1, MUC2 etc.

The various functions of mucin on the eye are lubrication and protection of the ocular surface, anchoring of the aqueous tear layer to the ocular surface, protection of the epithelium from sheer force damage, drying and bacterial invasion and to provide a hydrophobic scaffold to hold other anti-microbial proteins to the eye[28,29]. Based on the conformation of the mucin molecule, mucins are classified as secretory mucins or membrane-associated mucins[30]. Secretory mucins are further divided into two groups. The first group consists of large gel-forming mucins and the second group consists of small soluble mucins.

Membrane-associated mucins form the glycocalyx which is a dense barrier to pathogen penetrance, at the epithelial cell–tear film interface[25]. The membrane associated mucins anchor the mucin on the apical surface of the epithelial cells by a hydrophobic transmembrane domain, they consist of a short cytoplasmic tail that extends into the cytoplasm, and an extracellular domain that reaches into the tear film[29].

Secretory mucins are secreted by the goblet cells and are responsible for the rheological properties of mucous while membrane-spanning mucins might interact with intercellular proteins[29].The genes which code for the secreted mucins are all found clustered in the same locus on chromosome 11p15[29,30].

The exact mechanisms for identification of soluble forms of the membrane-associated mucins in the tear film are still unknown[25,31,32]. Some of the possible mechanisms include ectodomain shedding which consists of cleavage and release of the extracellular domain into the tear film, posttranslational processing of the mucin into two subunits where one subunit remains anchored in the plasma membrane, and

the other soluble subunit is packaged in secretory granules and released into the tear film. Also some data suggested the mucin shedding from the cell surface over time leaves the oldest cells without microplicae and membrane associated mucins. The oldest cells adhere to the mucus of the tear film and lose their adhesive character with the loss of the mucin, and their removal via the nasolacrimal duct. Secretory mucins due to their anionic character move easily over the mucins composing the glycocalyx because of the repulsive forces between them[25].

During blinking, the secretory mucins act as a "cleaning crew," by moving through the tear fluid and collecting debris that can then be removed via the nasolacrimal duct. The large gel-forming mucins are probably the largest glycoproteins known based on their high molecular weight and are considered gel forming because they are responsible for the rheological properties of mucus[33]. The small soluble mucins lack cysteine-rich D domains and are present as monomeric species.

The conjunctival goblet cells secrete the majority of ocular mucins[26]. The ocular mucins are also produced by the stratified squamous epithelium of the cornea and conjunctiva, and there is new evidence that suggests that the lacrimal gland also contributes to mucin production[31,34,35].

Corneal and conjunctival stratified squamous cells contain the membrane-associated mucins MUC1, MUC4, and MUC (16.20,21,22). MUC1 is a likely candidate for the glycocalyx as it is present in the apical cell membranes of the superficial ocular surface cells[36,37]. Soluble forms of MUC1, MUC4, and MUC16 have also been detected in the tears and MUC4 has been detected in the lacrimal gland. MUC2 is a gel-forming secretory mucin, and MUC7 is a soluble secretory mucin which has been identified in tears and are both present in the conjunctiva but

the exact cellular source in the conjunctiva is unknown. MUC7 is also secreted by the lacrimal gland[35]. MUC5AC is a large gel-forming mucin, which is expressed by the goblet cells of the conjunctival epithelium and has been identified in tears in some individuals. MUC5AC is the major mucin providing the scaffolding of the mucus individuals which is present at the ocular surface[29].

Aqueous Layer

The middle aqueous layer of the tear film lies immediately beneath the oily layer. It is produced from the secretion of the lacrimal gland which is located in the superior lateral orbit. The aqueous layer is secreted onto the ocular surface from ducts in the superior fornix. There are also numerous scattered accessory lacrimal glands embedded within the conjunctival stroma which contribute to this aqueous layer. This layer consists of water, electrolytes, proteins, peptide growth factors, immunoglobulins, cytokines, vitamins, antimicrobials and hormones secreted by the lacrimal glands. The different electrolytes present in the tear film are sodium, potassium, magnesium, calcium, chloride, bicarbonate, and phosphate ions[38]. The electrolytes contribute to maintaining epithelial integrity of the ocular surface and are responsible for the osmolarity of tears, and act as a buffer to maintain a constant pH[39,40]. A global feature of dry eye syndrome is an increase in the osmolarity of the aqueous layer which damages the ocular surface directly and indirectly by triggering inflammation. Electrolyte concentration of this layer is similar to that of serum, resulting in an average osmolarity of 300mOsm/L[41].

The primary defence system of the ocular surface is composed of the nonspecific immunity conferred by lysozyme, lactoferrin, β-lysin, complement, defensins, and group II phospholipase A2 and the specific immunity of antibodies,

such as secretory immunoglobulin A[38]. In aqueous-deficient dry eye syndrome, the concentration of lysozyme, lactoferrin, lipocalin and immunoglobulin A are reduced which compromises the integrity of the defence system and may make the ocular surface more susceptible to infection, in addition to the symptoms of dry eye[44].

The antimicrobial proteins namely lysozyme, lactoferrin, and lipocalin are regulated and secreted in response to an intracellular stimulus which have a rate of secretion that approximately matches the rate of tear flow. Therefore, their concentration remains relatively constant with various flow rates[45].Immunoglobulins are constitutively produced and transported to the tear film from the conjunctiva. Thus, reflex tearing decreases the concentration of immunoglobulins and a decrease in tear flow increases their concentration[45].Lysozyme is an antimicrobial enzyme which lyses the bacterial cell walls in the same manner as penicillin. It is one of the most vital protein components in the tear film and acts in association with β-lysin which is an enzyme that attacks bacterial cell membranes. Lactoferrin is a protein which binds to metal, has antimicrobial properties and may enhance antibody activity against certain microorganisms. Lipocalin is a lipid-binding protein which scavenges potentially harmful hydrophobic molecules and has been shown to inhibit bacterial and fungal infections through sequestering microbial siderophores[46]. It interacts with meibomian lipids contributing to a stable tear film. It delivers them to the aqueous layer, and when mixed with other tear components, may be responsible for the high viscosity and low surface tension of tears[47].

The main and accessory lacrimal glands constantly replenish the aqueous volume. Most non-reflex tear production is from the glands of Krause and Wolfring, accessory lacrimal glands located in the palpebral conjunctiva of the upper eye lid and the superior conjunctival fornix. The lacrimal glands can provide a great volume of

aqueous tears when the ocular surface is presented with a noxious stimulus, such as a foreign body, chemical irritant, or epithelial injury.

A reflex loop that links the ocular surface, central nervous system stimulation, and the glands of the ocular surface drives the tear production neurally. The lacrimal functional unit comprises the cornea, conjunctiva, and meibomian glands of the ocular surface, the main and accessory lacrimal glands, and the neural pathways that connect them[48]. Afferent sensory nerves of the cornea and conjunctiva synapse with higher-order sensory neurons, autonomic, and motor efferent nerves in the brainstem. The tear production reduces and dryness occurs when that stimulus is interrupted by local or general anaesthesia, corneal nerve transection after LASIK, or neurotrophic infection. The autonomic nerve fibers innervate lacrimal and accessory glands, meibomian glands and conjunctival goblet cells.

Motor fibers from the facial nerve innervate the orbicularis oculi muscle and stimulate the blink reflex which distributes tears evenly over the ocular surface[49].

Lipid Layer

The meibomian oil secreted by the meibomian glands that are located within the tarsal plates forms the anterior layer of the tear film and is the major barrier to the evaporation from the ocular surface[50]. It is made up mainly of lipids and is referred to as the Tear Film Lipid Layer (TFLL).

The lipid layer is also responsible for providing stability to the tear film through interaction with the aqueous-mucin phase, providing a smooth optical surface for the cornea, and acting as a barrier against foreign particles.

McCulley and Shine have proposed that the anterior lipid layer is made up of two phases which are a polar surfactant phase and a nonpolar phase[50,51]. The polar

lipid phase is made up mainly of phospholipids and glycolipids and is multifunctional. The highly structured polar lipid layer acts as a surfactant between the hydrophilic aqueous mucin layer and the thick, nonpolar lipid layer which facilitates interaction with the aqueous–mucin layer and provides a barrier and offers a structural component on which the nonpolar phase depends[52]. The nonpolar phase is mainly composed of wax, cholesterol esters and triglyceride. It provides the air-tear film interface and is responsible for retarding evaporation[52].

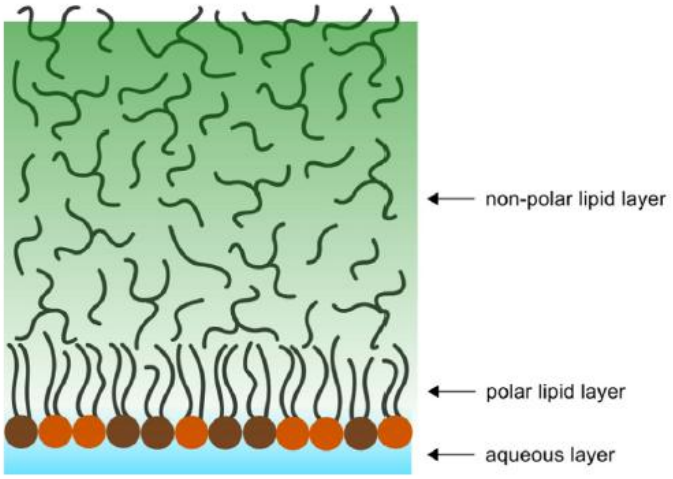


Fig 3. Tear Film Lipid Layer (TFLL)[53]

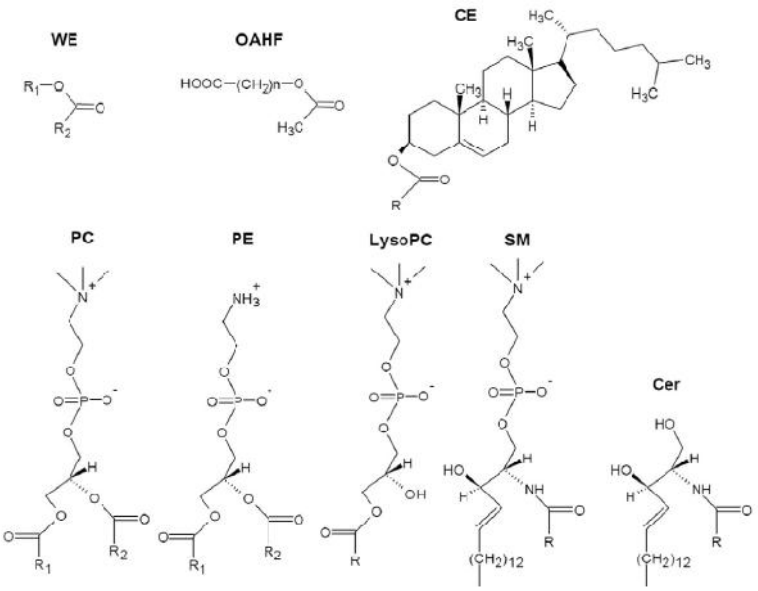


Fig 4[53]. The most abundant lipid classes in the tear film. Non-polar: wax esters (WE), cholesterol esters (CE). Polar : (O-acyl)-u-hydroxy fatty acids(OHAF), phosphatidylcholines(PC), phosphatidylethanolamines(PE), lysophosphatidylcholines(Lyso PC), sphingomyelins(SM), ceramides(Cer).

The most abundant lipid classes in the tear film:

1) Non-polar lipids are wax esters (WE), cholesterol esters (CE)
2) Polar lipids are O-acyl-u-hydroxy fatty acids, phosphatidylcholines, phosphatidylethanolamines,lysophosphatidylcholines, sphingomyelins, ceramides. The evaporation is decreased by approximately 90% to 95% by a normal tear film lipid layer[54,55]. The thickness of the lipid layer affects the rate of evaporation and it has been postulated that a reduction in thickness may cause evaporative dry eye[56]. The thickness of the polar sub-layer was estimated to be 2–9 nm[57]. The thickness of the polar sub-layer together with the mean total tear film lipid layer thickness of 42 nm, the thickness of the non-polar phase is 33–40 nm. Hence, at least 80% of the tear film lipid layer thickness can be attributed to the sublayer composed of non-polar lipids. The exact mechanisms are yet to be known, whether the lipid composition may also affect evaporation because it has been shown that the phospholipid content of meibomian oil is reduced in patients with dry eye and meibomian lipid composition is changed in anterior and posterior blepharitis[58].

The melting range of meibomian oil is dependent on lipid composition and may be lowered by the presence of branched and unsaturated fatty acids and alcohols[59]. The low melting range of meibomian oil which is 19.5°C to 32.0°C allows the lipid to exist in a relatively solid state and contributes to tear film stability. It is attributed to the variety of lipids present in the meibomian oil, facilitates meibomian delivery and contributes to tear film stability as the surface corneal temperature (32°C) is close to the upper limit of the melting range[59].

Meibomian oil secretion is aided by blink action and is a continuous process, occurring 24 hours per day during waking and sleeping hours[59]. The rate of secretion is controlled by neural, hormonal, and vascular influences. Considering that the preocular tear film holds 9 µg of lipid, the lid margin reservoir of oil in an adult male

has been estimated to contain approximately 300 µg of meibomian oil, which is more than adequate to refresh the lipid layer after each blink[60,61].

ROLE OF CONJUNCTIVA AND CORNEA

Though the tear film is the main protection against mechanical, bacterial, viral or chemical attack, the conjunctiva and the cornea of the eye form the last barrier of defence in preventing further penetration. The defensive mechanisms of each of these structures is different. The conjunctiva is highly reactive, richly supplied by blood vessels, lymphatics, and immunocompetent cells like Langerhans cells forming an effective immune system. The cornea, on the other hand, is quite unreactive, since it avoids inflammatory reactions in an attempt to preserve its transparency. The defence mechanisms arise from the limbic vessels and tear film including the tight junctions, local IgA, and the electrical charge on the epithelium[62].

Some of the features that operate to form a potent immunologic defence system for the ocular surface are the intrinsic anatomic barriers, bacteriostatic substances, mucus, normal flora, local antibodies and local T cell responses, similar to those seen in other mucosal surfaces[63].

Sensory innervation of the cornea

Corneal sensation is a function of the long ciliary nerves of the ophthalmic division of the fifth (trigeminal) cranial nerve. It is estimated that there are approximately 7000 sensory receptors per square millimetre in the human corneal epithelium, implying that injuries to individual epithelial cells may be adequate to give a sensation[64].

Traditionally, it was believed that most sensory nerves enter into the cornea along a transverse meridian (3 and 9-o'clock). However, presently it is believed that

nerve bundles enter the peripheral mid-stromal cornea in a radial fashion, along different meridians of the stroma. The thick stromal nerve trunks move from the periphery towards the center below the anterior third of the stroma due to the organization of the collagen lamellae[64]. Soon after entering the cornea, the main stromal bundles branch repeatedly and dichotomously into smaller fascicles that ascended into progressively more superficial layers of the stroma. Eventually, the stromal nerve fibers abruptly turn 90 degrees, penetrate Bowman's layer, and proceed toward the corneal surface. Throughout the peripheral and central cornea, the nerves penetrate the Bowman's layer[65].

After penetrating Bowman's layer, the large nerve bundles divide into degrees and continue parallel to the corneal surface, between Bowman's layer and the basal epithelial cell layer, forming the sub-basal nerve plexus. Sub-basal fibers subsequently form branches that turn upward and enter the corneal epithelium between the basal cells to reach the wing cells, where they terminate[65,66].For normal blinking and tearing reflexes, intact corneal innervation is important, which in turn is essential for maintaining the integrity of the ocular surface. Under normal physiological conditions, sensory nerves in the cornea transmit an afferent stimulation signal through the ophthalmic division of the trigeminal nerve to the brain stem. The parasympathetic and sympathetic nerves that innervate the lacrimal gland tranmit the efferent signal to the lacrimal gland that drives tear production and secretion[67].Damage to this neural pathway influences both basal and stimulated tear production by interrupting the normal regulation of lacrimal gland secretion. This is one of the major pathogenic pathways in induction of postoperative dry eye in patients undergoing ophthalmic surgeries. Most surgical procedures that cause denervation of the cornea result in impaired epithelial wound healing, increased

epithelial permeability, decreased epithelial metabolic activity and loss of cytoskeletal structures associated with cellular adhesion[68]. The normal corneal epithelium has the highest density of sensory nerve endings throughout the human body. These receptors are located between the wing cell layers of the corneal epithelium and are protected from direct environmental stimulation by the overlying tear film and the intact surface epithelial cells. The environmental stimuli have greater access to the sensory nerve endings which is an important cause for the marked symptoms of ocular irritation experienced by dry eye patients, even in mild cases, in the early stages of dry eye, in the presence of an unstable tear film and superficial punctate keratopathy.

However, it has been demonstrated that hyposecretion of tears may lead to pathologic alterations in corneal nerves and a decrease in corneal sensitivity which subsequently increase the dry eye state in these patients[69].The exposure of nerve endings, in addition to tear hyperosmolarity and increased expression of a number of inflammatory cytokines, including interleukin (IL)-1alpha and IL-1beta, IL-6, and tumor necrosis factor-alpha may cause injury to the corneal nerves and trigger neuraldegeneration[70]. Furthermore, the presence of nerve fibers invaginating corneal epithelial cells and keratocytes suggests that both cell types are directly innervated. Immunocytochemical staining indicates the presence of different neuropeptides within the cell soma and peripheral axonal fibers of corneal neurons suggesting that they are functionally heterogeneous. Thus, so far seventeen different neuropeptides and neurotransmitters have been described in the corneal nerves[71]. It is observed that intact corneal nerve fibers exert trophic influences on the corneal epithelium and neuroregulation is responsible for maintenance of the integrity and repair of the ocular surface[68].The mechanism underlying corneal damage is commonly believed to be related to reduced levels of neurotransmitters in patients with reduced corneal

sensation and dry eye, for instance, following surgery. Neuroimmunomodulation of the cornea may involve Peptidergic transmitters which are present in nerve fibers[65]. Enhanced epithelial cell proliferation is strongly mediated by the neurotransmitters and nerve growth factors released from corneal nerve endings. For instance, it has been reported that acetylcholine derived from the corneal sensory nerve endings, enhances epithelial cell growth by epithelial mitosis in the cornea which is due to increased intracellular levels of cyclic guanosine monophosphate[72].

REGULATION OF TEAR PRODUCTION

A reflex loop involving a neuronal loop with the cornea, conjunctiva, meibomian and lacrimal glands regulates the production of tears. The ophthalmic branch of trigeminal nerve carries the afferent impulses, which are triggered by subconscious stimulation of the corneal free nerve endings. These impulses integrate with the central nervous system, and the sympathetic pathway and the efferent to the lacrimal and meibomian glands and the goblet cells in conjunctiva are formed by the fibres via pterygopalatine ganglion. Therefore, coordinated secretion of all the major tear film components maintains the ocular surface homeostasis which is stimulated by the efferent branch of the neuronal loop. Irritation of the ocular surface due to excess evaporation, less humidity or contact lens material chronically stimulate the afferents causing an increase in lacrimal secretion.

Blinking facilitates the formation of the complexly structured precorneal tear film by spreading of the tear fluid evenly. Between two blinks, the tear film architecture that gets disturbed due to environmental factors and evaporation of fluid is reconstructed and spread by the action of orbicularis oculi and levator muscles in the eyelid[73].

The pathway for reflex blinking is better understood than that of spontaneous blinking. Trigeminal sensory nerve fibres which projects to the motor neurons of facial nerve stimulates orbicularis oculi muscle which then results in closure of the upper eyelid. Therefore, the corneal afferents, second order trigeminal brainstem nuclei and the motor neurons of orbicularis oculi are the three main components of the reflex blinking pathway.

A continuous firing of impulses of corneal cold thermoreceptors, levels of dopamine in the central nervous system, cognitive status and a combination of many factors acting at different levels maintains spontaneous blinking[73].

A reduction in the reflex stimulated lacrimal gland secretion and reduction in the blink rate which thereby increases the evaporative loss of tears are the two ways which favours the development of dry eye by decreasing the sensory drive from the ocular surface. Thus in conditions like post-refractive surgery or even normal aging there is damage to sensory nerves, affecting the reflex arc which results in reduced tear secretion and dry eye symptoms.

A study by Li XM et al showed that after cataract surgery, the incidence of dry eye increased dramatically, the lacrimal river line became narrow, break-up time and schirmers test I values decreased in patients after cataract surgery[74]. Khanal et al in their study found that deterioration in corneal sensitivity and tear physiology is seen immediately after phacoemulsification. They found that deterioration in corneal sensitivity and tear physiology is seen immediately after phacoemulsification[75]. Cho YK et al had concluded, in terms of subjective symptoms of dry eye, there is indeed an aggravation of dry eye symptoms after cataract surgery[76]. Liu and his coworkers compared 25 diabetic cataract patients with 20 age-matched non-diabetic cataract patients[77]. They found that tear secretion was reduced in diabetic cataract patients

after phacoemulsification, which worsened dry eye symptoms and predisposed those patients to ocular damage. However, in non-diabetics there was no significant, persistent change in tear film. Kasetsuwan et al reported that signs and symptoms of dry eye occurred as early as 7 days post-phacoemulsification and the severity pattern improved over time[78].

Dry eye syndrome

In a restructured definition of dry eye and its classifications by the International Dry Eye Workshop, dry eye is defined as a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface.It is a disorder of the lacrimal functional unit (LFU), an integrated system comprising of lacrimal glands, ocular surface (cornea, conjunctiva, and meibomian glands) , lids and the sensory and motor nerves that connect them[79].Sensory fibres from Trigeminal nerve arising from the ocular surface run to the superior salivary nucleus in the pons, from where efferent fibers pass to the pterygopalatine ganglion in the nervus intermedius. Here, postganglionic fibers arise, which terminate in the lacrimal gland, nasopharynx, and vessels of the orbit. Trigeminal afferents and the somatic efferent fibers of the seventh cranial nerve form another neural pathway which controls the blink reflex. Higher centers feed into the brainstem nuclei, and there is a rich sympathetic supply to the epithelia and vasculature of the glands and ocular surface[79].

Any disease or damage to any component of the LFU (the afferent sensory nerves, the efferent autonomic and motor nerves, and the tear-secreting glands) can destabilize the tear film and lead to an ocular surface condition that expresses itself as

dry eye. Sensory impulses, which arise from the ocular surface, a vital unit of LFU, play an important role in the maintenance of resting tear flow, and this is thought to be the main area of possible damage in ophthalmic surgeries involving the cornea. When the interactions between stabilizing tear film constituents are compromised by decreased tear secretion, delayed clearance, and altered tear composition, Tear film stability, a hallmark of the normal eye, is threatened. This will lead to ocular surface inflammation as a secondary consequence. In response to ocular irritation, reflex tear secretion is envisioned as the initial compensatory mechanism, but, with time, inflammation accompanying chronic secretory dysfunction and a decrease in corneal sensation eventually compromises the reflex response and leads to an even greater tear film instability[79].

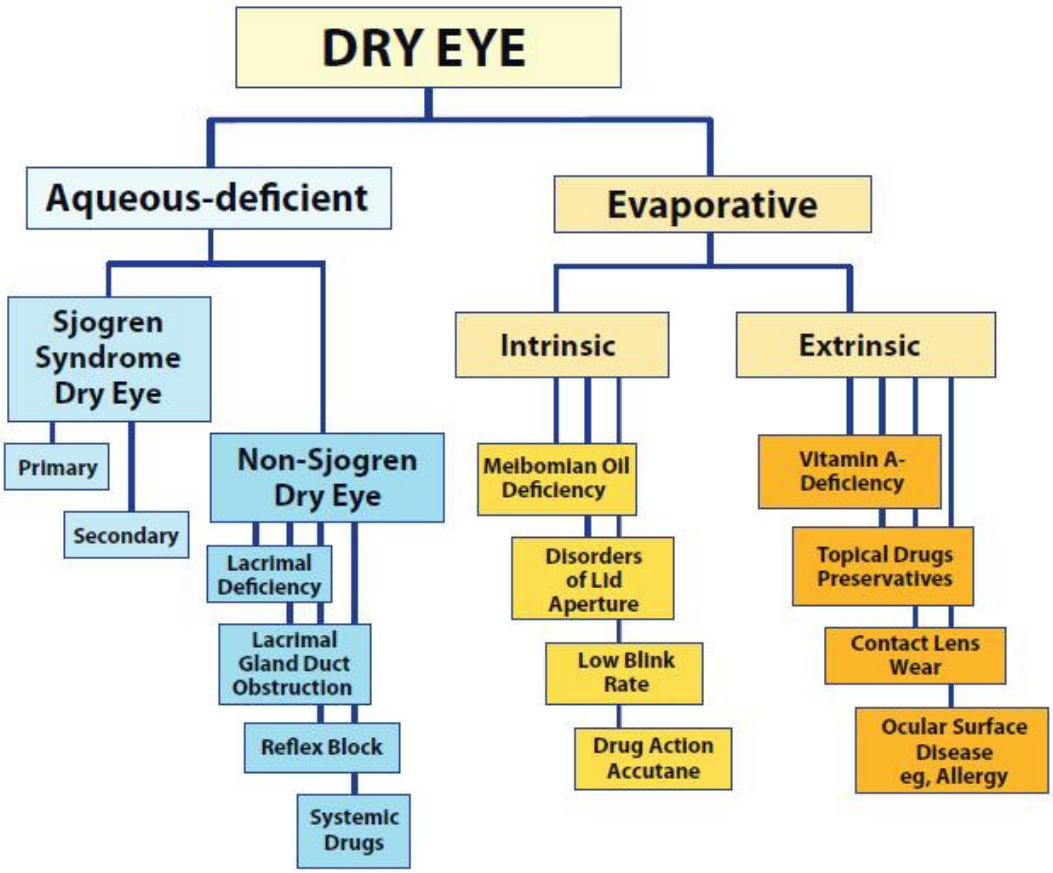


Fig 5. Classification of Dry Eye[79]

The major classes of dry eye are aqueous tear-deficient dry eye (ADDE) and evaporative dry eye (EDE). Dry eye can be initiated in any of these classes, but they are not mutually exclusive. It is recognized that disease initiated in one major subgroup may coexist with or even lead to events that cause dry eye by another major mechanism. This is part of a vicious circle of interactions that can amplify the severity of dry eye[79].

The major classes and subclasses of dry eye are

1. Aqueous Tear-Deficient Dry Eye (Tear Deficient Dry Eye; Lacrimal Tear Deficiency)

ADDE implies that dry eye is due to a failure of lacrimal tear secretion. In any form of Dry eye, where there is lacrimal acinar destruction or dysfunction there is reduced lacrimal tear secretion and volume which results into dryness[80,81]. Reduced aqueous tear pool will cause tear hyperosmolarity although the water evaporates from the ocular surface at normal rates. This Tear film hyperosmolarity causes hyperosmolarity of the ocular surface epithelial cells and stimulates a cascade of inflammatory events involving MAP kinases and NF*k*B signalling pathways[82,83] and the generation of inflammatory cytokines (interleukin (IL)-1α; -1β; tumor necrosis factor (TNF)-α) and matrix metalloproteinases (MMP-9)[84].

When lacrimal dysfunction is due to lacrimal gland infiltration and inflammation, inflammatory mediators are generated in the gland. These inflammatory mediators generated in the glad are assumed to find their way into the tears and be delivered to the ocular surface.

However, when such mediators are detected in the tears, it is difficult to know whether they derive from the lacrimal gland itself or from the ocular surface (conjunctiva and cornea).

ADDE has two major subclasses, Sjogrens syndrome dry eye (SSDE) and non-SSDE.

a. Sjogren Syndrome Dry Eye

Sjogren syndrome (SS) is an exocrinopathy in which the lacrimal and salivary glands are targeted by an autoimmune process; other organs are also affected. The lacrimal and salivary glands are infiltrated by activated T-cells, which cause acinar and ductular cell death and hyposecretion of the tears or saliva. Expression of autoantigens at the surface of epithelial cells (eg, fodrin, Ro and La) is caused by the inflammatory activation within the glands[83] and the retention of tissue-specific CD4 and CD8 T-cells[86].

The local release of inflammatory cytokines or the presence of circulating antibodies (eg, anti-M3 antibody) directed against muscarinic receptors within the glands, causes a potential reversible neurosecretory block, amplifying the Hyposecretion[87-89].

There are two forms of SS

Primary SS consists of the occurrence of ADDE in combination with symptoms of dry mouth, in the presence of autoantibodies, evidence of reduced salivary secretion and with a positive focus score on minor salivary gland biopsy[91].

Secondary SS consists of the features of primary SS together with the features of an overt autoimmune connective disease, such as rheumatoid arthritis, which is the most common, or systemic lupus erythematosis, polyarteritis nodosa, Wegener's

granulomatosis, systemic sclerosis, primary biliary sclerosis, or mixed connective tissue disease[79].

The precise triggers leading to autoimmune acinar damage are not fully known, but risk factors include genetic profile, androgen status (a low androgen pool favoring an in flammatory environment within the target tissues), and exposure to environmental agents, ranging from viral infections affecting the lacrimal gland to polluted environments.

A nutritional deficiency in omega-3- and other unsaturated fatty acids and unsupplemented intake of vitamin C has also been reported in patients with SS[92].

It is commonly accepted that environmental factors leading to increased evaporative water loss from the eye (Eg. low humidity, high wind velocity, and increased exposure of the ocular surface) may act as a trigger by causing inflammatory events at the ocular surface through a hyperosmolar mechanism.

The ocular dryness in SSDE is due to lacrimal hyposecretion and the accompanying characteristic inflammatory changes in the lacrimal gland, together with the presence of inflammatory mediators in the tears and within the conjunctiva[93].

b. Non-Sjogren Syndrome Dry Eye

Non-SSDE is a form of ADDE due to lacrimal dysfunction, where the systemic autoimmune features characteristic of SSDE has been excluded. The most common form is age-related dry eye, to which the term KCS has sometimes been applied in the past.

The different forms of Non -SSDE are:

1) Primary Lacrimal Gland Deficiencies

Age-Related Dry Eye: With increasing age in the normal human population, there is an increase in ductal pathology that could promote lacrimal gland dysfunction by its obstructive effect[94]. These alterations include periductal fibrosis, interacinar fibrosis, paraductal blood vessel loss and acinar cell atrophy[94].

Congenital Alacrima: Congenital alacrima is a rare cause of dry eye in youth. It is also part of certain syndromes including the autosomal recessive, triple A syndrome (Allgrove syndrome), in which congenital alacrima is associated with achalasia of the cardia, Addison's disease, central neurodegeneration, and autonomic dysfunction. It is caused by mutations in the gene encoding the protein ALADIN, which plays a role in RNA and/or protein trafficking between the nucleus and cytoplasm[95].

Familial Dysautonomia: Lacrimal dysfunction is a major feature of the autosomal recessive disorder, familial dysautonomia (Riley Day syndrome), in which a generalized insensitivity to pain is accompanied by a marked lack of both emotional and reflex tearing, within a multisystem disorder. There is a developmental and progressive neuronal abnormality of the cervical sympathetic and parasympathetic innervations of the lacrimal gland and a defectivesensory innervation of the ocular surface, which affects both small myelinated and unmyelinated trigeminal neurons[96]. The chief mutation affects the gene encoding an IκB kinase-associated protein.

2) Secondary Lacrimal Gland Deficiencies

Lacrimal gland infiltration: Lacrimal secretion may fail because of inflammatory infiltration of the gland, as in Sarcoidosis where the infiltration of the lacrimal gland by sarcoid granulomata may cause dry eye. In lymphoma, the infiltration of the

lacrimal gland by lymphomatous cells causes dry eye. In AIDS, dry eye may be caused by lacrimal gland infiltration by T-cells. However, in AIDS-related dry eye, unlike the situation in SSDE, there is a predominance of CD8 suppressor cells, rather than CD4, helper cells.In Graft vs host disease (GVHD), dry eye is a common complication of GVHD disease, occurring typically around 6 months after hematopoietic stem cell transplantation. It is caused in part by lacrimal gland fibrosis due to co-localization of periductal T-lymphocytes (CD4 and CD8) with antigen-presenting fibroblasts[97].

Lacrimal gland ablation: The ducts of the main lacrimal gland pass through its palpebral part, so that excision of the palpebral part will be expected to have the same effectas excision of the main gland. Dry eye may be caused by partial or complete ablation of the lacrimal gland at any age, but is not an mandatory consequence, probably because accessory gland and conjunctival secretion may compensate in some cases[81].

Lacrimal gland denervation: Parasympathetic denervation of the human lacrimal gland may cause dry eye[98].

3) Obstruction of the Lacrimal Gland Ducts

Obstruction of the ducts of the main palpebral and accessory lacrimal glands leads to aqueous-deficient dry eye and may be caused by any form of cicatrising conjunctivitis.In these disorders, it is not uncommon for conjunctival scarring to cause a cicatricial obstructive MGD. In addition, lid deformity influences tear film spreading by affecting lid apposition and dynamics[79].

Specific conditions are

Trachoma: Trachoma is a cause of blindness on a global scale, in which corneal opacity and blindness are caused by a combination of tarsal and conjunctival scarring, trichiasis and a cicatrizing meibomian gland obstruction. Dry eye is part of the overall picture, resulting from lacrimal duct obstruction, lid mal-apposition, and a deficient tear film lipid layer[99].

Cicatricial pemphigoid and mucous membrane pemphigoid: Cicatricial and mucous membrane pemphigoid are mucocutaneous disorders characterized by blistering of the skin and mucous membranes, leading to severe and progressive conjunctival scarring. Dry eye may be caused by lacrimal obstruction, cicatricial MGD, and/or poor lid apposition.

Erythema multiforme: This is an acute, self-limited mucocutaneous disorder usually Precipitated by drugs, infection or malignancy. Conjunctival scarring can lead to dry eye in the manner outlined above.

Chemical and thermal burns: Diffuse burns may cause sufficient scarring to cause dry eye[100].

4) Reflex Hyposecretion

a) Reflex Sensory Block

A reduction in sensory drive from the ocular surface is thought to favor the occurrence of dry eye in two ways, first, by decreasing reflex-induced lacrimal secretion, and, second, by reducing the blink rate and thus, increasing evaporative loss.

Contact Lens Wear: A reduction in corneal sensitivity occurs in wearers of hard- and extended wear- contact lenses (CLs), possibly contributing to dry eye symptoms in this group of patients.

Diabetes: Diabetes mellitus has been identified as a risk factor for dry eye in several studies, including large population studies. It has been suggested that the association may be due to diabetic sensory or autonomic neuropathy or to the occurrence of microvascular changes in the lacrimal gland[101].

Neurotrophic keratitis: Extensive sensory denervation of the anterior segment, involving the cornea and the bulbar and palpebral conjunctiva, as a component of herpes zoster ophthalmicus or induced by trigeminal nerve section, injection, or compression or toxicity, can lead to neurotrophic keratitis. This condition is characterized by features of dry eye, such as tear instability, diffuse punctate keratitis, and goblet cell loss, and also, most importantly, the occurrence of an indolent or ulcerative keratitis, which may lead to perforation. The sensory loss leads to a reduction of lacrimal secretion and a reduction in blink rate. In addition, it is expected that there is a loss of trophic support to the ocular surface after sensory denervation, due to a deficient release of substance-P or expression of nerve growth factor[102].

b) Reflex Motor Block

Central damage to the VII cranial nerve, involving the nervus intermedius, leads to dry eye due to loss of lacrimal secretomotor function. The nervus intermedius carries postganglionic, parasympathetic nerve fibers (of pterygopalatine ganglion origin) to the lacrimal gland. Dry eye is due to lacrimal hyposecretion in addition to incomplete lid closure[79].

2. Evaporative Dry Eye

EDE is due to excessive water loss from the exposed ocular surface in the presence of normal lacrimal secretory function. Its causes have been described as *intrinsic*, where they are due to intrinsic disease affecting lid structures or dynamics,

or *extrinsic*, where ocular surface disease occurs due to some extrinsic exposure. The boundarybetween these two categories is inevitably blurred[79].

a. Intrinsic Causes

1) Meibomian Gland Dysfunction

Meibomian gland dysfunction, or posterior blepharitis, is a condition of meibomian gland obstruction and is the most common cause of evaporative dry eye[103]. Its multiple causes and associations include dermatoses, such as acne rosacea, seborrhoeic dermatitis, and atopic dermatitis. Less common but important associations include the treatment of acne vulgaris with isotretinoin, which causes reversible meibomian gland atrophy, loss of acinar density on meibography, and reduced volume and increased viscosity of expressed excreta. MGD can be primary or secondary, simple or cicatricial.

2) Disorders of Lid Aperture and Lid/Globe Congruity or Dynamic

Drying of the ocular surface can be due to poor lid apposition or due to lid deformity, leading to exposure or poor tear film resurfacing. Increasing palpebral fissure width correlates with increased tear film evaporation. Dry eye problems may be caused by problems of lid congruity after plastic surgery of the lids.

3) Low Blink Rate

Drying of the ocular surface may be caused by a reduced blink rate, which lengthens the period during which the ocular surface is exposed to water loss before the next blink[79].

b. Extrinsic Causes

1) Ocular Surface Disorders

Disease of the exposed ocular surface may lead to imperfect surface wetting, early tear film breakup, tear hyperosmolarity, and dry eye. Causes include vitamin A deficiency and the effects of chronically applied topical anesthetics and preservatives.

2) Contact Lens Wear

Contact lens wear is prevalent in the developed world. The causes of CL-related symptoms and of lens intolerance are, therefore, of personal and general economic importance. discomfort and dryness are the primary reasons for CL intolerance[79].

3) Ocular Surface Disease

There is evidence that various forms of chronic ocular surface disease result in destabilization of the tear film and add a dry eye component to the ocular surface disease[79].

4) Allergic Conjunctivitis

Allergic conjunctivitis takes several forms, which include seasonal allergic conjunctivitis, vernal keratoconjunctivitis, and atopic keratoconjunctivitis. The general mechanism leading to disease is that exposure to antigen leads to degranulation of IgE-primed mast cells, with the release of inflammatory cytokines. Surface irregularities on the cornea (punctate epithelial keratitis and shield ulcer) and conjunctiva can lead to tear film instability and, hence, to a local drying component to the allergic eye disease[79].

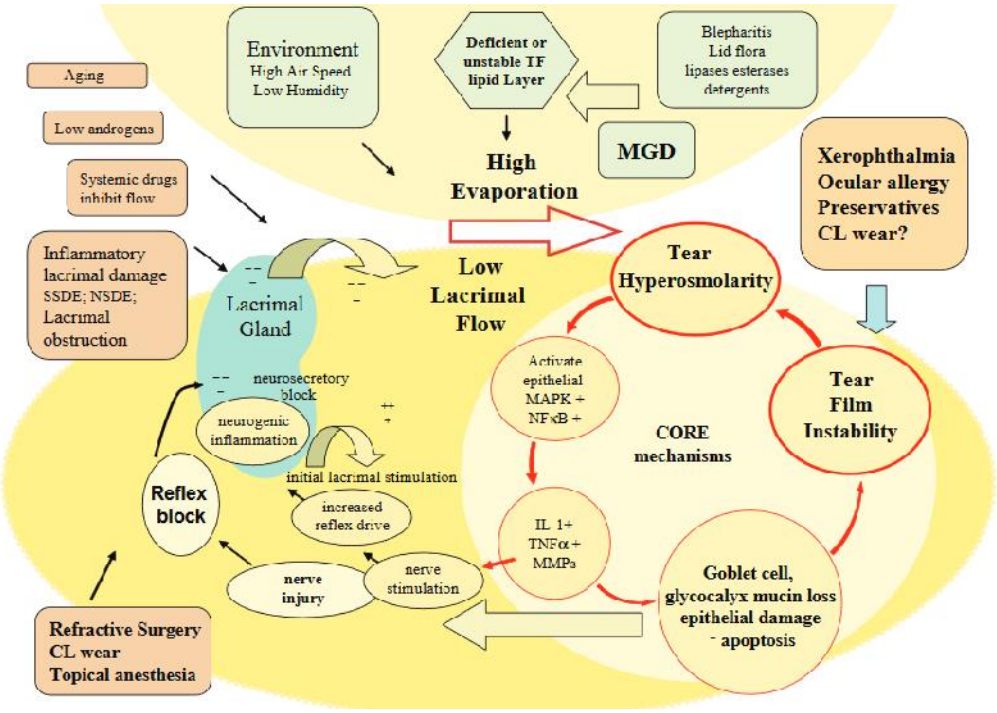


Fig 6. Pathophysiology of Dry eye disease[79]

Decreasing reflex-induced lacrimal secretion (tear deficient dry eye) and by reducing the blink rate and, consequently, increasing evaporative loss (EDE) are two exclusive and functionally diverse mechanisms causing dry eye due to disruption of a sufficient sensory drive from the ocular surface.

Corneal nerve alterations, either as a primary reason for tear hyposecretion or just the outcome of dryness of the ocular surface, have crucial effects on the integrated system of the LCU and can compromise various aspects of it, such as blinking, the tear reflex, and trophism of the epithelial cells, thus neatly contributing to the increase of the vicious circle of hypo-tearing leading to inflammation causing cell/nerve damage.

Different risk factors for dry eye have been identified, which include increasing age, various systemic diseases including diabetes, arthritis, allergy, and systemic medications, hormonal changes (menopause), neural alterations, ocular conditions (glaucoma, pterygium, meibomian gland dysfunction, lacrimal duct obstruction, contact lens wear) or environmental influences (climate, cigarette

smoking) and many others. Most dry-eye symptoms result from an abnormal, non-lubricative ocular surface that increases shear forces under the eyelids and diminishes the ability of the ocular surface to respond to environmental challenges. Dry eye manifestations could be described as visual fatigue, secretion, foreign body sensation, eyelids heaviness, dryness, uncomfortable eyes, pain, tears, blurred vision, itchiness, photophobia, and eye redness[79].

ASSESSMENT OF TEAR FILM

There are various methods to assess the tear film and its functioning. Clinical tests in an attempt to measure the tear production have been in use since over hundred years. Some of these include:

1. Schirmers test
2. Tear breakup time
3. Tear osmolarity
4. Ocular surface staining
5. Phenol red test
6. Tear meniscometry

SCHIRMERS TEST

The Schirmers test is one of the most routinely used tests for tear film. It is a simple test assessing the aqueous tear production. The first test is performed by inserting a strip of no.42 Whatman paper folded at five millimeter and placed in the lower fornix, the length of wetting of the strip in millimeters is noted at end of five minutes. This represents the total tear secretion. The Schirmers II measures reflex secretion and is performed similarly after application of a topical anesthetic before inserting the strip. Less than 6mm of wetting is considered diagnostic of dry eye. Value below 10mm for Schirmers without anesthesia, being diagnotic of dry eye. Strip meniscometry is a variation of this, and is done by placing a strip of

polyethylene terephthalate covered with a urethane-based material with a central ditch of nitrocellulose membrane filter paper in natural blue dye. A cut off value for 5 seconds is taken as 4mm.

TEAR BREAKUP TIME:

This is a commonly used test to assess tear film stability. The break up time is the interval between a complete blink and the appearance of the first dark spot in the tear film. It is best observed by staining with dyes like fluorescein or lissamine green and viewing under a cobalt blue filter. The subject is instructed to blink naturally three times and then to not blink until instructed[104].

A value of less 10 seconds is considered abnormal and studies suggest a cut off of 5 seconds when lesser or more controlled amount of fluorescein is used. Since the test can be affected by dyes, humidity or temperature of the environment, non-invasive breakup time measurements has now become popular as it does not require instillation of a dye and it is done by the observing the reflection of a grid pattern on the cornea. Automated assessment has also been developed for this test[105].

TEAR OSMOLARITY

Tear osmolarity is measured by using specialized osmometers. The tear osmolarity is classified as normal (302.2 ± 8.3 mOsm/L), mild-to moderate (315.0 ± 11.4 mOsm/L) and severe (336.4 ± 22.3 mOsm/L)[106].

STAINING OF OCULAR SURFACE:

The damage to the epithelial cells on the exposed surface of the eye can be viewed with the help of staining with various dyes. The commonly used dyes are sodium fluorescein, lissamine green and Rose Bengal. Fluorescein stains cells that have a disrupted tight junctions or glycocalyx rose Bengal stains the dead cells and

epithelial cells with a disrupted mucin or glycocalyx coating. Lissamine green is a vital dye and only stains a cell if the cell membrane is damaged, irrespective of absence of mucin. Fluorescein 2% and lissamine green 1% are the ideal concentrations to be used. Various grading systems for recording the staining severity on the cornea and conjunctiva are available such as van Bijsterveld system[107], the Oxford Scheme[108], the National Eye Institute/Industry Workshop guidelines[108], the Collaborative Longitudinal Evaluation of Keratoconus (CLEK) schema etc.

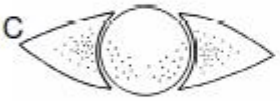
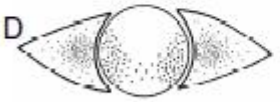
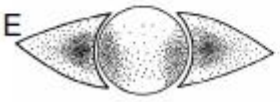
PANEL	GRADE	CRITERIA
A 	0	Equal to or less than panel A
B 	I	Equal to or less than panel B, greater than A
C 	II	Equal to or less than panel C, greater than B
D 	III	Equal to or less than panel D, greater than C
E 	IV	Equal to or less than panel E, greater than D
>E	V	Greater than panel E

Fig 7.The Oxford Grading system used to estimate surface staining of conjunctiva and cornea[109].

PHENOL RED TEST

The phenol red test measures the tear volume and is useful for detecting aqueous deficiency.

A cotton thread that is impregnated with phenol red is placed in the lower fornix. The colour turns to red when wetted by tears that are slightly alkaline. Therefore, the length of the red colour is noted in millimeters after 15 seconds. A value of less than 9mm is considered abnormal.

The advantage that is claimed over Schirmers is that the small size of the thread as well as the minimal amount of phenol red limit the elicitation of reflex tearing. However, some studies have shown no correlation between the test and the tear volume determined with other methods.

TEAR MENISCOMETRY

The assessment of tear meniscus by its height or volume is called meniscometry. It is one the most direct and non invasive methods to study the tear film volume. The tear meniscus height, cross sectional area and radius of curvature are measured by using a slit lamp.

Specialized systems with digital video recording are also available. These have an advantage that they do not require fluorescein application and provide dynamic visualization of the film. Optical coherence tomography has also been developed as a technology for assessing tear meniscus. It is simple and non invasive, although disadvantages are that the measurements are operator and instrument dependant, and the analysis is time consuming[110].

IMPRESSION CYTOLOGY

Impression cytology is a simple method for assessing various ocular surface disorders. This technique involves removal of the first three superficial layers of epithelium by applying cellulose acetate filters or biopore membranes. The cells are analyzed by methods like immunoblotting, microscopy, polymerase chain reaction, immunocytochemistry or flow cytometry. It helps detect early changes of metaplasia before it is clinically manifest. Various grading systems are applied to analyze the squamous metaplasia, based on the density, affinity for cytoplasm staining, morphology, nucleus-cytoplasm ratio of the conjunctival epithelial and goblet cells[111]. Squamous metaplasia and reduction in goblet cell count are noted when there is a disturbance in the ocular surface homeostasis.

MATERIALS AND METHODS

The present study was conducted in the Department of Ophthalmology, KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi to know the prevalence of dry eye in patients undergoing phacoemulsification surgery and also to analyze dry eye in relation to microscopic light exposure time. The study period was between 1stJanuary 2017 to 31st December 2017. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belagavi.

Source of data:

All patients diagnosed with cataract and admitted for the purpose of phacoemulsification cataract surgery at Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Method of collection of data

Study Design: A cross-sectional study

Study Period: One year – 1st January 2017 to 31st December 2017.

Sample Size:

Sample size of 100 cases.

Sample Size was calculated using the formula
$n = z^2 \times p \times q / d^2$
where n = sample size
z = constant (2)
$n = 4 \times p \times q / d^2$
p = 50
q = (100-p) = (100-50) = 50
d = 20% of p = 20 / 100 x 50 = 10
therefore, $n = 4 \times 50 \times 50 / 10^2 = 10000 / 100$
$= 100$

Selection Criteria:

Inclusion criteria

Patients with age related cataract more than 45 years willing to undergo phacoemulsification surgery.

Exclusion criteria

Patients with-

1. Pre existing ocular disease like:
    a. Dry eye
    b. Complicated cataract
    c. Glaucoma
    d. Uveitis
    e. Disorder of lids and nasolacrimal duct pathway
    f. Ocular allergies
    g. Pterygium
2. Previous ocular surgery
3. Current smoker
4. Any intra operative complications during this surgery
5. Insertion of Anterior chamber intraocular lens
6. Diabetes mellitus, Hypertension and on long term medication.

Methodology proper

1. All the patients who satisfy the inclusion criteria were included in the study. The patients were enrolled into the study and written informed consent was taken after explaining the procedure and associated risk.
2. Data regarding demographic parameters such as age, sex, occupation and address were noted on a predesigned proforma at the time of first visit.
3. Detailed history of following symptoms was noted:

- H/O Diminution of vision RE/LE
    A. Duration
    B. Gradual/Sudden
    C. Progression/static
    D. Distant/Near vision
    E. Visual improvement with bright light or dim light
    F. Painful/ Painless
- Diplopia/Polyopia
- Photophobia
- Flashes of light
- Coloured halos
- Floaters
- Watering
- Redness
- Discharge
- Black spots in front of the eye
- Curtain falling in front of the eyes

- H/O wearing glasses
- H/O Diabetes Mellitus, Hypertension.

4. History was followed by ocular examination on the day prior to surgery.

- Visual acuity testing for distance and near using Snellen's distant chart and Jaeger's near vision chart respectively, both unaided and aided.
- External ocular examination
- Slit lamp biomicroscopic examination
- IOP was measured with Non Contact Tonometer
    - ✓ Detailed Slit lamp examination was done for the evaluation of lens for the type of cataract and grading of the cataract
- A thorough posterior segment evaluation was done using direct and indirect ophthalmoscopy.
- Keratometry
- A-scan biometry was done for calculation of the power of the intraocular lens to be used.
- Patency of the lacrimal drainage pathway was checked by lacrimal sac syringing.
- Basal parameters such as pulse rate, blood pressure, random blood sugar levels were assessed.
- Xylocaine sensitivity test was done.
- **Pre-operative Dry eye work-up -** The patients were assessed for dry eyes and tear film was quantified using

  - Schirmer’s test I

  - Tear film break up time

  - Lissamine green staining of cornea and conjunctiva.

Surgical technique

- All the patients underwent Phacoemulsification surgery under peribulbar block.

The steps of the surgery-

- Under all aseptic precautions eye painted with povidone iodine and draped, universal eye speculum put.
- Superior rectus bridle suture was taken.
- Fornix based conjunctival flap raised, hemostasis achieved with wet field cautery.
- Scleral incision taken of 6.5mm which is made 1.5mm posterior to surgical limbus with 11 number blade at 12 o'clock position.
- Selfsealing sclerocorneal tunnel was made using a crescent knife and dissection continued 1 mm into clear cornea
- Side port entry was made at 9 o’clock and 3 o’clock position.
- Anterior chamber was entered from the anterior limit of sclerocorneal tunnel using a 2.8 mm keratome
- Trypan blue dye was used.
- Formation of a capsulorrhexis with a cystitome.
- Hydrodelineation.
- Lens removal through phacoemulsification and aspiration of the lens substance
- Aspiration of the remaining lens cortex with a bimanual irrigation/aspiration hand pieces.
- Injection of viscoelastic material into the anterior chamber followed by insertion of an Intraocular Lens into the capsular bag.
- Topical antibiotic eye drops were put.
- Eye padded and patched.

Intra-operative measurement-

-Intra-operatively, the total duration of microscopic light exposure during surgery was noted down in every case, which was measured from the start of microscopic light exposure on the ocular surface till the end of the surgery.

Post-operative measurement-

-On the day following surgery the following was assessed

1. Uncorrected and pinhole visual acuity was noted.
2. Detailed slit lamp examination was done
3. IOP was noted using noncontact tonometer

The patients were assessed on post-operative 1 week , 1 month and 2 months and during this period, tear film was quantified using

- Schirmer's test I
- Tear break up time
- Lissamine green staining of cornea and conjunctiva.

Statistical Analysis

The data obtained was tabulated on Microsoft Excel spreadsheet. The categorical data was expressed as ratios and percentages. The prevalence of dry eye in patients undergoing phacoemulsification surgery was expressed in percentages. The association of dry eye with sex and age was determined by Chi-square test. Continuous data was expressed as mean ± standard deviation (SD). Pearson's correlation coefficient was used to determine correlation between Schirmers test-I and TBUT with microscopic light exposure time. At 95% confidence interval (CI), a probability value ('p' value) of less than or equal to 0.050 was considered to be statistically significant.

RESULTS

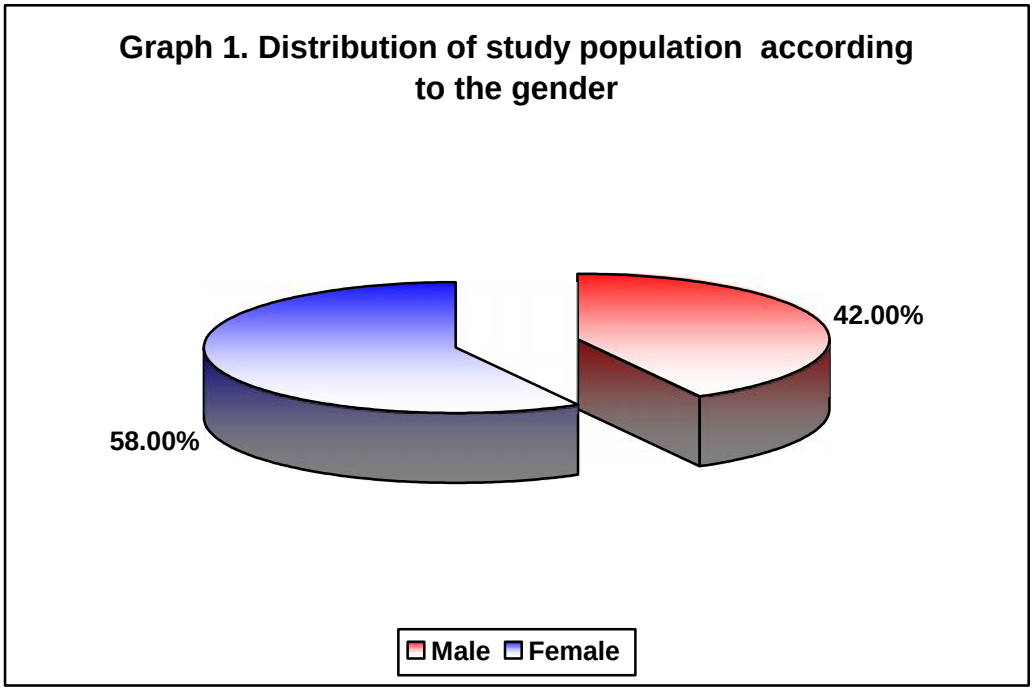
The present study was conducted in the Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during a study period of 1 year from 1st January 2017 – 31st December 2017 on 100 subjects undergoing phacoemulsification cataract surgery.

In our study, pre-operatively, the patients were assessed for dry eyes and tear film was quantified using Schirmers test I, Tear film break up time (TBUT) and Lissamine green staining of cornea and conjunctiva. Intra-operatively, the total duration of microscopic light exposure time during surgery was noted down in every case, which was measured from the start of microscopic light exposure on the ocular surface till the end of the surgery. The patients were then assessed on post-operative 1 week, 1 month and 2 months and during this period, again the tear film was quantified using Schirmer's test I, Tear break up time (TBUT), Lissamine green staining of cornea and conjunctiva. The data obtained was tabulated as below.

The data obtained was tabulated on Microsoft Excel spreadsheet. The categorical data was expressed as ratios and percentages. The prevalence of dry eye in patients undergoing phacoemulsification surgery was expressed in percentages. The association of dry eye with sex and age was determined by Chi-square test. Continuous data was expressed as mean ± standard deviation (SD). Pearson's correlation coefficient was used to determine correlation between Schirmers test-I and TBUT with microscopic light exposure time. At 95% confidence interval (CI), a probability value ('p' value) of less than or equal to 0.050 was considered to be statistically significant.

Table 1. Distribution of study population according to the gender

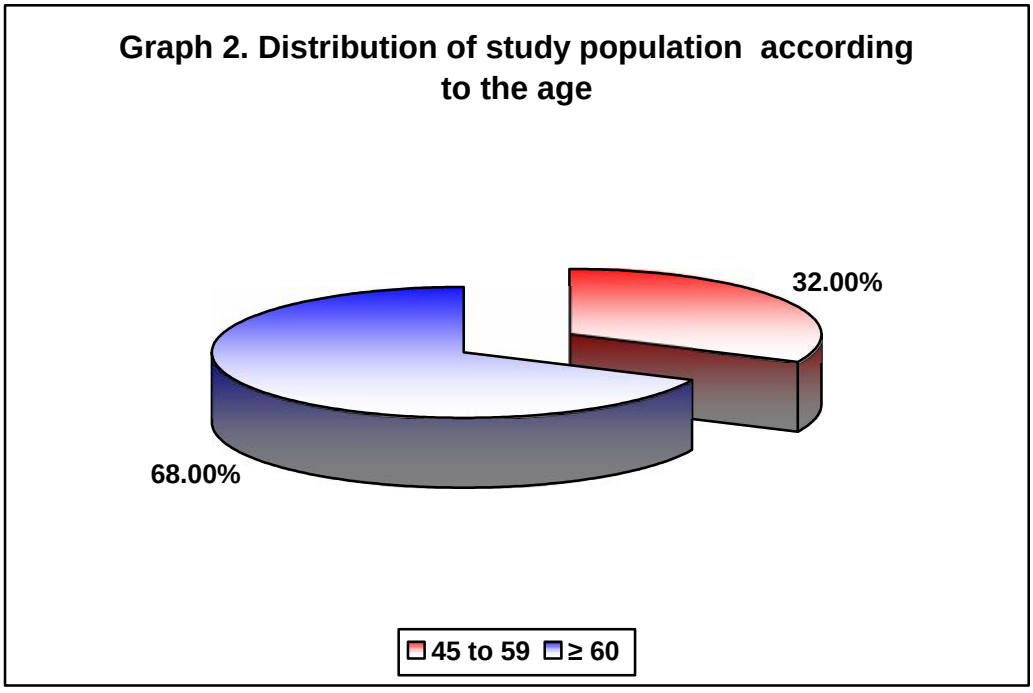
Gender	Distribution (n=100)	
	Number	**Percentage**
Male	42	42.00
Female	58	58.00
Total	**100**	**100.00**



In our study, out of 100 cases who underwent phacoemulsification, studied for dry eye evaluation, 58% were females and 42% were males.

Table 2. Distribution of study population according to the age

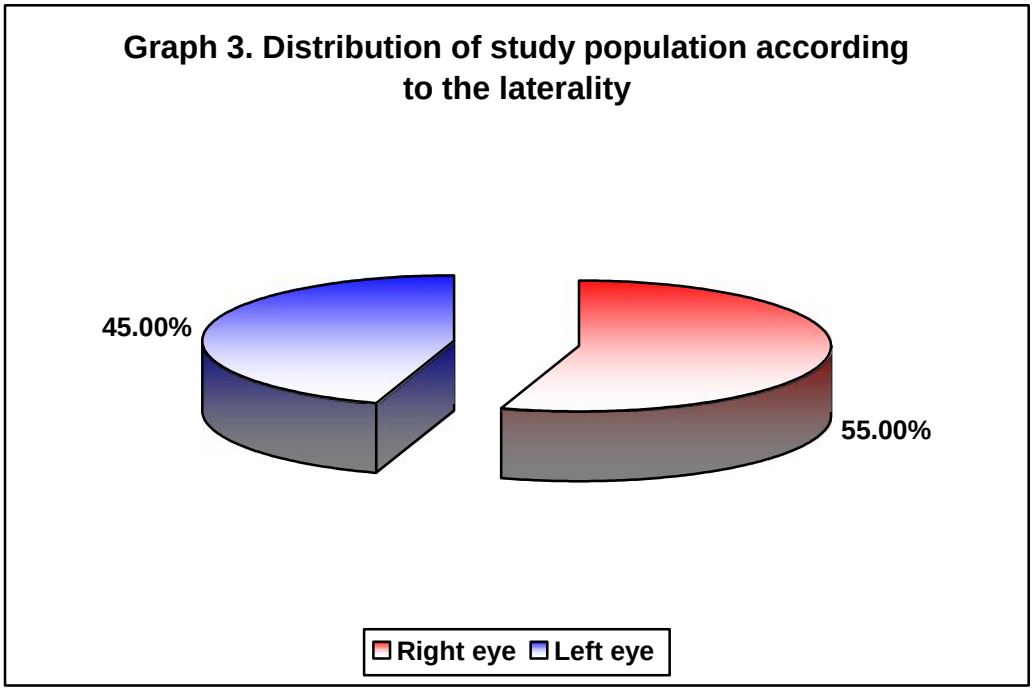
Age group (Years)	Distribution (n=100)	
	Number	**Percentage**
45 to 59	32	32.00
≥ 60	68	68.00
Total	**100**	**100.00**



In our study group, patients above the age of 45 years were taken for the study. 68% of the patients were in the age group of 45-59 years and 32% were 60 years and above.

Table 3. Distribution of study population according to the laterality

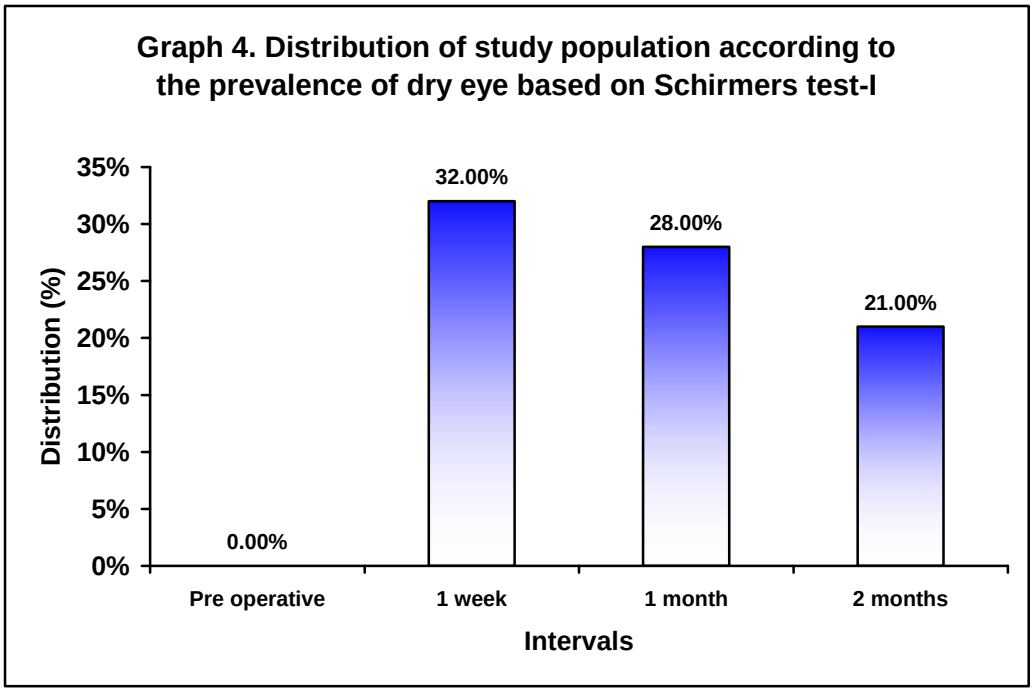
Laterality	Distribution (n=100)	
	Number	Percentage
Right eye	55	55.00
Left eye	45	45.00
Total	**100**	**100.00**



In our study, 45 cases (45%) underwent phacoemulsification surgery for Left eye cataract and 55 cases (55%) underwent phacoemulsification surgery for Right eye cataract.

Table 4. Distribution of study population according to prevalence of dry eye based on Schirmers test-I

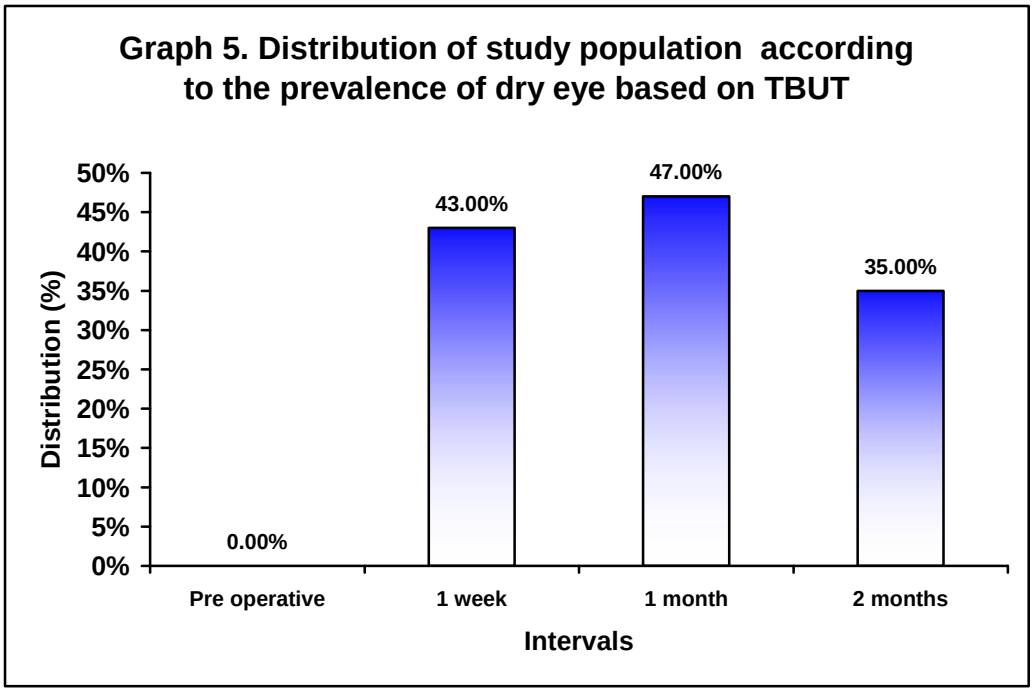
Intervals	Distribution (n=100)	
	Number	Percentage
Pre operative	0	0.00
1 week	32	32.00
1 month	28	28.00
2 months	21	21.00



On analysis of ST-1, 32% of the cases had dry eye at 1 week post phacoemulsification surgery, 28% at 1 month and 21% at 2 months.

Table 5. Distribution of study population according to prevalence of dry eye based on TBUT

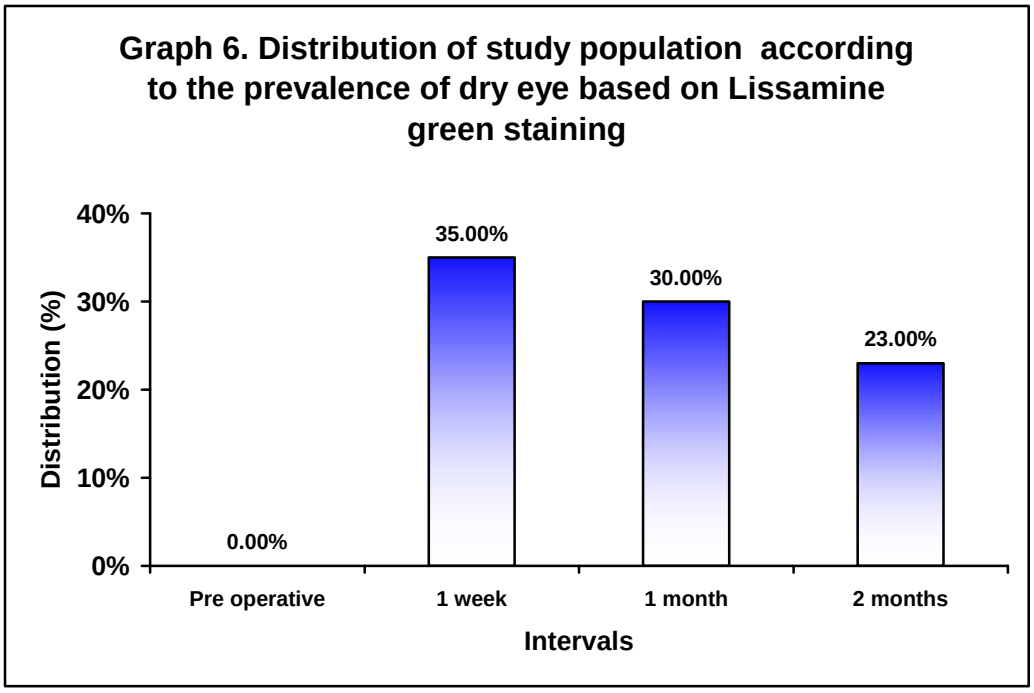
Intervals	Distribution (n=100)	
	Number	Percentage
Pre operative	0	0.00
1 week	43	43.00
1 month	47	47.00
2 months	35	35.00



On analysis of TBUT, 43% of the cases had dry eye at 1 week post phacoemulsification surgery, 47% at 1 month and 35% at 2 months.

Table 6. Distribution of study population according to prevalence of dry eye based on Lissamine green staining

Intervals	Distribution (n=100)	
	Number	Percentage
Pre operative	0	0.00
1 week	35	35.00
1 Month	30	30.00
2 Months	23	23.00

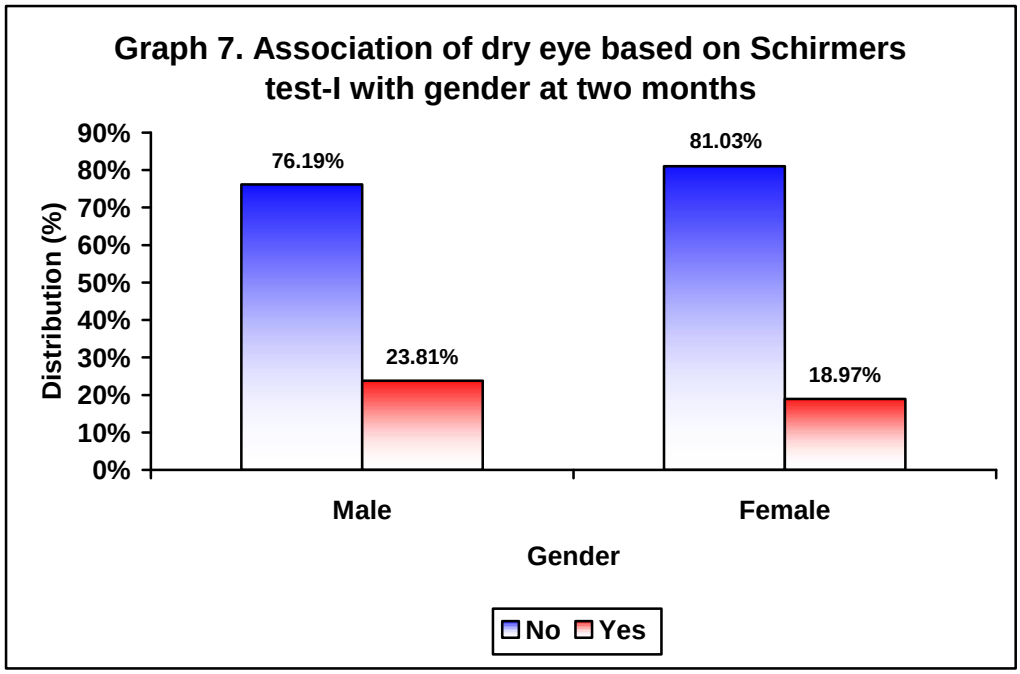


On analysis of LG staining , 35% of the cases had dry eye at 1 week post phacoemulsification surgery, 30% at 1 month and 23% at 2 months.

Table 7. Association of dry eye based on Schirmers test-I with gender at two months

Sex	Dry eye syndrome at two months				Total (n=100)	
	No		Yes			
	No.	%	No.	%	No.	%
Male	32	76.19	10	23.81	42	42.00
Female	47	81.03	11	18.97	58	58.00
Total	**79**	**79.00**	**21**	**21.00**	**100**	**100.00**

p = 0.623

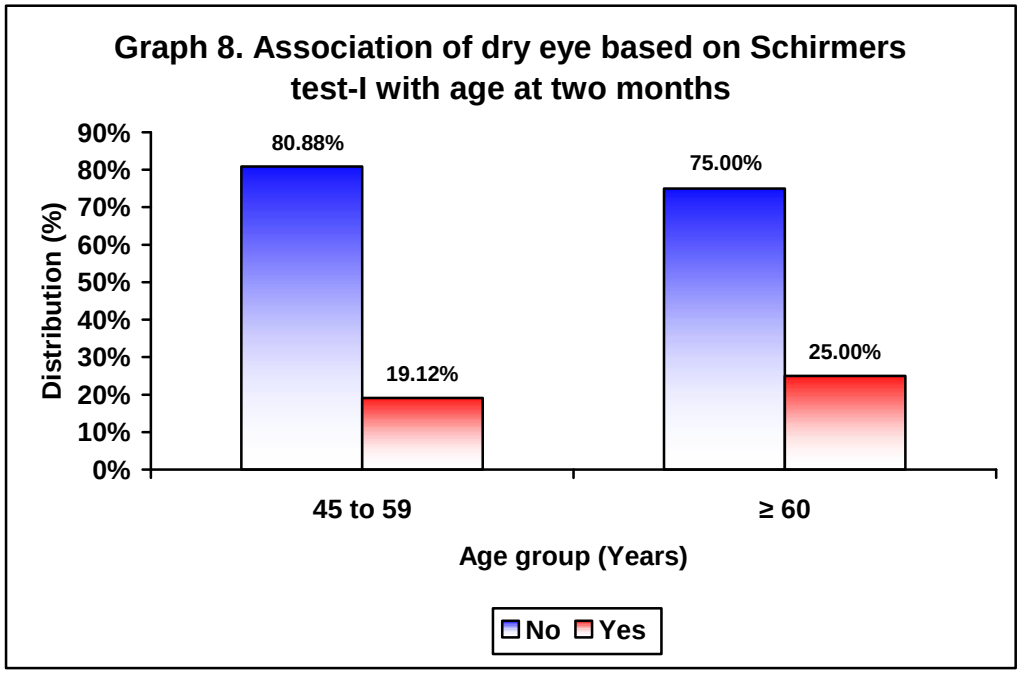


On analysis of Schirmers test-1 at 2 months post-op, dry eyes persisted in 10 males (23.81%) out of the total 42 males and in 11 females (18.97%) out of the total 58 females. So, the total number of patients who had dry eyes at 2 months postop were 21.

Table 8. Association of dry eye based on Schirmers test-I with age at two months

Age group (Years)	Dry eye at two months				Total (n=100)	
	No		Yes			
	No.	%	No.	%	No.	%
45 to 59	55	80.88	13	19.12	68	68.00
≥ 60	24	75.00	8	25.00	32	32.00
Total	**79**	**79.00**	**21**	**21.00**	**100**	**100.00**

p = 0.600

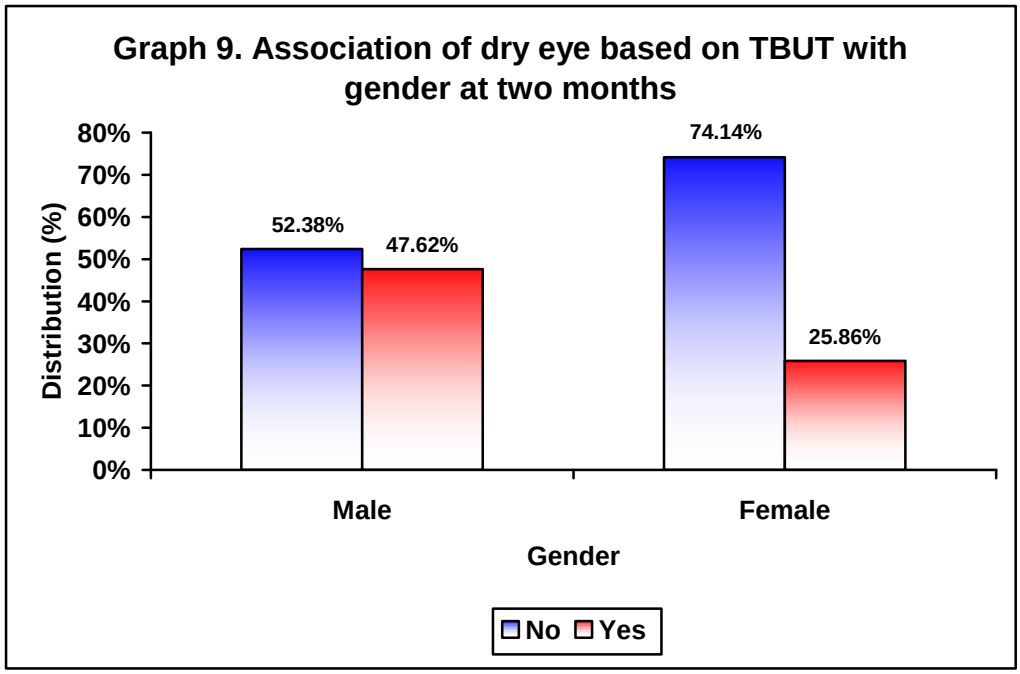


On analysis of Schirmers test-1 at 2 months post-op, in the age group of 45-59 years, dry eyes persisted in 13 cases (19.12%) out of the total 68 cases in that group and in the age group 60 years and above, 8 cases (25%) had dry eyes out of the total 32 cases in that group. So, the total number of patients who had dry eyes at 2 months postop were 21.

Table 9. Association of dry eye based on TBUT with gender at two months

Gender	Dry eye at two months				Total (n=100)	
	No		Yes			
	No.	%	No.	%	No.	%
Male	22	52.38	20	47.62	42	42.00
Female	43	74.14	15	25.86	58	58.00
Total	**65**	**65.00**	**35**	**35.00**	**100**	**100.00**

p = 0.034

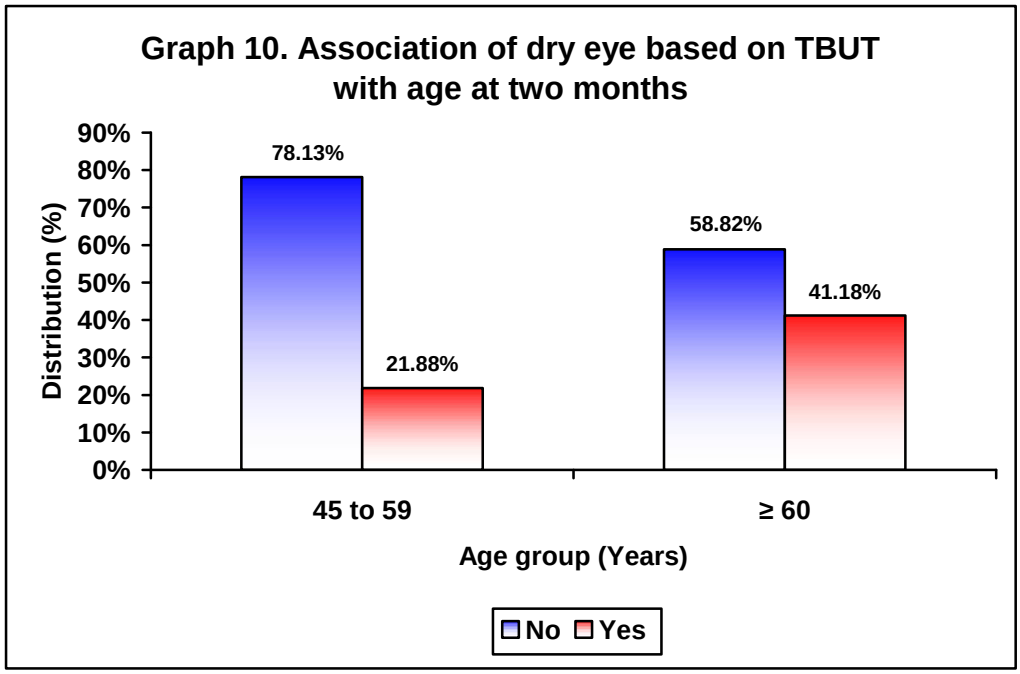


On analysis of TBUT at 2 months post-operatively, dry eyes persisted in 20 males (47.62%) out of the total 42 males and in 15 females (25.86%) out of the total 58 females. So, the total number of patients who had dry eyes at 2 months postop were 35.

Table 10. Association of dry eye based on TBUT with age at two months

Age group (Years)	Dry eye at two months				Total (n=100)	
	No		Yes			
	No.	%	No.	%	No.	%
45 to 59	25	78.13	7	21.88	32	32.00
≥ 60	40	58.82	28	41.18	68	68.00
Total	**65**	**65.00**	**35**	**35.00**	**100**	**100.00**

p = 0.074

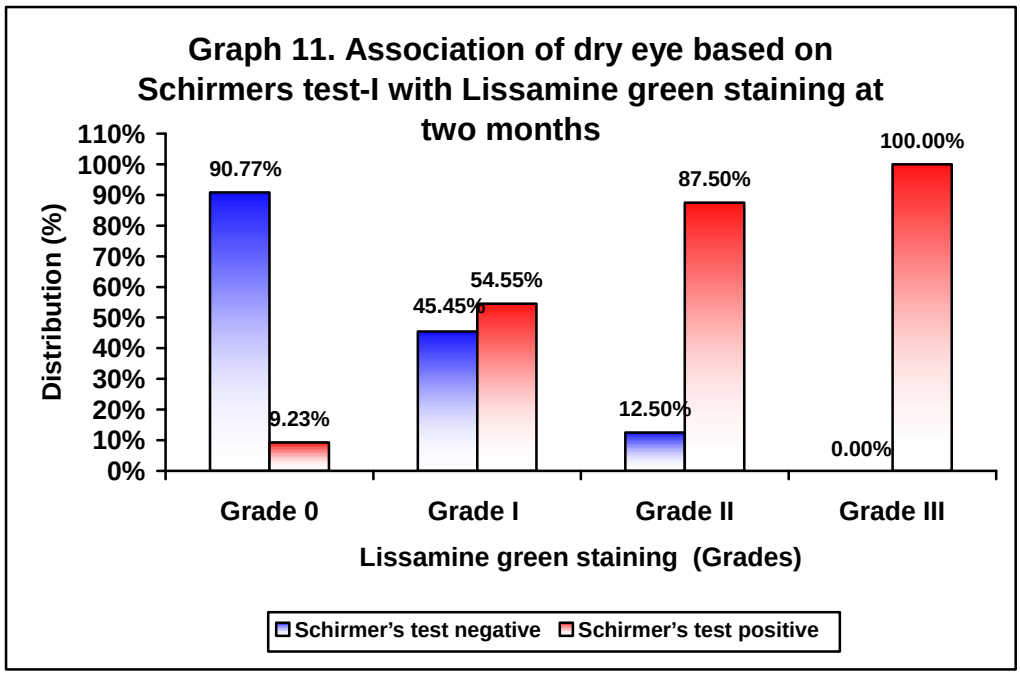


On analysis of TBUT at 2 months post-op, in the age group of 45-59 years, dry eyes persisted in 7 cases (21.88%) out of the total 32 cases (32%) in that group and in the age group 60 years and above, 28 cases (41.18%) had dry eyes out of the total 68 cases(68%) in that group. So, the total number of patients who had dry eyes at 2 months postop were 35.

Table 11. Association of dry eye based on Schirmers test-I with Lissamine green staining at two months

Lissamine green staining (Grades)	Schirmers test at two months				Total (n=100)	
	No		Yes			
	No	%	No	%	No	%
Grade 0	59	90.77	6	9.23	65	65.00
Grade I	5	45.45	6	54.55	11	11.00
Grade II	1	12.50	7	87.50	8	8.00
Grade III	0	0.00	2	100.00	2	2.00
Total	**65**	**75.58**	**21**	**24.42**	**86**	**100.00**

p<0.001

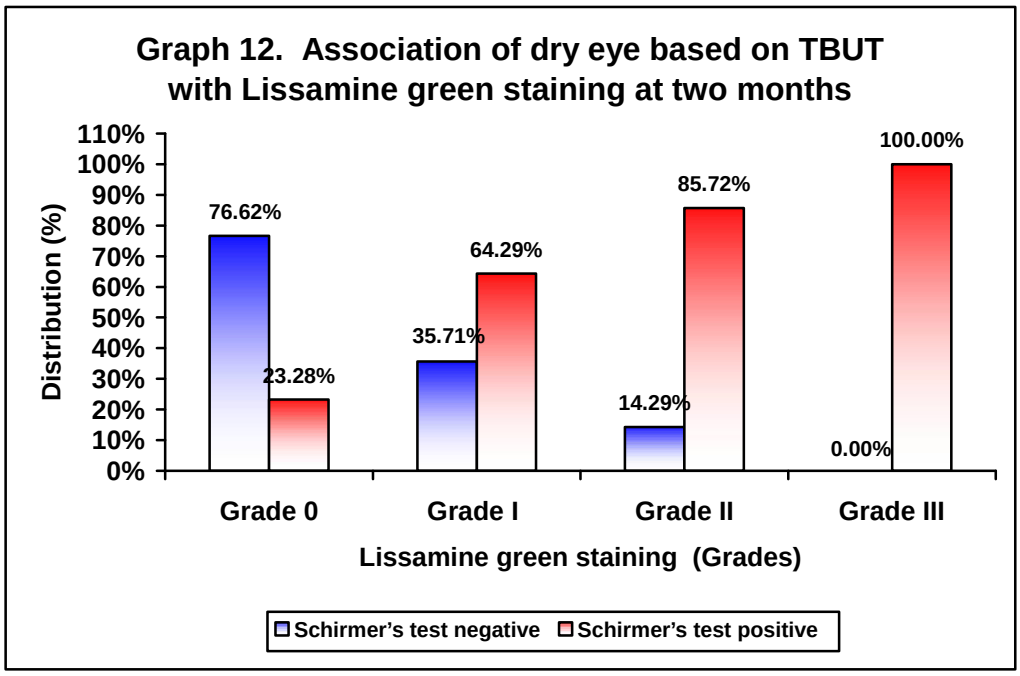


Out of the 65 cases who showed grade-0 LG staining, 6 cases(9.23%) had decreased ST-1 values, out of the 11 cases who showed grade-I LG staining, 6 cases(54.55%) had decreased ST-1 values, out of the 8 cases who showed grade-II LG staining, 7 cases(87.50%) had decreased ST-1 values, and the 2 cases who showed grade-III LG staining, showed decreased ST-1 values at 2 months.

Table 12. Association of dry eye based on TBUT with Lissamine green staining at two months

Lissamine green staining (Grades)	TBUT at two months				Total (n=100)	
	No		Yes			
	No	%	No	%	No	%
Grade 0	59	76.62	18	23.38	77	77.00
Grade I	5	35.71	9	64.29	14	14.00
Grade II	1	14.29	6	85.71	7	7.00
Grade III	0	0.00	2	100.00	2	2.00
Total	**65**	**65.00**	**35**	**35.00**	**100**	**100.00**

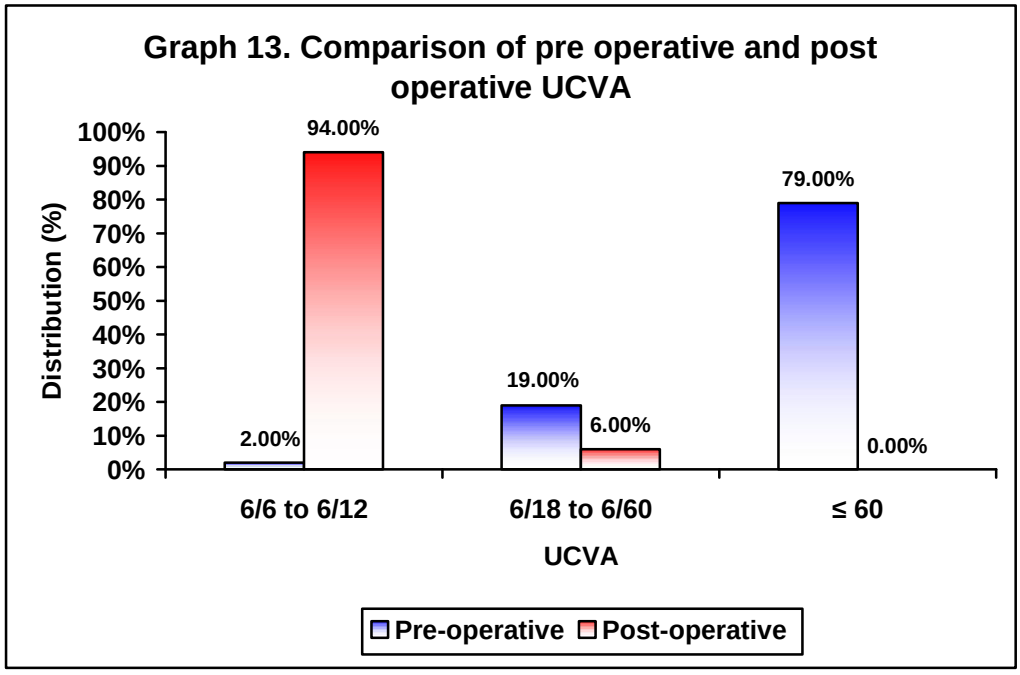
p<0.001



Out of the 77 cases who showed grade-0 LG staining, 18 cases(23.28%) had decreased TBUT test values, out of the 14 cases who showed grade-I LG staining, 9 cases(64.29%) had decreased TBUT test values, out of the 7 cases who showed grade-II LG staining, 6 cases(85.72%) had decreased TBUT test values, and the 2 cases who showed grade-III LG staining, showed decreased TBUT test values at 2 months.

Table 13. Comparison of pre operative and post operative UCVA

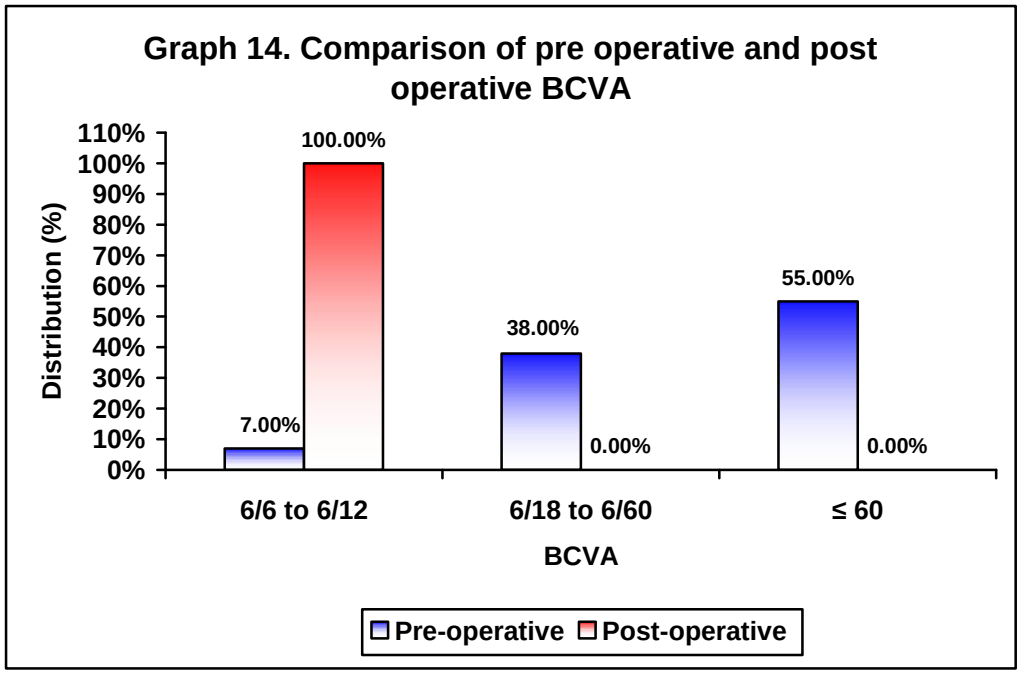
UCVA	Interval			
	Pre-operative		Post-operative	
	No.	%	No.	%
6/6 to 6/12	2	2.00	94	94.00
6/18 to 6/60	19	19.00	6	6.00
≤ 60	79	79.00	0	0.00
Total	**100**	**100.00**	**100**	**100.00**



In our study, pre-op, 2 patients (2%) had UCVA in the range between 6/6-6/12, 19 patients (19%) had UCVA in the range between 6/18-6/60 and the rest 79 patients(79%) had UCVA of less than 6/60. And post-op, 94 patients (94%) had UCVA in the range between 6/6-6/12, 6 patients(6%) had UCVA in the range between 6/18-6/60 and no patients less than 6/60.

Table 14. Comparison of pre operative and post operative BCVA

BCVA	Interval			
	Pre-operative		Post-operative	
	No.	%	No.	%
6/6 to 6/12	7	7.00	100	100.00
6/18 to 6/60	38	38.00	0	0.00
≤ 60	55	55.00	0	0.00
Total	**100**	**100.00**	**100**	**100.00**



In our study, pre-op, 7 patients(7%) had BCVA in the range between 6/6-6/12, 38 patients(38%) had BCVA in the range between 6/18-6/60 and 55 patients(55%) had BCVA of less than 6/60. And post-op all patients(100%) had BCVA in the range between 6/6-6/12.

Table 15. Descriptive data on Schirmers test-I, TBUT and Lissamine green staining for dry eye

Variables	Distribution (n=100)		Median	Range	
	Mean	SD		Minimum	Maximum
ST-I pre op (mm)	19.55	8.20	17.00	10.00	35.00
ST-I one week (mm)	15.70	9.44	13.50	2.00	35.00
ST-I one month (mm)	16.11	8.72	14.00	1.00	33.00
ST-I two months (mm)	16.71	8.05	15.00	2.00	34.00
TBUT pre op (seconds)	15.14	4.66	15.00	10.00	28.00
TBUT one week (seconds)	10.76	5.26	10.00	3.00	30.00
TBUT one month (seconds)	10.97	5.48	10.00	2.33	32.00
TBUT two months (seconds)	12.28	5.05	12.00	4.33	31.00
MLET (minutes)	22.72	5.75	22.77	13.34	34.90

The mean of the ST-1 values pre-op was 19.55 ± 8.20 with a minimum value of 10 and maximum of 35, at one week post-op, the mean was 15.10 ± 9.44 with minimum value of 2 and maximum of 35, at one month post-op, the mean was 16.11 ± 8.72 with minimum value of 1 and maximum of 33, at two months post-op, the mean was 16.71 ± 8.05) with minimum value of 2 and maximum of 34.

The mean of the TBUT test values was 15.14 ± 4.66 with a minimum value of 10 and maximum of 28, at one week post-op, the mean was 10.76 ± 5.26 with minimum value of 3 and maximum of 30, at one month post-op, the mean was 10.97 ± 5.48 with minimum value of 2.33 and maximum of 32, at two months post-op, the mean was 12.28 ± 5.05 with minimum value of 4.33 and maximum of 31.

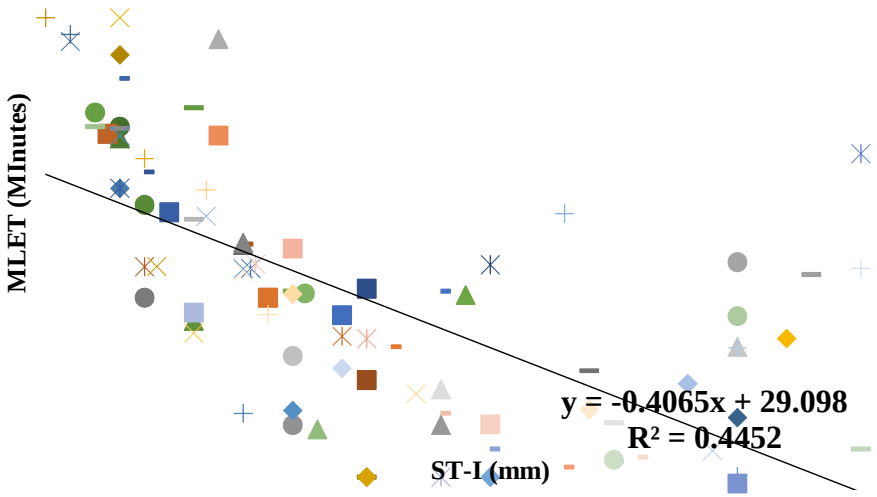
Intra-operatively, the mean of the MLET values was 22.72 ± 3.49 with a minimum value of 13.34 and maximum of 34.90.

Table 16. Correlation of MLET with ST-I and TBUT.

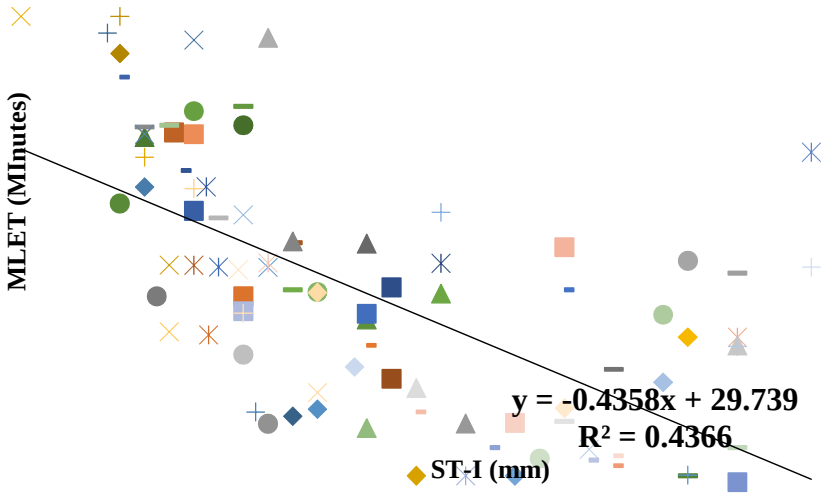
Tests and intervals		Distribution (n=100)	
	***r* value**	**R^2**	**p value**
MLET and ST-I at one week	-0.667	**0.445**	**<0.001**
MLET and ST-I at one month	-0.661	**0.436**	**<0.001**
MLET and ST-I at two months	-0.607	**0.368**	**<0.001**
MLET and TBUT at one week	-0.759	**0.575**	**<0.001**
MLET and TBUT at one month	-0.693	**0.479**	**<0.001**
MLET and TBUT at two months	-0.579	**0.335**	**<0.001**

This table shows negative correlation between MLET and the dry eye test values which is highly significant($p < 0.001$).

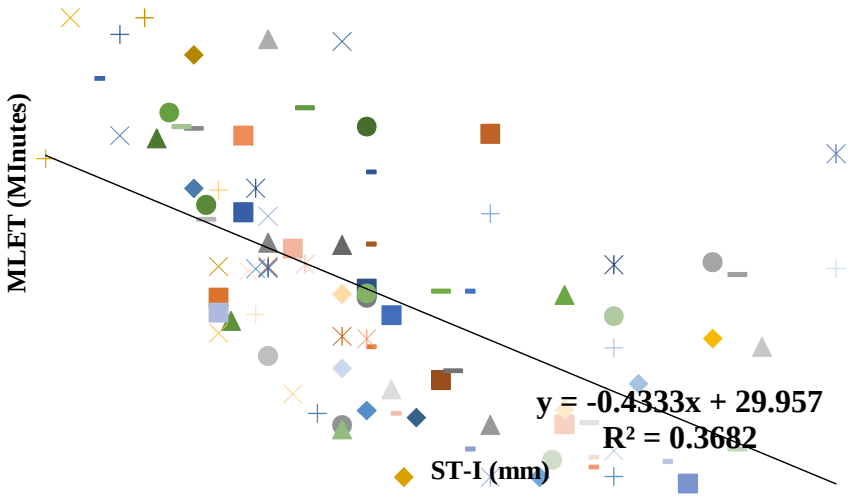
Graph 15. Correlation between MLET and ST-I at one week



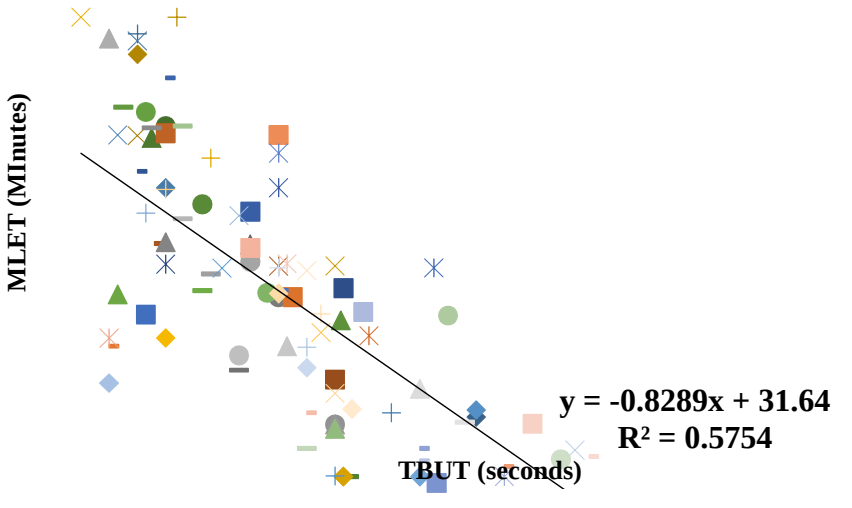
Graph 16. Correlation between MLET and ST-I at one month



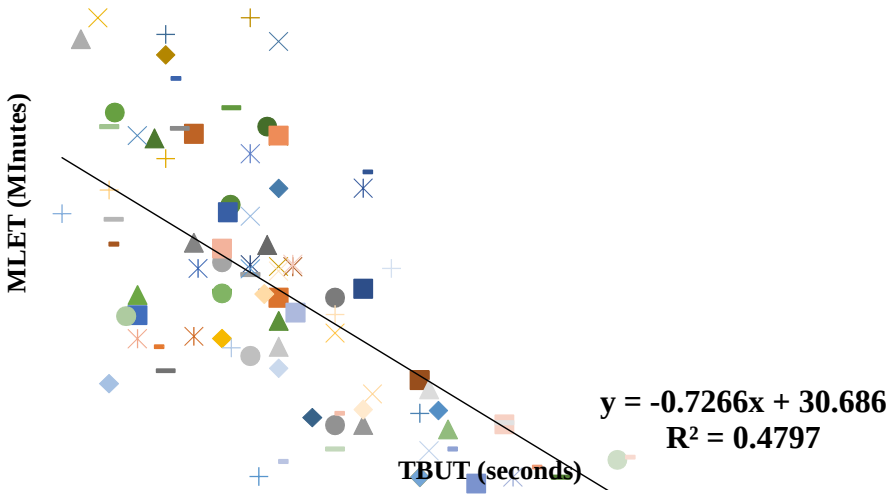
Graph 17. Correlation between MLET and ST-I at two months



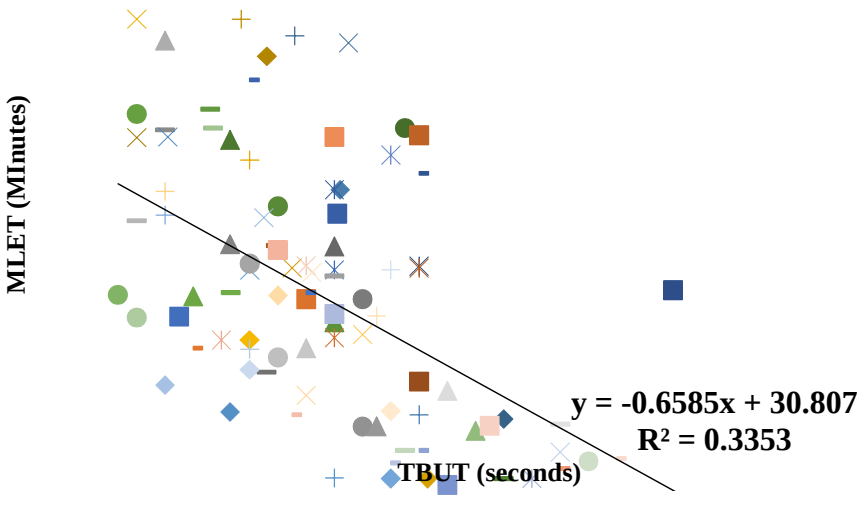
Graph 18. Correlation between MLET and TBUT at one week



Graph 19. Correlation between MLET and TBUT at one month



Graph 20. Correlation between MLET and TBUT at two months



DISCUSSION

The present study was conducted in the Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi during a study period of 1 year from 1st January 2017 – 31st December 2017 on 100 subjects undergoing phacoemulsification cataract surgery. In the developing world, cataract is currently the main cause of avoidable blindness accounting for majority of the blindness. Cataract surgery has evolved from couching which was, without doubt, one of the oldest surgical procedures to the present day phacoemulsification surgery. Cataract surgery has undergone significant developments over the past two decades. A high proportion of cataract patients who are candidates for cataract surgery have dry eye, furthermore, there are evidences suggesting aggravation or initiation of dry eye following cataract surgery[112,73]. Different surgical modalities like small incision cataract surgery and phacoemulsification with scleral or corneal incisions have varying effect on tear film stability which leads to disruption of corneal nerves[113]. A main cause of dissatisfaction in such cases has been shown to be eye fatigue and foreign body sensation due to dry eye syndrome.

A previously done study has compared the preoperative and postoperative changes in dry eye symptoms and/ or dry eye test values that worsened significantly after cataract surgery[76]. A difficulty in assessing dry eye is that there is no gold standard test[79].As a result various diagnostic tools with different sensitivities and specificities are used to diagnose dry eye. Cataract surgery can affect or interrupt the neurogenic response of the ocular surface and decrease tear secretion.

In our study, pre-operatively, the patients were assessed for dry eyes and tear film was quantified using Schirmers test I, Tear film break up time (TBUT) and Lissamine

green staining of cornea and conjunctiva. 45 cases (45%) underwent phacoemulsification surgery for Left eye cataract and 55 cases (55%) underwent phacoemulsification surgery for Right eye cataract. Intra-operatively, the total duration of microscopic light exposure time during surgery was noted down in every case, which was measured from the start of microscopic light exposure on the ocular surface till the end of the surgery. The patients were then assessed on post-operative 1 week, 1 month and 2 months and during this period, again the tear film was quantified using Schirmer's test I, Tear break up time (TBUT), Lissamine green staining of cornea and conjunctiva.

In our study, out of 100 cases who underwent phacoemulsification surgery, studied for dry eye evaluation, 42% were males and 58% were females, with a M:F ratio of 1:1.4. On analysis of Schirmers test-1 at 2 months post-operatively, dry eyes persisted in 10 males (23.81%) out of the total 42 males and in 11 females (18.97%) out of the total 58 females. So, the total number of patients who had dry eyes at 2 months postoperatively were 21. Also, on analysis of Tear film breakup time at 2 months post-operatively, dry eyes persisted in 20 males (47.62%) out of the total 42 males and in 15 females (25.86%) out of the total 58 females. So, the total number of patients who had dry eyes at 2 months postoperatively were 35. This study shows that the males have higher predisposition to developing dry eye disease based on TBUT and Schirmers test 1.

In a study by Shantanu Bhattacharjee et al, they found higher incidence of dry eye after cataract surgery in male group, 55% after 7 days and 71% at the end of 12th week which is similar to our observations[114].

However, most of the studies have demonstrated higher preponderance of dry eyes among female patients which is different from the observations made in our study.

In a study by Kamla Dodia et al, they reported more number of dry eye cases after cataract surgery in females[115]. Similarly, a study by Shankar S. Ganvit et al, showed that out of patients showing decrease TBUT and shorter Schirmer test-1 values after cataract surgery, 66.7% were females[116].

In a study by Miyake K et al, they found that females patients with dry eye symptoms before surgery had significant risk factors for postoperative dry eye[117]. They further analyzed the risk factors leading to postoperative dry eye and found that female patients and patients with shorter TBUT, with greater ocular surface staining score, or having the dry eye-related subjective symptom such as eye fatigue, eye pain, dull sensation, eye discomfort, dryness, and photophobia, all preoperatively, are at risk to encounter postoperative dry eye.

In a study by Divya N et al, out of the patients showing decrease TBUT and shorter schirmer 1 test values, 66.7% were females[118].

In many studies, a higher prevalence of the dry eye is reported in females than in males in the general population. This is may be because of an association of dry eyes in post-menopausal females which is well known.

In our study group, patients above the age of 45 years were taken for the study. 68% of the patients were in the age group of 45-59 years and 32% were 60 years and above.

However in a study conducted by Gayatri mohan et al, the maximum no of cases were in 61-70 age group, the minimum age was 50 years and the maximum age

was 82 years, which is different from our study as it has maximum number of cases in the older age group[119].

In our study, on analysis of Schirmers test-1 at 2 months post-operatively, in the age group of 45-59 years, dry eyes persisted in 13 cases (19.12%) out of the total 68 cases in that group and in the age group 60 years and above, 8 cases (25%) had dry eyes out of the total 32 cases in that group. So, the total number of patients who had dry eyes at 2 months postoperatively were 21 and also on analysis of Tear film breakup time at 2 months post-operatively, in the age group of 45-59 years, dry eyes persisted in 7 cases (21.88%) out of the total 32 cases (32%) in that group and in the age group 60 years and above, 28 cases (41.18%) had dry eyes out of the total 68 cases (68%) in that group. So, the total number of patients who had dry eyes at 2 months postoperatively were 35. This study shows that the old age has higher predisposition to developing dry eye disease based on TBUT and Schirmers test 1.

Similarly in a study by Gupta M et al dry eye was caused in all age groups, but was more in the older age group and stayed for a longer time. Only 38% in 45-55 years age group and 50% in 56-65 years age group at 12 weeks had dry eye whereas 66-75 years age group showed the same course except at 12 weeks and the tests values were below the baseline in all cases and had dry eye. The disparity in recovery in different age groups may be because of more nuclear sclerosis in older age group, more Phaco-time and more exposure to microscopic-light[120].

In a study conducted by Kamla Dodia et al they reported mean age of 60.03 years with more number of cases which were reported in higher age group (>65 years). With increasing age restoration of tear film is delayed[115].

A study by Shantanu Bhattacharjee et al demonstrated the delayed restoration of tear film with increasing age i.e. 57% in the age group of 66 – 85 yrs. as compared to 43% in the age group of 45 – 65 yrs. at the end of 12th week[114]. They concluded that dry eye risk increases with age[114].

In our study, on analysis of Schirmers test-1 , 32% of the cases had dry eye at 1 week post phacoemulsification surgery, 28% at 1 month and 21% at 2 months. Pre-operatively, the mean of the schirmers test values was 19.55 $\pm$ 8.20 which reduced to 15.70 ± 9.44 at one week post-op which eventually stabilized to 16.71 ± 8.05 at 2 months follow-up.

On analysis of Tear film breakup time, 43% of the cases had dry eye at 1 week post phacoemulsification surgery, 47% at 1 month and 35% at 2 months. Pre-operatively, the mean of the TBUT test values was 15.14 ± 4.66 which reduced to 10.76 ± 5.26 at one week post-op, which stabilized till one month follow-up with a mean of 10.97 ± 5.48 and eventually increasing slowly at two months follow-up when the mean was 12.28 ± 5.05.

In a study by Gupta M et al all age groups showed TBUT and Schirmer test values in post-operative period lowest at 1st week which gradually improved at 4th week and came near to baseline by 12th week. The gradual recovery of the Schirmer values over time was postulated to be due to the recovery of the sensitivity, thus enhancing the feedback neural loop of the cornea and lacrimal gland. Although the baseline values were still not reached at 3 months after surgery[120].

In a study conducted by P. K. Sahu et al**,** they found that deterioration in corneal sensitivity and tear physiology is seen immediately after phacoemulsification, and the tear functions recover within 1 month[121].

In a study by Peter Mark G et al, patients without dry eye disease, who underwent clear cornea phacoemulsification, did not develop dry eye disease after the surgery. Temporary reduction in physiologic tear levels seen one week post-surgery gradually returned to near-normal baseline levels by the third postoperative month[122]. In a study conducted by Ngamjit Kasetsuwan et al, the incidence of dry eye after phacoemulsification was 9.8% and the symptoms and signs of dry eye occurred as early as seven days post-phacoemulsification and the severity pattern improved over time[123].

In a study conducted by Sankar S. Ganvit et al, 3 months after cataract surgery 14% of patients showed dry eye symptoms,12% showed decrease in TBUT,11% had lower value of schirmer1 and 8% had lower value of schirmer with anaesthesia. They observed reduced schirmer 1 in 18%, TBUT in 30% and dry eye symptoms in 26%, on 30th post-operative day[116].

In a study by Munish Dhawan et al, the incidence of dry eye after phacoemulsification was 11% and symptoms and signs of dry eye occurred as early as seven days post-phacoemulsification and the severity pattern improved over time[124].

In a study by Shantanu Bhattacharjee et al, they reported dry eye in 84% patients after 7 days and only in 14% patients after 12 weeks[114].

In a study by Kyung eun han et al, at 1 month and 3 months post-op, they observed statistically significant decrease in TBUT but no change in Schirmers test in patients after cataract surgery[125].

In a study conducted by Mohana Sinha et al, there was a significant decrease in TMH, TBUT and ST-I values at 1 week, 1 month and 3 months. The mean values

of TMH, ST-1 and TBUT were below normal at 1 week, 1 month and 3 months follow-up, the lowest value was recorded at 1 month[126].

In our study, on analysis of Lissamine green staining , 35% of the cases had dry eye at 1 week post phacoemulsification surgery, 30% at 1 month and 23% at 2 months. Similarly, in a study conducted by Gupta M et al, they concluded that in all age groups Lissamine conjunctival staining in post-operative period was lowest at 1st week which gradually improved at 4th week and came near to baseline by 12th week. Although the baseline values were still not reached at 3 months after surgery[120].

In a study by Ngamjit Kasetsuwan et al, the severity peaked in day 7 and symptoms and signs improved over time. The abnormal ocular surface staining showed typical inter-palpebral staining pattern caused by dryness. The high incidence of abnormal Oxford Scheme grading after phacoemulsification may be due to neurogenic inflammation[123].

In a study by Miyake K et al, at 4 weeks after surgery, fluorescein staining score increased significantly and it was confirmed that cataract surgery increased the fluorescein ocular surface staining score[117].In this study on analysis of association between Schirmers test-1 and lissamine green staining at 2 months postoperatively, out of the 65 cases who showed grade-0 lissamine green staining, 6 cases(9.23%) had decreased Schirmers test values, out of the 11 cases who showed grade-I lissamine green staining, 6 cases(54.55%) had decreased Schirmers test values, out of the 8 cases who showed grade-II lissamine green staining, 7 cases(87.50%) had decreased Schirmers test values, and the 2 cases who showed grade-III lissamine green staining, showed decreased Schirmers test values at 2 months.

In this study on analysis of association between Tear film breakup time and lissamine green staining at 2 months postoperatively, out of the 77 cases who showed grade-0 lissamine green staining, 18 cases(23.28%) had decreased TBUT test values, out of the 14 cases who showed grade-I lissamine green staining, 9 cases(64.29%) had decreased TBUT test values, out of the 7 cases who showed grade-II lissamine green staining, 6 cases(85.72%) had decreased TBUT test values, and the 2 cases who showed grade-III lissamine green staining, showed decreased TBUT test values at 2 months.

In a study conducted by Mohana Sinha et al, majority 89% of the patients had no fluorescein staining at the onset of the study, the staining reached the peak at 1 month (32%) and gradually decreased by the end of 3 months (15%)[126]. At the end of the study 14.4% of the patients had moderate grade stain while the rest had no stain. The fluorescein staining of the cornea was of mild to moderate in 75% of the cases while 13.04% had severe grade at the end of 3 months[126].

In our study, pre-operatively, 2 patients (2%) had UCVA in the range between 6/6-6/12, 19 patients (19%) had UCVA in the range between 6/18-6/60 and the rest 79 patients (79%) had UCVA of less than 6/60. And post-operatively, 94 patients (94%) had UCVA in the range between 6/6-6/12, 6 patients (6%) had UCVA in the range between 6/18-6/60 and no patients less than 6/60.

In our study, pre-operatively, 7 patients (7%) had BCVA in the range between 6/6-6/12, 38 patients (38%) had BCVA in the range between 6/18-6/60 and 55 patients (55%) had BCVA of less than 6/60. And post-operatively all patients (100%) had BCVA in the range between 6/6-6/12.

In a study conducted by Abhinav Khadke et al, a statistically significant association was noted between dry eye and diminution of vision (p=0.001). Visual acuity for each patient at post-op 3 months with snellen chart showed that maximum patients had 6/9 vision (58.7%) followed by 6/12 vision in 138 patients.(35.4%)[127].

In our study, the mean of the MLET values was 22.72+/-3.49 with a minimum value of 13.34 and maximum of 34.90. This study shows a negative correlation between MLET and the dry eye test values which is highly significant($p < 0.001$).In a study conducted by P. K. Sahu et al, the decline of dry eye parameters after cataract surgery when compared to preoperative values was observed to be directly related to an increase in operating microscope light exposure time and a negative correlation was noted between microscope light exposure time and dry eye test values[121]

According to a study on Dry Eye After Cataract Surgery in Korean Journal of Ophthalmology, long microscopic light exposure times can have an adverse effect on dry eye test values[76]. Aside from retinal phototoxicity, microscopic light exposure may aggravate dry eye symptoms and signs in both the dry eye and non-dry eye group[76]. In a study by Gupta M et al, one of the risk factors for the disparity in recovery in different age groups was more exposure to microscopic-light[120].

CONCLUSION

We conclude from this study that Phacoemulsification surgery is indeed capable of inducing dry eye. The values were worse in the early post-operative period which gradually increased over 2 months. Therefore, prior to cataract surgery, patients must be informed about the possible increase in dry eye, however the condition might remain the same in some cases. Intra-operative exposure to microscopic light should be minimized by appropriate use of filters and shortening the exposure time.

Although we noticed an improvement in the tear film status, 2 months after surgery, however, a longer follow-up period and a larger sample size is required for conclusive evidence.

SUMMARY

Dry eye is a frequently encountered ocular problem in our tropical climate. Dry eye disease produces discomfort and reduced vision due to tear film instability. It is per se not a disease entity, but a symptom complex occurring as a sequelae to deficiency or abnormalities of tear film, exposing the corneal and conjunctival epithelium to evaporation. It is one of the most important factors influencing the quality of life in elderly population.

Cataract is an opacity of the natural crystalline lens. It is currently the main cause of avoidable blindness especially in the developing world accounting for about three quarters of blindness. Cataract surgery has evolved from couching which was, without doubt, one of the oldest surgical procedures to the present day phacoemulsification surgery. A high proportion of cataract patients who are candidates for cataract surgery have dry eye and also there is aggravation or initiation of dry eye following cataract surgery. A main cause of dissatisfaction in such cases has been shown to be eye fatigue and foreign body sensation due to dry eye syndrome. Different surgical modalities like small incision cataract surgery and phacoemulsification with scleral or corneal incisions have varying effect on tear film stability which leads to disruption of corneal nerves.

We enrolled 100 cataract cases who were non-dry eye for phacoemulsification surgery. The subjects were assessed for dry eyes and tear film was quantified using Schirmers test I, Tear film break up time (TBUT) and Lissamine green staining of cornea and conjunctiva pre-operatively. Intra-operatively, the total duration of microscopic light exposure time during surgery was noted down in every case, which was measured from the start of microscopic light exposure on the ocular surface till the end of the surgery. The patients were then assessed on post-operative 1 week , 1

month and 2 months and during this period, again the tear film was quantified using Schirmer's test I, Tear film break up time (TBUT), Lissamine green staining of cornea and conjunctiva.

In our study we found that the males and old age have higher predisposition to developing dry eye disease based on TBUT and Schirmers test 1. On analysis of Schirmers test-1, we noted that 32% of the cases had dry eye at 1 week post phacoemulsification surgery, 28% at 1 month and 21% at 2 months and on analysis of Tear film breakup time,43% of the cases had dry eye at 1 week post phacoemulsification surgery, 47% at 1 month and 35% at 2 months. On analysis of Lissamine green staining, 35% of the cases had dry eye at 1 week post phacoemulsification surgery, 30% at 1 month and 23% at 2 months. The dry eye test values were lowest in the early post-operative period which gradually improved at 2 months but not reaching the pre-operative status. On assessing the comparison of dry eye test values with MLET, we found a negative correlation between MLET and the dry eye test values which is highly significant($p < 0.001$).

Thus we conclude from this study that Phacoemulsification surgery is indeed capable of inducing dry eye. The values were worse in the early post-operative period which gradually increased over 2 months. Therefore, prior to cataract surgery, patients must be informed about the possible increase in dry eye, , however the condition might remain the same in some cases. Intra-operative exposure to microscopic light should be minimized by appropriate use of filters and shortening the exposure time.

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INFORMED CONSENT

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

ID NO. ☐☐☐

Mr/Mrs/Ms_____

You are invited to participate in our research study titled **"**PREVALENCE OF DRY EYE FOLLOWING PHACOEMULSIFICATION SURGERY AND ITS RELATION TO INTRAOPERATIVE RISK FACTOR - A ONE YEAR CROSS SECTIONAL STUDY**"** Conducted by Dr._____, Post Graduate in M.S. Ophthalmology under the guidance of Dr. _____ , Professor, The Department of Ophthalmology, J.N. Medical College, Belagavi-590010.

Respected Sir/Madam we request you to enroll yourself to participate in our study as you are eligible for doing so. Your participation in the study is voluntary. Your decision whether or not to participate in the study will not affect your relationship with the hospital. If you decide to participate you are free to withdraw at any time.

Objective and Purpose of the of the study: - To study the prevalence of dry eye in patients undergoing phacoemulsification surgery and to analyze dry eye in relation to associated intra-operative risk factor.

Procedure Involved: - If you agree to enroll yourself in this study, you will be asked to give detailed history. Then you will be clinically examined in detail by slit-lamp examination, fundoscopy, tonometry for measurement of intraocular pressure. Syringing for patency of the lacrimal sac, keratometry and A scan ultrasonography

and investigations like blood pressure measurement, random blood sugar will be done. Then you will be undergoing Phacoemulsification surgery.

Risks and Benefits :- As such no major risks are involved, rare complications of surgery includes posterior capsular rent, vitreous loss, lens dislocation, expulsive choroidal hemorrhage for which all necessary precautions will be taken.

Your participation may benefit you and others suffering from same ailment in future, by helping us learn more about the disease process and better treatment modalities.

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

Costs for participating in this research: - There will not be any extra cost incurred by the participant. The participant will however have to pay for the investigations which are the part of the existing management protocol for this ailment. There is no commitment for any reimbursement or any other compensation for the participant.

Privacy and Confidentiality:- The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission.

Authorization to Publish Results: - When the results of the research are published or discussed, in a conference, no information will be displayed that would

disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

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

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

1) Chief Investigator, Dr. _____ , P.G, Department of Ophthalmology, JNMC, Belagavi. Contact No. _____

2) Dr. _____ , Professor, Guide, Department of Ophthalmology, JNMC, Belagavi. Contact No: _____.

If you need any further information regarding your rights as a study participant contact.

3) Dr. _____ ,CHAIRPERSON, Professor of Pathology and Chairman, Institutional Ethics Committee, JNMC, Belagavi.

Consent for participation in research trial

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

Subject Name: _____

Signature or the Left Thumb Print of Subject: _____

Witness Name: _____

Signature of Witness: _____

Investigators Name: _____

Signature of Investigator: _____

Date: _____

Place: _____

PROFORMA

PATIENTS ID NO: ☐☐☐

NAME: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

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AGE: ☐☐ Years

SEX: ☐ (1-Male; 2-Female)

ADDRESS: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

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CONTACT NUMBER:- ☐☐☐☐☐☐☐☐☐☐

OP NUMBER: ☐☐☐☐☐☐☐☐

IP NUMBER: ☐☐☐☐☐☐☐☐

DATE OF ADMISSION: ☐☐ / ☐☐ / ☐☐☐☐

DATE OF DISCHARGE: ☐☐ / ☐☐ / ☐☐☐☐

IS THE PATIENT ELIGIBLE FOR STUDY? (1-YES; 2-NO) ☐

HAS INFORMED CONSENT BEEN GIVEN? (1-YES; 2-NO) ☐

FINAL RESULT INFORMATION ☐

1- INELIGIBLE
2- ELIGIBLE- REFUSAL
3- ELIGIBLE- PARTICIPATING

SURGEON’S NAME: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

SURGEON’S SIGNATURE: ______________________

DATE: ☐☐ / ☐☐ / ☐☐☐☐

CHIEF COMPLAINTS:

DIMINUTION OF VISION RE ☐

Duration: _____ days/ months/years

LE ☐

Duration: _____ days/ months/years

HISTORY OF PRESENT ILLNESS:

1 .DIMINUTION OF VISION 1- Gradual; 2- Sudden ☐

1- Progressive; 2- Static ☐

1- Painless; 2- Painful ☐

1- For distance; 2- For near ☐

2. DIPLOPIA/POLYOPIA 1- Present; 2- Absent ☐

3. COLOURED HALOS 1- Present; 2- Absent ☐

4. BLACK SPOTS BEFORE THE EYES 1- Present; 2 - Absent ☐

5. WATERING 1- Present; 2 - Absent ☐

6. REDNESS 1- Present; 2 - Absent ☐

7. DISCHARGE 1- Present; 2 - Absent ☐

8. H/O WEARING GLASSES (1-Distance; 2-Near; 3-Both) ☐

Duration: ☐☐ months/years

PAST HISTORY:

TRAUMA TO THE EYE: 1- Present; 2- Absent ☐

OCULAR SURGERY: 1- Present; 2- Absent ☐

Type of surgery: _____

Duration: ☐☐ months/years

DIABETES: 1- Present 2- Absent ☐

Duration: ☐☐ months/years

HYPERTENSION: 1- Present 2- Absent ☐

Duration: ☐☐ months/years

ANY OTHER MEDICAL DISORDERS: _____

PERSONAL HISTORY:

SMOKING: 1- Present; 2- Absent ☐

Duration: ☐☐ months/years

ALCOHOLISM: 1- Present; 2- Absent ☐

Duration: ☐☐ months/years

ANY OTHER ADDICTIONS: _____ ☐

Duration: ☐☐ months/years

GENERAL PHYSICAL EXAMINATION:

General Appearance:

1- Well built, 2- Moderately built, 3- Poorly built, 4- emaciated ☐

Pallor: 1- Present 2- Absent ☐

If present 1- Mild 2- Moderate 3- Severe ☐

Pulse: ☐☐☐ / minute

BP:- ☐☐☐ / ☐☐☐ mm of hg

Temperature: ☐☐☐ degree Fahrenheit

Respiratory rate: ☐☐ / minute

SYSTEMIC EXAMINATION:

CVS: 1- Normal 2- Abnormal ☐
if 2, specify: _____

RS: 1- Normal 2- Abnormal ☐
if 2, specify: _____

CNS: 1- Normal 2- Abnormal ☐
if 2, specify: _____

Per Abdomen: 1- Normal 2- Abnormal ☐
if 2, specify: _____

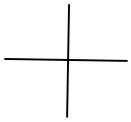
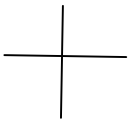
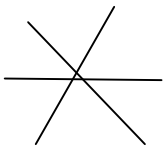
OCULAR EXAMINATION:

Head posture: 1- Erect, 2- Tilted ☐

Visual Axis: 1- Parallel, 2- Deviated ☐

Facial Symmetry: 1- Symmetrical, 2- Asymmetrical ☐

Extraocular movements:

RE-  LE-  Binocular :- 

(N- Normal, R- Restricted) ☐

1) Visual Acuity:

	RE	LE
DISTANT		
PINHOLE		
NEAR		
AIDED		

2. Adnexa (1- Normal; 2-Abnormal)	☐	☐
3. Sclera (1- Normal; 2- Congested)	☐	☐

4. Conjunctiva (1-normal; 2-conjunctival congestion; 3-ciliary congestion; 4-chemosis)	☐	☐
5. Cornea (1- normal; 2-opacity; 3-vascularisation)	☐	☐
6. Anterior chamber (1- normal depth; 2-shallow; 3-deep)	☐	☐
7. Iris (1-normal colour & pattern; 2-Abnormal)	☐	☐
8. Pupil: Size- ____ in mm Shape- 1- Round & Regular; 2-Abnormal Reaction: Direct (1. Present, 2. Absent) Indirect (1. Present, 2. Absent) Near reflex (1. Present, 2. Absent)	☐ ☐ ☐ ☐	☐ ☐ ☐ ☐
9. Lens Clarity- 1. Clear, 2. Opaque Cataract - (1) , PCIOL - (2) Cataract if present- 1.immature 2.mature 3. hyper mature	☐ ☐ ☐	☐ ☐ ☐

FUNDUS	RE	LE
GLOW		
MEDIA		
DISC		
C:D RATIO		
BLOODVESSELS		
BACKGROUND		
MACULA		

PRE OPERATIVE DRY EYE EVALUATION -

TEST OBSERVATION INFERENCE

SCHIRMER'S TEST 1

TEAR BREAKUP TIME

LISSAMINE GREEN STAINING

DIAGNOSIS:-

IMPRESSION:-

INVESTIGATIONS:

1. Ocular

A) IOP:

RE: _____ mm of hg
LE : _____ mm of hg

B) Blood sugar: _____mg%

C) Blood Pressure:_____mm of hg

D) TESTS FOR LACRIMAL PATENCY

E) KERATOMETRY

F) A-SCAN

INTRA-OPERATIVE EVALUATION - _____ EYE PHACOEMULSIFICATION

UNDER LOCAL ANAESTHESIA ON _____

MICROSCOPIC LIGHT EXPOSURE TIME - _____

POST OPERATIVE DRY EYE EVALUATION

TEST	1 WEEK	1 MONTH	2 MONTHS
SCHIRMER'S TEST			
TEAR BREAKUP TIME			

LISSAMINE GREEN

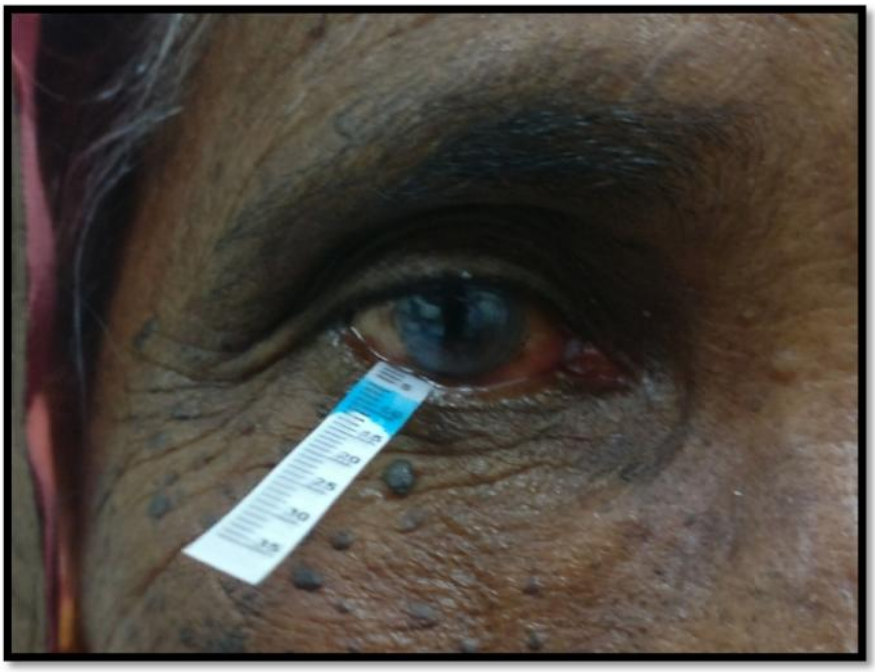
STAINING

INFERENCE

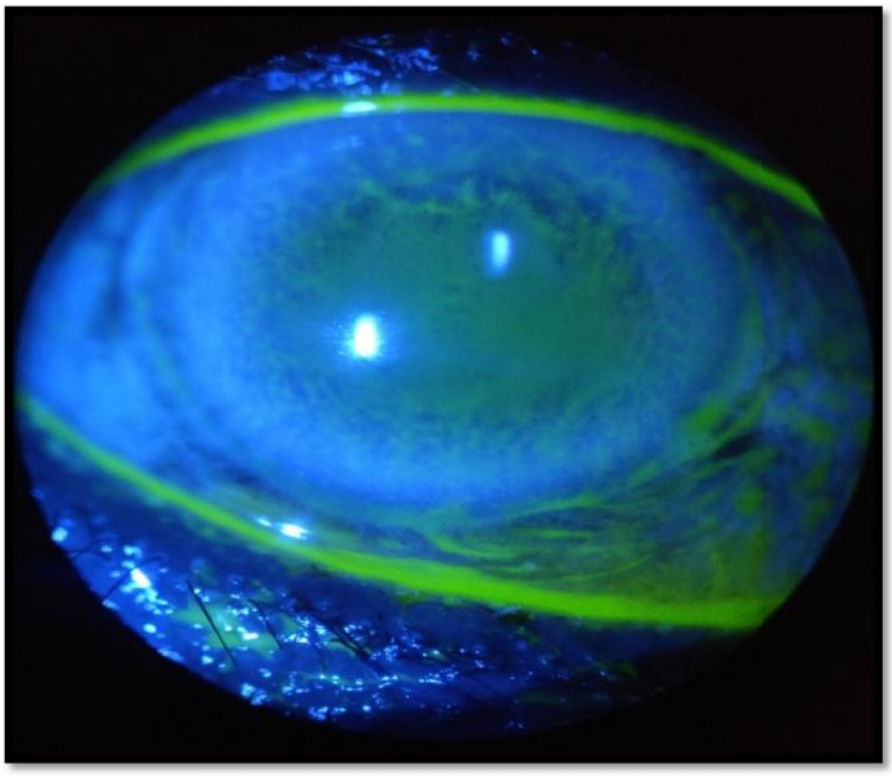
INFERENCE:

Photograph 1 - Slit lamp examination

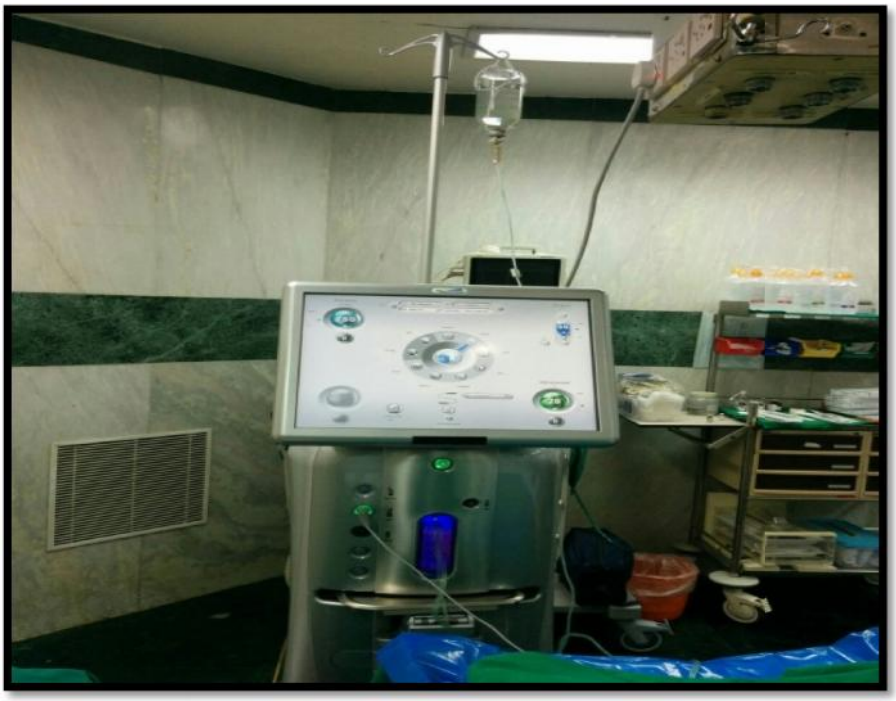
Photograph 2 - Performing Schirmers test - I



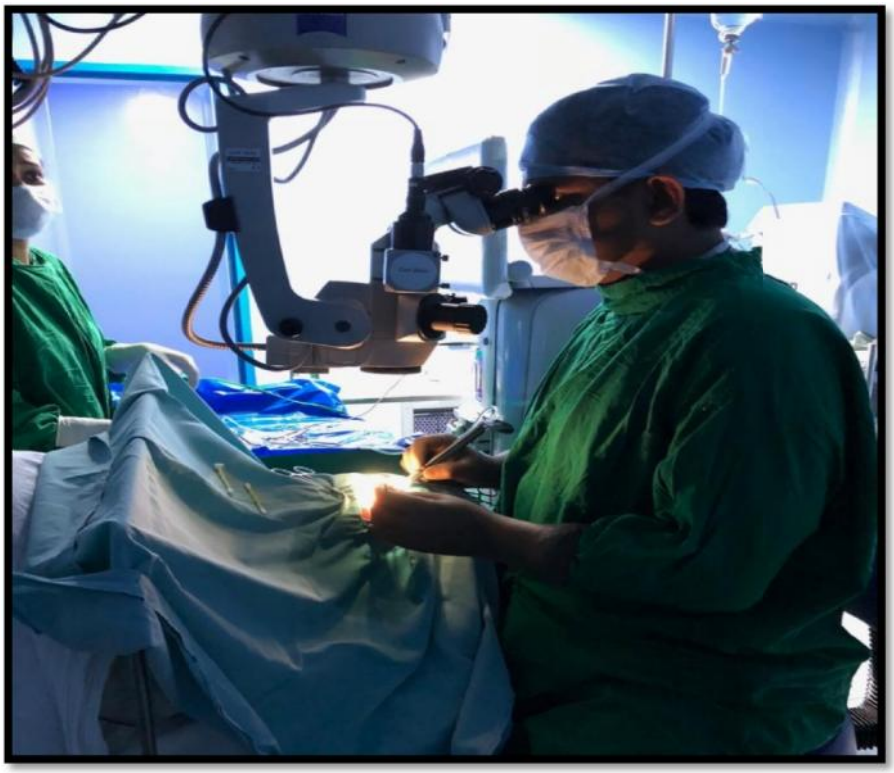
Photograph 3 – Schirmers test - I



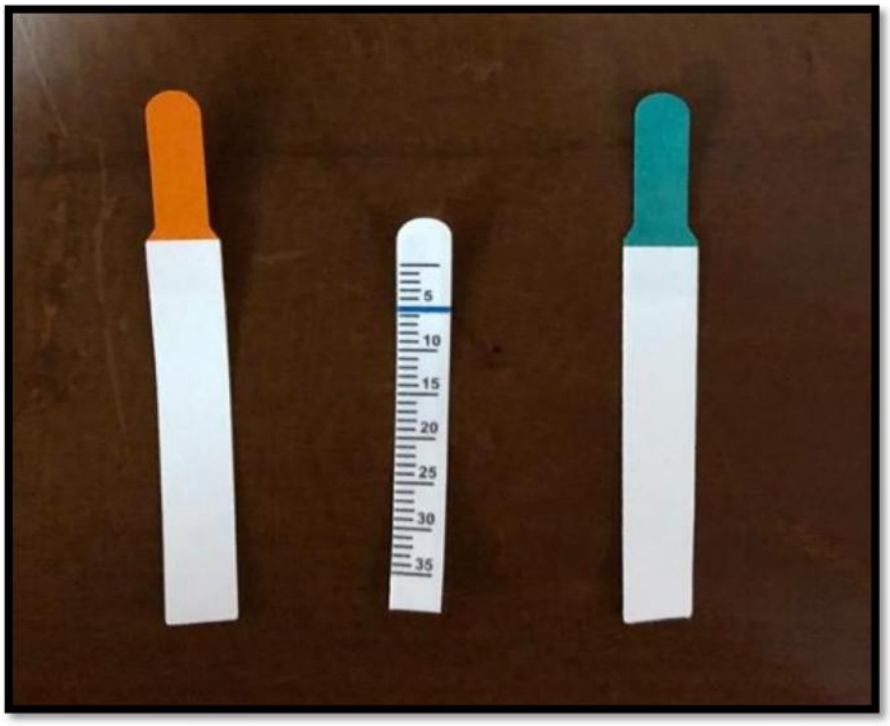
Photograph 4 – TBUT



Photograph 5 – Phacoemulsification machine with OT setup



Photograph 6 - Ongoing phacoemulsification surgery



Photograph 7 – Fluorescein, Schirmers and LG strips

KEY TO MASTER CHART

BCVA	–	Best corrected visual acuity
F	–	Female
L	–	Left eye
LG	-	Lissamine Green
M	–	Male
MLET	–	Microscopic Light Exposure Time
R	–	Right eye
ST-I	–	Schirmers test-I
TBUT	-	Tear film breakup time
UCVA	–	Uncorrected Visual Acuity
VA	–	Visual Acuity

Serial Number	In Patient Number	Age (Years)	Gender	Laterality	Pre-operative VA		ST - I (mm)				TBUT (seconds)				LG staining				MLET (Minutes)	VA at 2 months	
					UCVA	BCVA	Pre operative	1 week	1 Month	2 Months	Pre operative	1 week	1 Month	2 Months	Pre operative	1 week	1 Month	2 Months		UCVA	BCVA
1	781221	50	F	R	6/36(p)	6/24(p)	18	30	12	17	22	17	11.2	18	0	0	0	0	18.02	6/9	6/6(P)
2	781231	70	F	R	6/36(p)	6/24(p)	30	15	16	18	10	12	15	15	0	0	0	0	19.6	6/6(P)	6/6
3	790256	70	F	R	CF-1m	CF-1m	35	10	15	14	11.6	9	9.6	12	0	0	0	0	25.3	6/12	6/9
4	790158	60	F	R	CF-1m	CF-1m	13	5	6	10	10	5	10	5	0	3	2	1	29.9	6/9(P)	6/6(P)
5	797232	65	F	R	CF-1m	CF-1m	20	20	18	25	17	6	9	15	0	0	0	0	24.47	6/9	6/6
6	799340	66	F	L	6/36	6/18	11	5	10	15	12	6	9.6	14.5	0	3	2	1	30.3	6/9	6/6(P)
7	799880	68	M	R	CF-2m	6/36	13	3	4.5	5	10.6	5	6	10.6	0	4	3	2	34.2	6/12	6/6(P)
8	799893	80	F	L	CF-1/2m	CF-1/2m	26	10	12	15	13.6	5.6	4	9.6	0	0	0	0	25.34	6/12	6/9(P)
9	801313	68	M	L	CF-1m	6/60	23	24	25	18.5	21	8.6	6	9.6	0	0	0	1	20	6/9(P)	6/6(P)
10	802050	54	F	R	HMCF	HMCF	12	5	5	8	10.5	5	6	9.61	0	3	3	2	33.33	6/9	6/6
11	812569	68	M	R	CF-2.5m	CF-2.5m	31	15	16	15	24	12.3	13	24	0	0	0	0	23.46	6/9(P)	6/6
12	817345	50	M	R	HMCF	HMCF	12	5	6	6.5	12.3	5.5	5.6	8.3	0	3	2	2	29.8	6/12(P)	6/6(P)
13	817555	55	F	L	CF-2m	CF-2m	15	3	8	14	23	5	10	12.5	0	4	2	0	33.9	6/9(P)	6/6
14	818192	65	M	R	6/36(p)	6/24	12	6	8	11	18.3	10	10.5	15	0	2	1	0	24.39	6/9	6/6
15	822349	45	F	R	CF-CF	CF-CF	28	6	6.5	15	10.3	10	12	13	0	2	1	1	23.08	6/6(P)	6/6
16	823064	60	F	R	CF-1/2m	CF-1/2m	10	2	5	6	10	6.4	9	8.7	0	4	3	2	34.9	6/12	6/9
17	823256	55	F	R	CF-3m	CF-3m	12	6	7.5	15	10	5	13	15	0	3	2	0	28.39	6/9	6/6(P)
18	824563	70	F	L	6/60	6/36	28	15	28	28	22	12.5	20	18	0	0	0	0	15.5	6/18	6/9
19	825293	58	F	L	CF-1/2m	CF-1m	12	5	6	8	15	6	10	12.2	0	3	2	1	27.7	6/9	6/6
20	827057	70	F	L	CF-2m	CF-2m	12	4.5	7.5	20	14.1	6	7	15	0	3	2	0	30	6/12	6/9
21	834567	70	F	R	CF-2m	CF-2.5m	12	10	12	11	13	6	7	8.3	0	2	1	0	25.4	6/6(P)	6/6
22	835723	45	F	R	6/60	6/36(p)	10	6.5	7	9	15	12	10	10.5	0	1	1	1	24.4	6/12	6/9
23	836739	60	F	R	6/60	6/36	10	5	8.5	10.5	15.6	10	13	12	0	0	0	0	27.7	6/9(P)	6/6(P)
24	836888	50	F	L	CF-1m	CF-1m	12	6	5	8.5	15	7.3	8.3	10	0	2	2	1	27	6/9(P)	6/6(P)
25	842398	48	M	L	6/36	6/24(p)	15	10	10.5	13	16	14	15	15	0	1	0	0	18.2	6/9	6/6
26	842800	60	F	R	6/60	6/36(p)	32	32	33	31	23	22	23	20	0	0	0	0	13.39	6/9	6/6(P)
27	843362	65	F	L	6/18	6/12(p)	15	5	6	8	13	5.5	6.5	6	0	3	2	2	30.23	6/12	6/9
28	844450	65	F	L	6/24	6/24	23	15	17	16.5	21	12.3	15	15.3	0	0	0	0	15.5	6/18	6/9
29	846852	65	F	R	CF-1/2m	CF-1/2m	13	7	8	10	16	9	8.2	12.1	0	1	0	0	26.69	6/9	6/6(P)
30	847988	68	M	L	6/18	6/9(p)	15	8	15	9.5	17	12.2	10	12	0	1	0	0	22.1	6/9	6/6
31	857390	65	F	L	CF-3m	6/24	10	5	6	5	10	4.3	5	6.1	0	3	2	1	29.93	6/9	6/6(P)
32	857990	65	M	L	CF-1m	CF-1m	15	14	8.6	14	17	13.2	7	12	0	0	2	0	21.45	6/12	6/9(P)
33	865342	70	F	R	CF-2m	CF-2m	13	12	11	14	15	12	12	13	0	0	0	0	17.7	6/9	6/6(P)
34	865342	65	M	R	6/36	6/18	10	6	6	2	12	7.6	6	9	0	3	2	3	28.95	6/9(P)	6/6(P)
35	865440	60	M	R	6/60	6/18	10	5	5	4	12	6	6.2	9	0	3	3	2	32.34	6/18	6/12
36	866555	54	M	L	CF-2.5m	CF-2.5m	12	8	10	12.5	10	4.5	8.33	7.6	0	2	1	0	31.1	6/12	6/9
37	866560	69	M	R	CF-2m	CF-2m	12	12	13	15	16	17	15.66	8.3	0	0	0	0	18.32	6/12	6/9
38	867561	72	F	R	6/36	6/36	10	11	10	9	12	10.5	10	11	0	0	0	0	23.08	6/9(P)	6/6(P)
39	867321	71	M	R	CF-2m	CF-2m	20	18	19	20	15	12	13	13.5	0	0	0	0	17.7	6/9	6/6(P)
40	867322	52	M	L	6/24(p)	6/9(p)	10	5	1	3	10	3	3.6	5	0	3	4	3	34.9	6/9	6/6
41	867329	52	F	R	6/60	6/24(p)	12	10.5	9	11	14	15.5	7.15	12	0	1	0	0	24.32	6/12	6/6(P)
42	867523	65	M	L	6/60	6/18(p)	10	4	8	7	10.6	5.3	4.2	5	0	3	2	0	30.9	6/9(P)	6/9
43	867529	69	F	R	CF-3m	6/24(p)	29	30	28	25	12	12	9.3	12	0	0	0	0	15.53	6/12	6/9
44	867530	67	F	R	CF-3m	6/36	15	16	15	15	10	4	5.6	7	0	0	0	0	21.01	6/9	6/6(P)
45	867531	52	F	R	CF-2m	6/36(p)	30	33	30	30	10	7.6	9	12	0	0	0	0	24.06	6/9(P)	6/6(P)
46	867539	52	M	L	6/18	6/12(p)	29	32	28	29	10	6	8	9	0	0	0	0	21.36	6/9(P)	6/6(P)
47	867540	80	M	R	CF-4m	CF-4m	17	14	15	16	10	5.3	5	6.5	0	0	0	1	22.34	6/9	6/6
48	867554	45	M	R	CF-CF	CF-CF	19	19	18	23	10	4.3	5	7	0	0	0	1	23.2	6/9(P)	6/6
49	867560	65	F	R	CF-3m	CF-3m	12	10	11	10.5	11	8	9	9	0	0	0	0	24.3	6/12	6/9
50	867562	68	F	R	CF-2m	CF-2m	19	15.5	13.5	7	15	11.6	10.3	6	0	0	0	0	13.34	6/9(P)	6/6(P)
51	867566	69	F	L	CF-2m	CF-2m	30	30	28	29	10	9	8	9	0	0	0	0	24.58	6/9	6/6(P)
52	867570	48	M	L	CF-1m	CF-1m	28	24	25	22	18	15	16	17	0	0	0	0	14.49	6/12	6/9(P)
53	867583	65	F	L	CF-3m	CF-3m	22	18	23	19	12	10	9.3	11	0	0	0	0	23.36	6/9	6/6(P)
54	867590	61	F	R	6/12(p)	6/9(p)	14	12	12	18	12	7.3	8	8.33	0	0	0	0	23.36	6/9(P)	6/6(P)
55	867610	85	F	L	CF-3M	CF-3M	24	20	21	22	18	15	15	14	0	0	0	0	15.5	6/9	6/6
56	867639	56	M	L	6/60	6/36(P)	12	9	8	10	13	10	10	12	0	1	0	0	29.93	6/6(P)	6/6
57	867642	65	F	R	CF-1M	6/60	10	9	11	11	11	4	3	6	0	2	2	1	34	6/6(P)	6/6
58	867669	59	F	R	CF-3M	6/60	10	8	7	9	13.6	11.5	12	13	0	1	0	0	21.59	6/9(P)	6/6
59	867699	60	M	L	CF-3M	6/60	35	35	33	34	15	10	9	14	0	0	0	0	29.16	6/18	6/9
60	867714	60	F	L	CF-3M	6/36(P)	14	12.5	13	15	15	9.6	8	4.33	0	0	0	0	23.26	6/9	6/6(P)
61	867750	81	M	R	CF-2M	CF-2M	30	23	18	20	12.5	5.3	2.33	6	0	0	2	1	26.63	6/9(P)	6/6(P)
62	867751	45	M	L	6/12(p)	6/9	30	23	25	24	20	18	19	20	0	0	0	0	15.93	6/9(P)	6/6(P)
63	867753	62	M	L	PL+ PR ACC	PL+ PR ACC	14	8	9	8.5	10	6.6	4.16	5	0	3	2	1	26.39	6/12	6/9
64	867760	80	M	R	6/60	6/18(p)	35	30	32	33	22	15	18	17	0	0	0	0	14.4	6/9	6/6
65	867769	53	F	L	6/60	6/60	17	30	30	28	15	15.6	17	16	0	0	0	0	15.23	6/9	6/6(P)
66	867777	45	M	R	CF-CF	CF-CF	26	13	15	14	18	12	16	17	0	0	0	0	17.52	6/9(P)	6/9
67	867780	64	M	L	6/24	6/12(p)	12	8.5	10	11	10	8.6	9	9.5	0	1	1	0	26.52	6/9	6/6(P)
68	867789	60	M	L	6/36(P)	6/18	19	15	30	15	11	4	5	8	0	1	0	0	21.35	6/6(P)	6/6
69	867793	70	F	L	6/18(P)	6/18	14	12	10	11	12	8.6	9	10	0	0	0	0	20.62	6/9	6/6(P)

Serial Number	In Patient Number	Age (Years)	Gender	Laterality	Pre-operative VA		ST - I (mm)				TBUT (seconds)				LG staining				MLET (Minutes)	VA at 2 months	
					UCVA	BCVA	Pre operative	1 week	1 Month	2 Months	Pre operative	1 week	1 Month	2 Months	Pre operative	1 week	1 Month	2 Months		UCVA	BCVA
70	867812	74	M	R	6/24	6/18	10	8.5	8	9	11	6	4	6	0	0	1	1	27.63	6/9	6/6
71	867823	60	F	L	CF-1M	CF-1M	23	20	20	19	16	15	16	15	0	0	0	0	16.69	6/18	6/12
72	867834	60	M	R	CF-2M	CF-2M	10.5	4	7	7.5	11.6	6.6	4	7.7	0	4	3	2	30.3	6/12	6/9
73	867836	62	M	L	CF-2M	CF-2M	25	28	27	26	11	4	4	6	0	0	0	0	19.45	6/9	6/6(P)
74	867845	59	F	R	CF-3M	6/36(P)	12.5	12	23	12	10.2	9	8	10	0	0	0	0	25.15	6/9(P)	6/6(P)
75	867850	56	F	L	6/60	6/24(p)	32	30	30	31	15	10.3	10	11	0	0	0	0	21	6/9(P)	6/6(P)
76	867867	50	M	R	6/36(P)	6/24	10	17	13	12	11	12	13.33	11	0	0	0	0	19.02	6/9	6/6
77	867873	56	F	R	6/60	6/24	22	18	19	20	20	18	18.3	19	0	0	0	0	15.5	6/9(P)	6/6
78	867891	70	M	L	CF-3M	6/60	24	30	27	25	20	16	4.6	5	0	0	0	0	22.3	6/9	6/6
79	867899	50	M	R	6/60	6/24(P)	21	30	30	25	15	11	8.33	9	0	0	0	0	20.96	6/12(P)	6/6(P)
80	867901	53	M	L	CF-1M	CF-1M	19	18	17	16	15	11	12	10.5	0	0	0	0	18.2	6/9(P)	6/6(P)
81	867910	80	F	R	CF-1m	6/60	35	30	31	32	28	26	25	24	0	0	0	0	14.32	6/9(P)	6/6
82	867915	65	M	L	CF-2M	CF-3M	17	12	13	14	15	10	9.5	10	0	0	0	0	23.23	6/12(P)	6/9
83	867923	55	F	R	6/36(P)	6/36	17	8	10	9	15	13	10.6	12	0	1	0	0	22.45	6/9	6/6
84	867934	80	F	R	CF-3M	6/36(P)	29	24	25	25	24	20	22	22.5	0	0	0	0	14.2	6/12	6/9
85	867945	70	M	R	CF-3M	6/60	30	29	24	25	20	20.5	15.33	20	0	0	0	0	16.62	6/12	6/9(P)
86	867959	65	F	L	CF-5M	CF-5M	30	10.5	11	12.5	23	10.3	10.5	11	0	0	0	0	24.49	6/9(P)	6/6(P)
87	867963	80	F	L	6/60	6/60	35	28	28	30	28	30	32	31	0	0	0	0	14.65	6/9(P)	6/9
88	867980	59	F	L	CF-2M	CF-2M	12	11	10	10.5	15	11.5	12	13.5	0	0	0	0	22.37	6/9(P)	6/6(P)
89	867982	70	M	R	CF-2M	CF-3m	29	25	24	27	15	15	10	14	0	0	0	0	16.17	6/9	6/6(P)
90	867990	65	F	R	CF-3M	6/60	30	35	30	30	15	11	12	14.5	0	0	0	0	16.69	6/9(P)	6/6(P)
91	867993	63	F	L	CF-2M	CF-2M	15	14	14.5	14	12	11	10	9	0	0	0	0	20.1	6/9(P)	6/6
92	868003	49	M	R	6/60	6/60	23	20	21	23	20	19	18	17.5	0	0	0	0	17.73	6/9	6/6(P)
93	868012	64	F	R	CF-3M	CF-3M	18	18	17	16	17	15	15.33	16	0	0	0	0	19.21	6/9(P)	6/6(P)
94	868032	60	M	L	6/60	6/36(P)	12	10	9.8	10.2	12	11	10	11.2	0	1	1	0	24.2	6/9	6/6
95	868039	61	M	L	6/36	6/24	29	26	27	26	25	21	23	22	0	0	0	0	14.25	6/9(P)	6/9
96	868045	67	F	R	6/60	6/18(P)	29	25	22	22.5	25	20	22	21	0	0	0	0	16.23	6/12	6/6(P)
97	868053	80	M	R	6/60	6/36(P)	35	35	33	34	15	10	14	14	0	0	0	0	24.32	6/9	6/6
98	868055	55	F	L	6/60	6/36(P)	28	26	25	24	25	21	22.3	22	0	0	0	0	16.35	6/12	6/6(P)
99	868063	62	F	R	CF-1M	CF-1m	27	25	23	24	20	16.6	18	20	0	0	0	0	17.8	6/18	6/9
100	868072	76	F	L	CF-3M	CF-3m	26	24	23	23	18	12.6	13	14	0	0	0	0	18.35	6/12	6/9