"ASSESSMENT OF PREOPERATIVE AND INTRAOPERATIVE RISK FACTORS OF POSTERIOR CAPSULAR OPACIFICATION"

By
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Dissertation Submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

TAMAKA, KOLAR

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY
IN
OPHTHALMOLOGY

Under the Guidance of

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ABSTRACT

Background and Objective

Methods

Results

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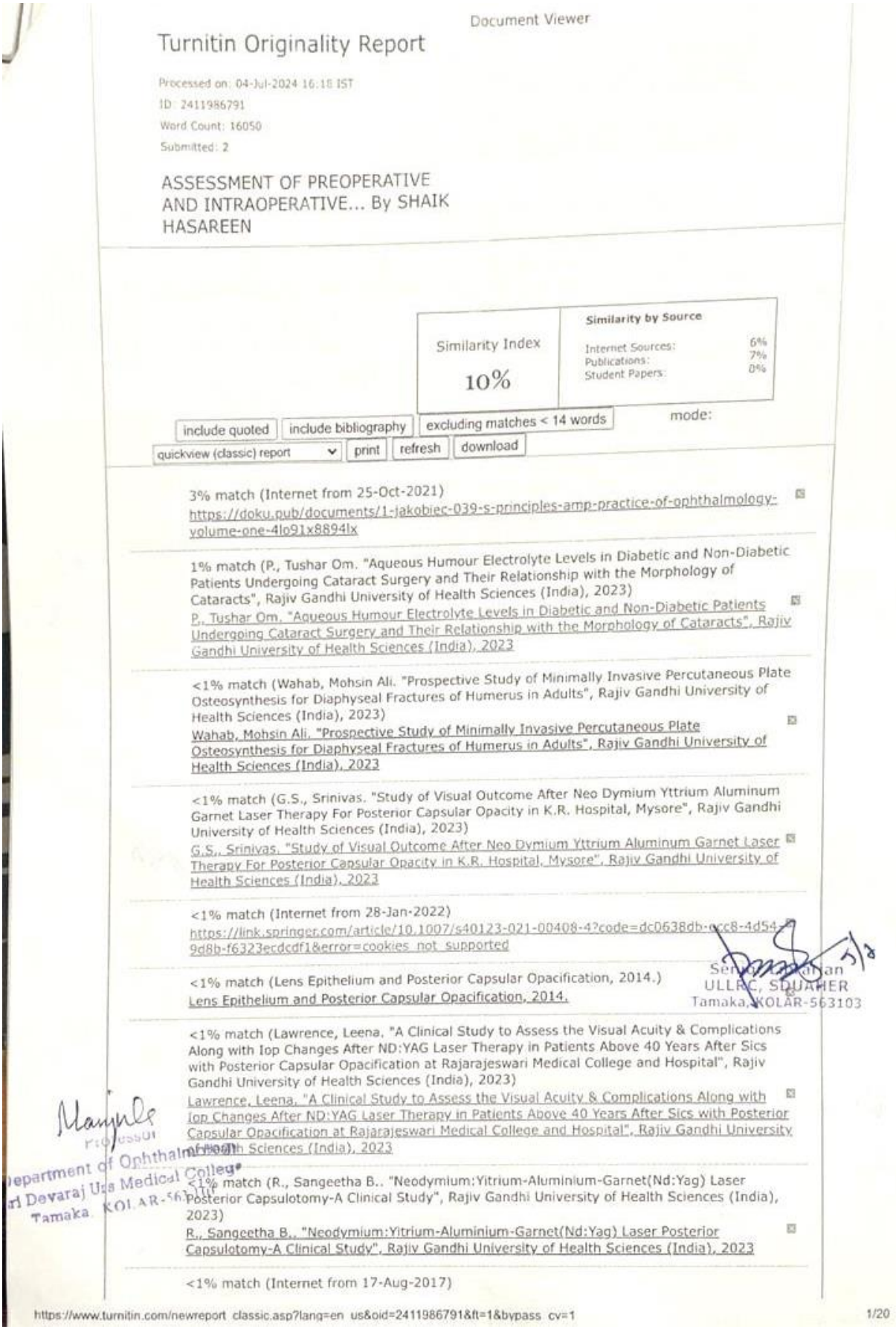

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LIST OF ABBREVIATIONS

ATP	Adenosine triphosphate
A.D	Anno Domini
BCVA	Best corrected visual acuity
B.C	Before christ
Ca^{+}	Calcium
CCC	Continuous curvilinear capsulorrhexis
CME	Cystoid Macular Oedema
ICCE	Intracapsular cataract Extraction
CRVO	Central retinal vein occlusion
ECCE	Extracapsular cataract Extraction
rhexis	Capsulorrhexis
BRVO	Branch retinal vein occlusion
gz	Germinative Zone
GSH	Glutathione
GSSG	Glutathione disulphide
HMP	Hexose monophosphate
ECCE	Extra capsular cataract Extraction
IOP	Intraocular pressure
IOL	Intra ocular lens
K^{+}	Potassium
LOCS	Lens opacities classification system
LEC	Lens epithelial cells
RNFL	Retinal nerve fibre layer

Na^+	Sodium
Nd-YAG	Neodymium yttrium aluminum garnet
NADPH	Nicotinamide adenine dinucleotide phosphate
PCO	Posterior capsular opacification
PMMA	Polymethylmethacrylate
RRD	Rhegmatogenous retinal detachment
SICS	Small incision cataract surgery
UGH	Uveitis,Glaucoma,Hyphaema
PPC	Posterior polar cataract
NS	Nuclear Sclerotic
PSC	Posterior subcapsular cataract
UCVA	Uncorrected visual acuity
MIP26	Major intrinsic protein 26
HSP	Heat shock protein

ABSTRACT

Background and Objective

Posterior capsular opacification (PCO) is a common complication following cataract surgery, significantly impacting patients visual acuity and quality of life. Modern advancements in ocular surgery, including intraocular lens (IOL) implantation and cataractous lens extraction, have improved postoperative outcomes, allowing patients to regain clear vision. But even after surgery, PCO can develop months or years later, and this continues to be a major obstacle to visual recovery. This study aims to identify the preoperative and intraoperative risk factors associated with PCO, focusing on age, diabetes status, oral steroid use, cataract type, IOL type and placement, capsulorrhexis size and surgical techniques.

Methods

In this prospective, cross-sectional study, 71 patients with cataracts noticed that their eyesight was decreased, and they had surgery. These patients were under observation for six months, after surgery throughout that period they visited the outpatient ophthalmology department of the R. L. Jalappa Hospital and Research Centre in Tamaka, Kolar. The hospital is affiliated with Sri Devaraj URS Medical College. September 2022 to December 2023 was the study period.

Results

Out of the 71 patients, 9.9% were under 40 years, 43.7% were aged 41-60 years, 43.7% were aged 61-80 years, and 2.8% were over 80 years. The study population included 39.4% males and 60.6% females, with 54.9% right-eye and 45.1% left-eye cases. Mature cataracts were present in 23.9% of patients, Nuclear sclerotic cataracts in 7.0%, Posterior polar cataracts in 5.6%, and Posterior subcapsular cataracts in 63.4%. The incidence of PCO was higher in

younger patients, with 100% in those under 40 years, 78.6% in the 40-59 age group, and 78.4% in those over 60 years. Among non-diabetic patients, 80.4% developed PCO, while 80.0% of diabetic patients did. All patients taking oral steroids developed PCO, compared to 77.4% of non-users. None of the patients with acrylic IOLs developed PCO, whereas 79.1% with PMMA IOLs did. PCO was observed in 78.3% of patients with IOLs placed in the capsular bag and 90.9% with IOLs in the sulcus. All phacoemulsification cases developed PCO, compared to 76.7% of SICS cases. PCO incidence was 77.8% in patients with a 5.5 mm size of capsulorrhexis and 81.8% in those with a 6 mm size of capsulorrhexis. PCO development was observed as early as one-month post-surgery, with the majority of cases occurring within six months.

Conclusion and interpretation

This study underscores the significance of both preoperative and intraoperative factors in the development of PCO. Younger patients, especially those under 40, have a higher likelihood of developing PCO. Key preoperative risk factors include oral steroid use, diabetes, and the grade of the cataract. Intraoperative factors, such as the size of capsulorrhexis, proper hydrodissection, and thorough cortical wash, are modifiable and can greatly influence the development of PCO. Placing an intraocular lens (IOL) in the capsular bag is preferable to sulcus implantation to lower the risk of PCO. Our findings indicate that 57 out of 71 cases developed PCO within six months, with some instances occurring as early as one-month post-surgery. This contrasts with previous studies that suggested PCO as a late complication. Overall, this study provides valuable insights for improving patient outcomes by addressing both preoperative and intraoperative risk factors to reduce the incidence of PCO.

Key words: Cataract, Posterior capsular opacification, IOL, capsulorrhexis, SICS, Phacoemulsification.

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INTRODUCTION

INTRODUCTION

Cataract dates to ancient times being the leading cause of preventable blindness worldwide, contributing to 33.4% of all blindness and the second most common cause of moderate and severe vision loss contributing to 18.4% as per global burden of disease, injuries and risk factor study. The numbers are low (<15%) in high income countries and high (>40%) in low-income countries [1].

Following a smooth cataract operation, a few months to a few years later, posterior capsular opacification—also known as after cataract or secondary cataract develops over the clear posterior lens capsule [2]. The postoperative proliferation of lens epithelial cells in the capsular bag is typically the cause of opacification of the posterior capsule[3].

It continues to be the most common cataract intraocular lens surgery complication. During the early stages of IOL procedures in the late 1970s and early 1980s, it was especially prevalent and severe[4].

Previously, there was a 25% to 50% incidence of posterior capsular opacification (PCO). Modern intraocular lenses and more recent advancements in cataract surgery have decreased the incidence of posterior capsular opacifications, however it is still a common side effect following cataract procedures[5].

Lens epithelial cell proliferation contributes to the pathophysiology of anterior capsular opacification and fibrosis in addition to the traditional PCO that occurs after surgery. There are two types of PCO, fibrous and pearl forms. It is possible for both kinds to coexist at times. There are numerous factors that are thought to increase the incidence of posterior capsular development after surgery. They consist of preoperative variables such as the patient's age. In patients who are young post cataract surgery, PCO is a significant issue, with

a frequency that is very close to 100%. It has also been observed to occur more frequently in younger patients after cataract surgery[6].

History of prolonged use of antimetabolites or steroids. Postoperative posterior capsular opacification is more likely in cases of posterior subcapsular cataract caused by steroids. In a similar vein, those who have long-term exposure to ionising radiation or take antimetabolites are more likely than others to develop post-operative posterior capsular opacification[7,8].

The most frequent reasons why diabetic people experience impaired visual acuity after cataract excision include the development of PCO [9].

It has been noted that individuals with diabetes have a higher incidence of PCO development than does the general population [10-13].

A factor in the development of posterior capsular opacity is cataractous lens grading. Its incidence is shown to be higher in patients undergoing surgery for dense posterior polar cataracts, dense posterior subcapsular cataracts, and particularly in patients with preexisting posterior capsule dense plaques[6].

The establishment of post-operative PCO is also influenced by intraoperative variables, such as:

Extracapsular cataract extraction (ECCE) became well-known in the 1980s because it was a cheap and relatively simple procedure to learn, and it retained the posterior capsule, allowing for IOL implantation. Nevertheless, this approach requires a 10–11 mm sutured incision.

For surgeons without access to a phacoemulsification machine, small incision cataract surgery (SICS) has emerged as a viable substitute. Foldable IOLs were introduced, and the phacoemulsification process significantly decreased the size of the incision [14].

A narrower capsulorhexis margin than the optic size of the intraocular lens lowers the risk of PCO following surgery. Minimising the establishment of posterior capsular opacities involves maximising the optic capsular bag contact[15,16].

Appropriately done hydrodissection enhanced cortical cleansing, Absence of cortical material reduces the chance of post-operative PCO development [17].

The kind of intraocular lens is one of the intraocular lens parameters that helps minimise posterior capsular opacification after surgery. PMMA intraocular lenses are more likely than hydrophobic acrylic lenses to cause posterior capsular opacification[18]. Comparing square-edged intraocular lenses to traditional round-edged intraocular lenses, it is also claimed that the former is better at minimising posterior capsular opacification following surgery. Comparably, PCO is more common with a decentred intraocular lens than with an in-bag placement[19] .

In addition to improving the patient's visual prognosis, reducing the incidence of posterior capsular opacification also avoids unnecessary procedures such as Nd-YAG laser capsulotomies, which can result in more discomfort and costs for the patient.

AIMS & OBJECTIVES

AIM AND OBJECTIVES:

1. To assess preoperative risk factors of posterior capsular opacification.
2. To assess intraoperative risk factors of posterior capsular opacification.

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

EMBRYOLOGY OF LENS

Surface ectodermal cells enclose the optic vesicle and thicken to form the lens placode, which is the first phase of lens development. After that, this placode invaginates into the developing optic cup and pinches off to produce the inverted lens vesicle. After being forced to undergo terminal differentiation, the cells that roughly correspond to the retinal half of the vesicle change from cuboidal to long fiber-like or simply fiber-like cells. The lumen of the vesicle is destroyed as these first, or primary, fibres extend along the visual axis. At this stage, the lens is made up of a monolayer of the remaining undifferentiated vesicular cells covering a ball of primary fibres [20].

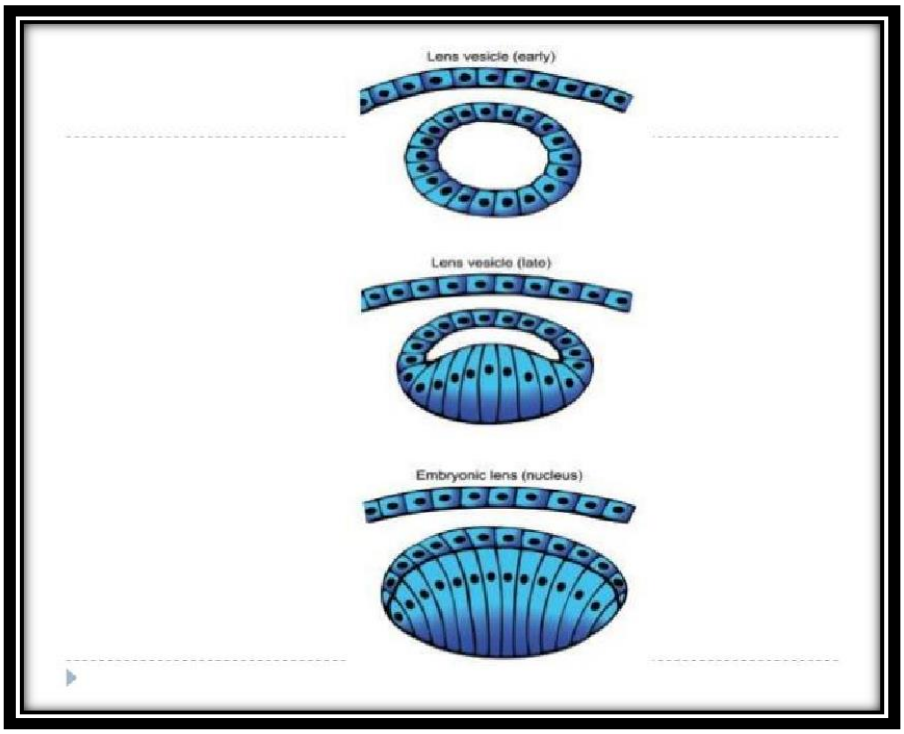


Figure 1: key structural events in the lens development

The anterior monolayer, referred to as the "lens epithelium," is the stratified epithelial-like tissue that functions as the germ cell layer of the lens throughout its life. Within the lens, stem cells are contained in the "Germinative zone (gz)," a narrow latitudinal band located at

the periphery of the epithelium just above the lens equator [21]. Some of the gz cells divide mitotically, and several of the daughter cells undergo terminal differentiation to become additional fibres; these are called secondary fibres because they are the second fibres to develop.

As more secondary fibers grow throughout life, their posterior ends extend above the capsule and below the basal membranes of the same previously formed fibers, while their anterior ends are embedded beneath the lens epithelium's apical membranes and above the anterior ends of previously formed fibers. Thus, the fibers of each layer rest above the fibers of the layer that came before them and below the fibers of the layer that came after them.

Furthermore, the lens epithelial cells and elongating fibers create a capsule that encloses the entire lens mass and resembles a basement membrane. Every fiber in the lens is permanently recorded and is sorted from the outside to the inside of the lens in ascending age order. The lens fibers continue to grow throughout life, although beyond the age of three, they do so much more slowly [22].

Because the hyaloid artery, which forms a plexus on the posterior capsule, supplies the lens in a fetus, the lens grows quickly. Mesenchyme forms this vascular posterior capsule. The thicker basal lamina forms the actual posterior capsule[23]

LENS ANATOMY

The lens, a clear crystalline structure with a biconvex orientation, is situated in a saucer-shaped depression known as the patellar fossa, between the iris and the vitreous. Its diameter is 9–10 mm, and its thickness changes with age from 3.5 mm at birth to 5 mm at the extremes of age. Its weight ranges from 135 mg for children aged 0 to 9 to 255 mg for adults aged 40 to 80.

Zinn's zonules, which are made of thin but sturdy fibers that support and connect the lens to the ciliary body, hold the lens in place[20].

The lens is composed of [24] i) Lens capsule

ii) Lens epithelium

iii) Cortex, and

iv) Nucleus

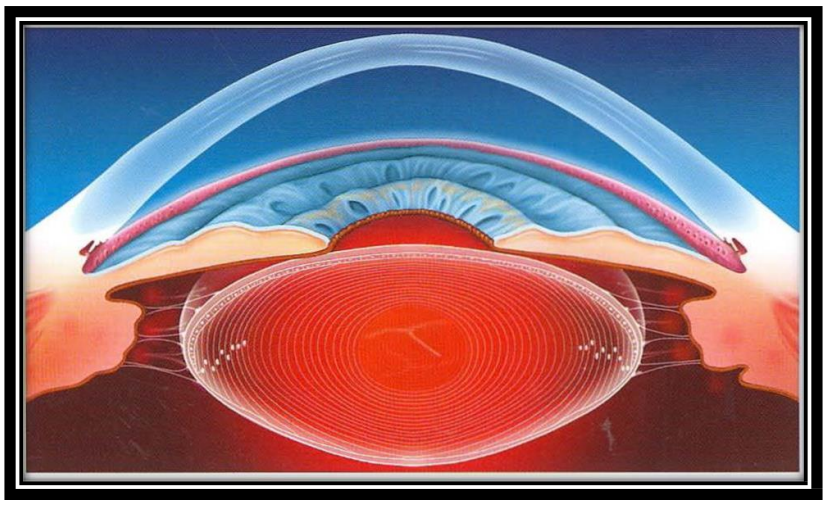


Figure 2: A highly simplified and schematic diagram showing Lens

LENS CAPSULE

The type IV collagen that the epithelial cells deposit forms the elastic, translucent basement membrane that makes up the lens capsule. The capsule can change shape during accommodative adjustments and holds the material that makes up the lens. The zonular fibers link to the lens capsule at this point through the zonular lamella, which is its outer layer.

The lens capsule is thinnest in the region of the central posterior pole, where it may be as thin as 2- 4 film, and thickest in the anterior and posterior pre-equatorial zones.

At birth, the anterior lens capsule is noticeably thicker than the posterior capsule, and this thickness grows with time.

The capsule appears homogenous, birefringent, transparent, and has a lamellar structure with fibers aligned parallel to its surface when viewed under a light microscope [24].

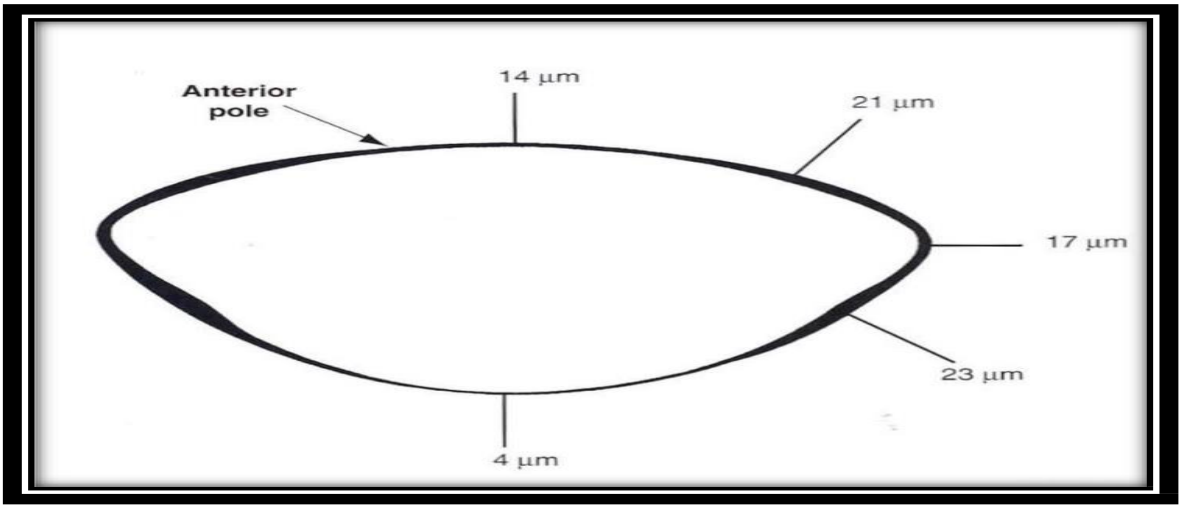


Figure 3: showing the thickness of lens capsule at different sites.

LENS EPITHELIUM

There is only the lenticular epithelium on the anterior surface of the lens, and it is located beneath the capsule. It is composed of cuboidal cells that, as they go closer to the equator, change to columnar shape. Since these cells are used up filling the core cavity of the lens vesicle during lens development, there is no epithelium on the posterior surface.

These polygonal epithelial cells have a convoluted membrane with numerous interdigitations, as demonstrated by electron microscopy observations[25].

LENS FIBRES

The elongation of epithelial cells results in the formation of lens fibers. Mature lens fiber constituent cells are devoid of nuclei. As the lens fibers mature during life, the cortex and nucleus of the lens become compactly formed.

Nucleus-The oldest fibers are located in the vicinity of the nucleus. It is composed of different zones that are created sequentially as the development progresses. These show as discontinuity zones under a slit lamp. Depending on the developmental stage, the lens's nucleus has multiple zones, which include:

Embryonic nucleus is composed of fibers that can form for up to three months throughout pregnancy. It consists of the elongating cells of the lens vesicle's posterior wall, which give rise to the primary lens fibers.

Fetal nucleus: This is the area from three months of gestation until delivery that surrounds the embryonic nucleus, which is equivalent to the lens. These fibres converge at the sutures, which are Y-shaped anteriorly and inverted posteriorly.

Infantile nucleus: This is the lens that is present from birth till adolescence.

Adult nucleus: This part corresponds to the lens fibers that begin to develop in adolescence and continue indefinitely after that [24].

CORTEX

The lens's outermost portion is where the youngest lens fibers are found. The older layers eventually get obscured by the more current layers as the lens ages. The cortex of the young adult lens is made up of every mature secondary fiber that was added during sexual maturation.

The cortex of an elderly lens is made up of all the mature secondary fibers that were added after middle age[20].

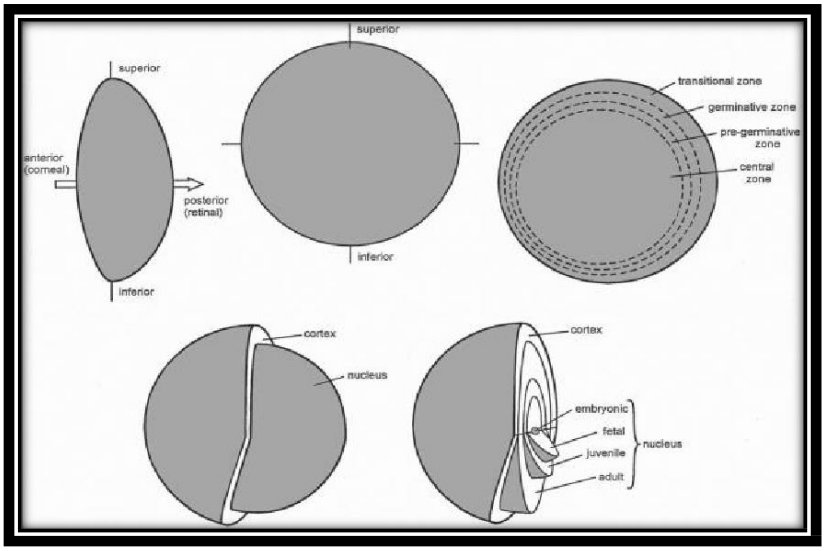


Figure 4: Diagrammatic representations of the gross shape, anatomic orientation, and developmentally defined regions of a normal human adultlens.

PHYSIOLOGY & BIOCHEMISTRY OF LENS

The molecular composition of the lens is quite different, with two-thirds made of water, one-third made of protein and the other constituents accounting to less than 1%[26].

Its high protein content keeps its refractive index high.About 65% of an adult human lens is made up of water, with the lens capsule making up the remaining 80%[27].

LENS PROTEINS

Three-quarters of the lens's net weight is made up of proteins. There are two classifications based on how soluble they are in water:-

i) Water soluble proteins- Crystallins account for 90% of total lens proteins.

ii) Water insoluble proteins- They are made up of aggravated crystallins, cytoskeletal proteins, and membrane proteins.

LENS CRYSTALLINS

i) Alpha-crystallins - Constitute up to 35% of the total lens proteins

ii) Beta-crystallins- The most abundant water soluble protein & constitutes 55%.

iii) Gamma crystallins- constitute 1-2% of total proteins

WATER INSOLUBLE PROTEINS

1. Membrane proteins constitue about 20-30% of total insoluble fraction of lens proteins.
2. Cytoskeletal proteins.

LENS LIPIDS :

It includes cholesterol, phospholipids and Glycosphingolipids. About 50-60% of lens lipids is cholesterol. Major phospholipids are Sphingomyelins.

SODIUM & POTASSIUM:

There is low sodium level and high potassium level in normal lens.

Sodium ranges from 14-26 meq /kg lens water.

Potassium ranges about 140 meq /kg lens water it is the predominant cation in the lens. Potassium levels are higher than in any other eye tissue.

CALCIUM:

The normal young lens has one of the lowest levels of calcium level of all tissues. Value of 0.14mg/mg dry weight in human lenses[27].

ANIONS:

The chloride, bicarbonate, phosphate, and sulfate anions are the primary anions in the lens. In the lens, phosphate is the main anion.

ORGANIC PHOSPHATES:

They also comprise a sizable portion of the lens's component parts. It contains pyridine and adenosine nucleotides. Glucose is phosphorylated by adenosine triphosphate (ATP). There have been reports of the presence of several additional nucleotides besides ATP, including mono and di- phosphates. Pyridine nucleotides participate in the oxidation-reduction processes by acting as coenzymes with the dehydrogenases.

GLUTATHIONE:

The range of the lens's glutathione content is 3.5–5.5 mm/g when the lens is wet.

Because of the lens's moist weight, there is a proportionate decrease in glutathione levels as people age.

Glutathione is a tripeptide composed of three amino acids: glutamic acid, cysteine, and glycine. The most reactive component that allows glutathione to exist in two different forms—oxidized form (GSSG) and reduced form (GSH)—is the cysteine group. Thus, glutathione plays a part in the lens's so called REDOX-SYSTEMS.

ASCORBIC ACID:

There is a documented large range of ascorbic acid levels in the lens, from 5 to 48 mg/100 gm wet weight. Ascorbic acid is carried in aqueous humor at a concentration fifteen times greater than in plasma. Ascorbic acid is present in the lens at a higher concentration than in the aqueous medium, but it is not actively transported or produced there [27].

LENS METABOLISMS:

GLUCOSE METABOLISM- The main source of energy is glucose. The lens requires an energy source at all times for the active transfer of ions, amino acids, preservation of lens dehydration, and lens transparency.

A necessary component for the lens's regular operation is glucose. The lens quickly depletes its internal energy stores (ATP, glucose, sorbitol, and fructose) when it is denied glucose. It also starts to retain water and become less transparent.

PROTEIN METABOLISM- It takes place in the epithelium and the outer cell layers.

The ATP generated after the digestion of carbohydrates is actively transferred from the aqueous with free amino acids. The nucleus of the lens has the slowest rate of protein synthesis of any part of the lens. The peptidases and proteases in the lens catalyze the degradation of proteins[27].

OXIDATION- REDUCTION PATHWAY- In the regular course of cellular metabolism, free radicals are created. The highly reactive free radicals have the potential to harm lens fibers.

The lens contains many enzymes, including glutathione peroxidase, catalase, and super oxide dismutase (SOD), which guard against oxidative damage and free radicals.

Superoxide disulfide (SOD) catalyzes the breakdown of superoxide anion (O2) to form hydrogen peroxide, which catalase then breaks down.

Glutathione reductase uses NADPH as a reducing agent to convert glutathione disulphide (GSSG), which is produced by glutathione peroxidase. Glutathione reductase then uses GSH to produce its own product. The HMP shunt produces NADPH.

The lens contains vitamin E and ascorbic acid, which work as free radical scavengers[28,29].

MORPHOLOGICAL CHANGES OF LENS WITH AGEING

As the lens ages, numerous morphological changes can be seen in the capsule, lens fibers, and epithelial cells. Lens epithelial cells' capacity to proliferate declines and total cell density falls[30]. As a result, epithelial cells get thinner and have flatter nuclei. They also develop vacuoles and electron-dense structures, as well as more cytoskeletal elements.The density of the cell surface projections increases noticeably with aging, increasing the surface area of the plasma membrane.

As the lens matures, specific cytoskeletal and plasma membrane proteins either completely disappear or become partially disrupted in the lens fibers, as shown at the ultrastructural level. The most important one is the reduction in cell-to-cell communication caused by the degradation of major intrinsic protein 26 (MIP26).

Actin, vimentin, and spectrin are cytoskeletal elements that are found in both the outer cortical fibers and the epithelial layer. However, as people age, they become more internalized, deteriorate, and only express themselves in the epithelial cells by the age of 80.

Throughout life, the ratio of cholesterol to phospholipid in the plasma membranes of fiber cells increases, leading to a decrease in membrane fluidity and an increase in structural order. These alterations start in the second decade and are most pronounced in the nucleus.

Reparations of these ruptures can stop opacities from forming. From the fourth decade onward, ruptures are found in the equatorial area of cortical fibre plasma membranes.

The lens capsule thickens, becomes less elastic, exhibits a loss of laminations, and the matrix proteins develop age-related cross-links. Collagen type IV is known to be present in the juvenile lens capsule and collagen I, III, and IV in the old lens capsule[31].

CHANGES IN LENS PHYSIOLOGY WITH AGEING

Changes to the cellular junctions occurs with ageing, resulting in the alterations of the cation permeability.

To create novel variations, the main gap junction protein MIP26 loses some of its amino acids. When a human lens is isolated and ages 80 years, its membrane potential will be 20 mV instead of 50 mV as it was when it was 20 years old.

While potassium (K^+) levels are mostly stable at about 150 mmol/L throughout life, the sodium (Na^+) concentration of the lens increases from 25 mmol/L at age 20 to 40 mmol/L by age 70.

By the time an individual reaches the age of 80, the Na^+:K^+ permeability ratio has increased six times, leading to a correspondingly higher rise in the sodium content of the lens. This rise is correlated with an increase in the lens's optical density [32].By the time an individual reaches 60 years old, free Ca^{2+} has increased from 10 μmol/L at age 20 to approximately 15 μmol/L [27].

AGE-RELATED CHANGES OF LENS CHROMOPHORES.

As people age, a variety of chromophores that absorb longer wavelength visible light at first, then blue light, gradually accumulate. These chromophores build up and cause the lens to gradually become yellower. As the lens ages, it may take on a brunescent appearance.

AGE-RELATED METABOLIC CHANGES

With advancing age, the lens's total metabolic activity as well as the activity of numerous oxidative and glycolytic enzymes decline. The lens mostly maintains its ability to manufacture proteins, fatty acids, and cholesterol at significant rates even as total metabolic activity declines. Reduced metabolic activity may indicate an aging lens rather than disease and is not a major factor affecting the production of new lens fibers.

OXIDATIVE STATUS OF THE AGED LENS

The lens needs to maintain its proper transparency and refractive quality throughout its life, even in the face of oxidative damage, to continue performing its primary role of concentrating visible light on the retina.

First of all, because the lens is continuously exposed to UV light, reactive oxygen species are produced and oxidation reactions take place. Second, the concentration of other lenticular photosensitizers increases with age.

From birth until approximately the age of 15, glutathione peroxidase levels rise, and then they gradually fall throughout adulthood. In lenses older than 70 years, it drops by 50–60%[33].

CHANGES IN CRYSTALLINS

Lens crystallins structure, stability, and function are necessary to keep the lens clear and prevent lens opacification, or cataracts. By the age of 45, up to 80% of an old lens's nuclear proteins, including α-crystallin, may be insoluble, which may lead to the development of senile cataract and a loss of lens clarity.

Throughout life, the lens is subjected to the combined impacts of oxidation, radiation, and posttranslational alteration. As we age, our crystallins undergo post translational alterations that cause them to unfold and eventually aggregate.

The presence of proteins in the lens, such as α-crystallins from the Heat Shock Protein (HSP) family, prevents early crystallin aggregation.By attaching to partially denatured proteins, these proteins function as molecular chaperones, avoiding irreversible protein aggregation that occurs with age.

CLASSIFICATION OF CATARACT:-

1) Congenital
2) Acquired further classified as
    a. Age-related
    b. Radiation induced
    c. Toxic
    d. Traumatic
    e. Ocular diseases
    f. Systemic diseases
    g. Metabolic disorders.

MECHANISMS OF CONGENITAL CATARACT-

Different forms of faulty crystallins, which are linked to the development of various forms of congenital cataracts, can arise from a variety of genetic abnormalities.

A cataract can develop from any injury to the lenticular or nuclear fibers. Intrauterine infections, metabolic diseases, and genetically transmitted illnesses are among the potential reasons.

Infectious causes of cataracts include-

1. Rubella (most common)
2. Epstein–barr virus
3. Herpes zoster
4. Syphilis
5. Herpes simplex
6. Cytomegalovirus
7. Toxoplasmosis and
8. Familial congenital cataracts like Galactosemia, pierre-robin syndrome, Down syndrome,trisomy 13, Lowe syndrome.

SENILE CATARACT

There are different types of age related cataract depending on the area of the lens opacity

1. Nuclear cataract
2. Cortical cataract
3. Sub capsular cataract
4. Mixed cataract

NUCLEAR CATARACT-

1. The rate at which nutrients, water, and antioxidants may get through the epithelium and into the lens nucleus cells decreases with age. Lens fiber production and homeostasis are altered by decreased lens epithelial cell density.
2. After the age of fifty, there is a rise in the cataractous lens's insoluble to soluble protein ratio.
3. Oxidative factors lead to cataract formation by altering the morphology of the human lens and causing metabolic damage. The most significant antioxidant in the lens is glutathione (GSH), followed by vitamin C and ascorbic acid.
4. Damage to cell membranes: Methionine and cysteine are the most susceptible amino acids to oxidation, especially in proteins that are connected to cell membranes. In cataractous lenses, membrane lipids are seen to have oxidized. Affect the way membrane-associated enzymes like $Ca2^{+}$ATPase and $Na^{+}K^{+}$-ATPase function, which causes epithelial cell death. Osmotic shock, crystalline aggregation, and lens opacification result from this.

CORTICAL CATARACT

Several mechanisms may initiate the cortical cataract, which includes -

1. Damage to the plasma membrane of lens fibre.
2. Depletion of protective molecules such as glutathione,
3. Excessive protein degradation,
4. Electrolyte and water balance disruption and
5. Injury to the calcium homeostasis regulating system. In the early stages of cortical cataract formation, these elements are connected. Opacity moves toward the nucleus and around the lens's periphery when calcium homeostasis is lost. Cortical cataracts cause injured cells to have higher calcium levels. Proteolysis, protein aggregation, and

light scattering are all brought on by high calcium levels.

SUB CAPSULAR CATARACT

The anterior subcapsular type of cataract is associated with fibrous metaplasia of the lens epithelium directly beneath the anterior lens capsule. Posterior subcapsular opacity develops due to swollen and migratory lens epithelial cells and causes opacity due to breakage of the swollen lens fibre cells. Opacification occurs exactly in the pupillary axis and particularly has a profound effect on vision.

TOXIC CATARACT

There are several drugs which are believed to cause cataract like Steroids, miotics, chlorpromazine, allopurinol, chloroquine, and amiodarone, on the other hand there are other drugs that are proven to retard cataract formation like non-steroid anti-inflammatory drugs[34]. 5% to 60% of patients have been reported to develop cataracts because of corticosteroid therapy; of which bilateral posterior subcapsular cataracts are especially common [35].

RADIATION

Ionizing radiation damages the germinal epithelium at the lens equator, which is how X-rays, neutrons, and gamma rays induce cataracts. The length of this process varies from 4 to 10 years based on the level of exposure. Feathered and dust-like opacities are the result of the faulty fibers migrating to the posterior pole.

TRAUMA

Both blunt and piercing traumas can harm a lens. The blunt force results in the globe equatorial expansion and antero-posterior shortening, which in turn causes the lens fibers in the cortex axial area to split apart, giving rise to a cataract with a floral shape.

METABOLIC DISEASES

They include Lowe syndrome, galactosemia, hypocalcemia, and diabetes mellitus.

CATARACT GRADING

The process of classifying cataracts using a standardised manner involves identifying the type, severity, or both condition.

They are basically of 2 types A) IN VITRO B) IN VIVO

The LOCS III system uses five standards for cortical and posterior subcapsular grading and six nuclear standards for colour and opalescence grading. It is currently accepted and utilized for grading cataract worldwide [36].

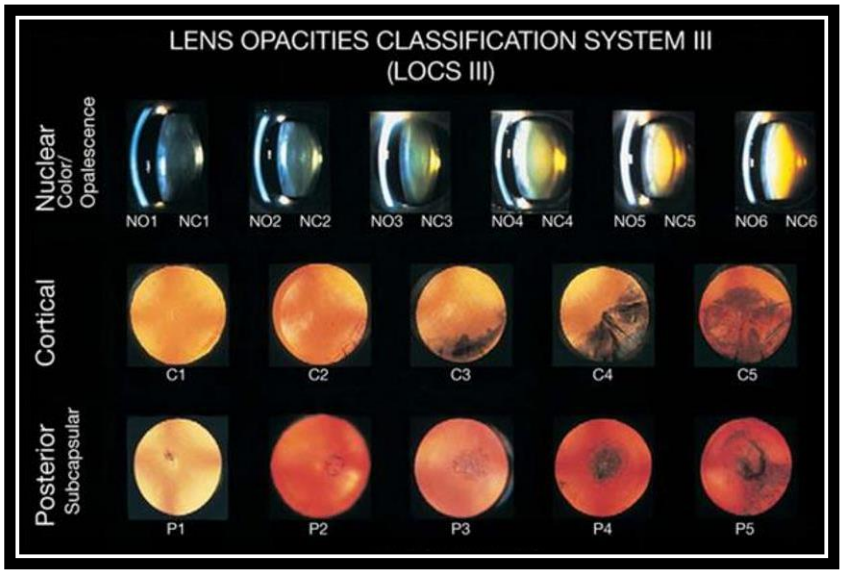


Figure 5 : LOCS III. Set of slides for grading standardized photographic images of opacity.

Other in vivo gradings were also described

WILMER GRADING

Four nuclear standard photos are used by the Wilmer system to grade nuclear opacity. Three factors determine the grades: visual acuity, density, and degree of opacity[37].

The only opacities seen under retro-illumination are categorized by calculating the proportion of the lens overall circumference that is accounted for by the combined posterior sub-capsular and cortical opacities.

OXFORD GRADING

It is a comprehensive method of assessment which grades the intensity from a scale of zero to five.it includes several characteristics like anterior clear zone, anterior subcapsular cataract, posterior sub capsular cataract, cortical spoke opacities, water clefts, vacuoles, focal dots, retro dots, nuclear brunescence and white nuclear scatter[38].

Japanese-CCESG system

This system also uses a set of standard photographs in grading cortical, nuclear, and subcapsular opacities. These cataracts are graded as early, moderate, or advanced[39].

HISTORY OF CATARACT SURGERY

The term cataract was introduced by **Constantinus Africanus (A.D 1018),** a monk and an Arabic oculist. He translated Arabic suffusion into Latin cataracta meaning the water fall [40].

Couching was the main technique for removing the cataract from the pupil for more than 200 years. Sushruta, an ancient Indian surgeon, wrote the earliest recorded account of couching approximately 600 B.C.

A surgeon sat facing the patient and conducted the couching. The midday sun was shining through a window as the patient sat, with light flowing across her or his face. The patient's head was further stabilized by having an assistance stand behind them. A sharp needle was inserted either through the clear cornea or the sclera approximately 4 mm temporally to the limbus. After that, the needle was blindly inserted behind the iris and toward the lens through the conjunctiva and sclera.

The white opacity would then be pushed downward (a technique known as depression) or

backward (a technique known as reclination) by the surgeon using a blunted needle.

Iraqi oculist Ammar (A.D 996–1020) produced the Book of Selection of Eye Diseases and gave instructions on how to suction a cataract using a hollow needle. Ammar's aspiration technique was used by Syrian surgeons in the twelfth and thirteenth centuries.

French oculist Jacques Daviel (1696–1762), who was born in Normandy, initiated a surgical innovation revolution that is still going strong today by outlining a brand-new, methodical procedure for removing cataracts from eyes. He described the specifics of this novel procedure in 1753.

Figure 6: Daviels approach of cataract through an inferior incision and the cataract is expressed out with finger pressure [41]

The surgical incision was moved to the upper portion of the eye by Pierre-Francois Benezet. Operating at the head of the table, he had the patient lie on his or her back. A German oculist named Carl Himly introduced pharmacologic mydriasis, which let the surgeon see better. Samuel Sharp (1753) presented the concept of removing the entire lens from the eye while keeping the capsule intact.

Phacoerysis was carried out by Ignacio Barraquer in 1917 using pneumatic forceps. Jose Barraquer, his son, invented an electric vacuum pump device with an erysiphake handle specifically designed for the purpose of extracting cataracts using suction.

The subsequent advancement in ICCE surgery was the discovery of chemical zonulolysis.

Using the enzyme α-chymotrypsin, Jose Barraquer (1958) showed the chemical zonulolysis remarkable potency.

In Poland, Krawawicz invented the cryoextractor in 1961. The risk of capsule rupture during extraction could be reduced by applying a tiny, cold probe to the lens surface and freezing it there to produce an ice ball that would fuse the lens's capsule, cortex, and nucleus.

THE RETURN TO ECCE

Even with the positive outcomes of ICCE, there was still a significant risk of potentially blinding side effects, such as cystoid macular edema and aphakic retinal detachment, which may be avoided by maintaining the posterior capsule. Additionally, smaller incisional cataract lesions were targeted.

However, the patient who was aphakic and needed glasses for optical rehabilitation was the main worry.

On November 29, 1949, Harold Ridley carried out the first artificial lens implant at St. Thomas Hospital in London. Nothing remained to sustain Ridley's posterior chamber lens after the ICCE.

Subsequently, Jaffe and associates significantly contributed by highlighting the fact that the Extracapsular treatment had a reduced rate of problems.

In a prospective research, Norman Jaffe, Henry Clayman, and Marc Jaffe demonstrated that there was less cystoid macular edema in uncomplicated ECCEs than in uncomplicated ICCEs, as demonstrated by angiography.

When Kelman first presented his phacoemulsifier in 1967, many intracapsular surgeons expressed concern due to the device potential for problems. A novel capsulotomy was the first creative thought to improve the safety of phacoemulsification. Gimbel presented his continuous tear capsulotomy and Neuhann explained his capsulorrhexis at the same time.

After realizing their significant contribution, Gimbel and Neuhann opted to co-publish a comprehensive description and identify the process as continuous curvilinear capsulorrhexis (CCC).

Gimbel demonstrated that the nucleus could be split inside the bag by breaking the nucleus in his Divide and conquer nucleofractis, which sparked a significant development in phacoemulsification.

Fine explained his "chip and flip" technique, which involves turning the epinucleus over and emulsifying it.

A crack and flip was described by Dillman and Maloney. Japan's Kunihiro Nagahara astounded the surgical community with his technique phaco Chop. Bimanual phacoemulsification using a 0.90-mm clean corneal incision; this method, which he named Phakonit, was created by Indian physician Amar Agarwal.

The choice to operate is mostly based on several indicators as well as patient demand, which has increased significantly in recent years as a result of the accessibility of premium implants and advancements in cataract surgery. It needs to be confirmed that the cataract caused the vision impairment.

Counselling patients to expect the desired result while also warning them that there are dangers associated with any surgical operation is an important element of patient preparation.

PREOPERATIVE EVALUATION IN CATARACT SURGERY

- Informed consent
- Ocular examination
- Systemic examination
- Serological tests & Blood glucose.

OCULAR EXAMNATION

1. Visual acuity
2. Pupillary assessment
3. Slit lamp examination
4. Fundoscopy
5. Axial scan biometry for IOL power determination.
6. Gonioscopy to be done if the anterior chamber is shallow.
7. Colour vision for gross macular function and 2 point discrimination for gross retinal function in advanced cataract.
8. Ultrasound scan of posterior segment is mandatory if the fundus is poorly seen.
9. Corneal topography to assess the pre-existing astigmatism
10. Corneal specular microscopy to rule out any endothelial dystrophies or to ensure the endothelial abnormalities.
11. Electrodiagnostic tests may be used in advanced cataracts.

For a patient to give their informed consent, the patient must be informed on the nature of the entire process, the surgical schedule, what to expect after surgery, and a review of any difficulties.

CATARACT SURGERY

There are different techniques of cataract surgery

i) Intracapsular cataract extraction (ICCE)- The lens is removed in toto along with the posterior capsule and it includes techniques such as cryoextraction, capsule forceps extraction, phacoerysis.

ii) Extracapsular cataract extraction (ECCE)- it mainly preserves the posterior capsule and is followed by in-the-bag IOL placement .It includes conventional ECCE, Small incision ECCE and phacoemulsification all of the techniques which preserve the posterior capsule.

iii) Phacoemulsification & femtosecond assisted phacoemulsification – are also ECCE type surgeries both of which use ultrasound energy to emulsify the nucleus. The femtosecond laser is used to create a capsulorrhexis and the rest of the surgery just follows the same steps as in phacoemulsification.

BASIC STEPS OF ECCE

SICS and phacoemulsification are commonly performed surgeries.

After anesthesia, the globe is fixed with a bridles suture.

In SICS after constructing a 5.5-6.5mm tunnel, anterior capsulotomy (CCC/can opener/ Envelope) is performed followed by a cortical cleaving hydrodissection . The nucleus prolapse and nucleus delivery is done and followed by Irrigation and aspiration of cortical matter and IOL implantation in the bag.

In phacoemulsification, a clear corneal tunnel of 2.5-3.2mm is made followed by anterior capsulotomy and hydro-dissection, the nucleus is emulsified with ultrasonic energy in the bag

followed by implantation of foldable IOL after a thorough cortical wash.

COMPLICATIONS OF CATARACT SURGERY

1.Preoperative complications

2.Intraoperative complications

3.Early postoperative complications

4.Delayed postoperative complications

5.IOL related complications

PRE-OPERATIVE COMPLICATIONS

1. Anxiety
2. Nausea and gastritis due to preoperative medicines such as acetazolamide and/or glycerol.
3. Retro bulbar haemorrhage
4. Oculo-cardiac reflex.
5. Globe perforation
6. Insufficient analgesia
7. Insufficient akinesia

COMPLICATIONS OF CATARACT SURGERY

Intraoperative complications

1. Superior rectus injury
2. Incision related complications

-Button holing of anterior wall of tunnel,

-premature entry into AC

3.Tear of descemet Membrane

4. Iris prolapse/ intra-operative floppy iris syndrome

5. Complications related to anterior capsulotomy

-Peripheral extension

-Small capsulotomy

6. Chamber collapse, Positive pressure, Thermal burns in phacoemulsification.

7. Posterior capsular tear

8. Improper placement of IOL

9. Zonular dialysis

10. Nucleus drop

11. Expulsive choroidal haemorrhage

Early post operative complications

1. Pain

2. Wound dehiscence, wound leak, wound rupture

3. Corneal oedema

4. Secondary Glaucoma

5. Postoperative anterior uveitis

6. Bacterial endophthalmitis

Late post-operative complications

1. Cystoid macular oedema

2. Pseudophakic bullous keratopathy

3. Retinal detachment

4. Epithelial ingrowth

5. Fibrous downgrowth

6. Secondary glaucoma

HISTORY OF IOL

After World War II ,Sir Harold Ridley implanted the first intraocular lens (IOL) in the St. Thomas Eye Institute, leading to the development of contemporary cataract surgery with IOL implantation.

Hospital in London after his observation of Perspex splinters from cockpit canopies which were not rejected by the body immune system by a patient. He has speculated on the possibility of making intraocular lenses with such material.

Ridley implanted his first lens in a 45-year-old woman on November 29, 1949, (PMMA) Polymethylmethacrylate was the substance used in the IOL.This is considered as one of the greatest milestones in medical history. Ridleys invention has become an accepted option for optical correction of aphakia. Thereafter tremendous changes and improvements were noticed in this field of medicine which had led to the development of different biomaterials and designs.

Table 1: Generations of IOLs

Generation	Types	Examples	Advantages	Disadvantages
1	PCIOL (1949-1954)	Ridley's PMMA IOL	Revolutionised the visual rehabilitation of patients after cataract surgery	Subluxation
2	RIGID ACIOL (1952-1962)	Strampelli tripod ACIOL CHOYCE Mark1 Dannheim ACIOL	No decenteration	UGH syndrome , Corneal decompensation
3	Iris fixated lens (1953-1975)	Binkorst iris loop lens Fydorov sputnik lens Jan worst iris claw lens	Can be used Both in ICCE and ECCE	Iris chaffing andAC inflammation
4	Flexible andsemi-flexible ACIOL (1963-1990)	Azar 91Z ACIOL ORC Inc stableflex Surgidev Kelman multiflex	Biocompatiblematerials Better fit and design	Corneal endothelial damage
5	Improved PCIOL (1975-1990)	All current 3-piece PMMA IOLs	Cost effective More stability Less chances of decentration	Need largeincision Astigmatism dueto wound
6	Foldable IOLs (1970s to present)	Silicone IOL	Heat resistant. Excellent tensile and tear strength	low refractive index can be pitted
		Hydrophobic acrylic IOL	Reduced rate of PCO Thinner lens good resistance to YAG laser	photopsia & glistening susceptible to damage by forceps
		Hydrophilic acrylic IOL	-Thin lens	Higher rate of PCO. Susceptible to damage
7	Multifocal IOLs (1986 to present)	Bulls eye type Annulus type Rezoom IOL	No need of a near addition required	Glare, halos Undesirable visual aberrations
8	Accommodative IOLs Toric IOLs (1994 to present)	Staar surgical IOLs AcrySof IQ toric IOL AcriComfort646TLC	Astigmatism corrected	Rotation of IOL

POSTERIOR CAPSULAR OPACIFICATION/ AFTER CATARACT

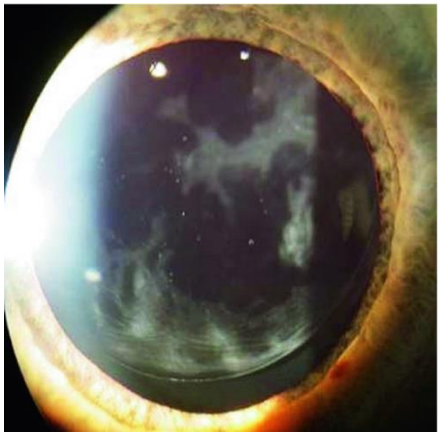
It is one of the vision impairing complication which occur in up to 50% patients post cataract surgery between 3months- 5years.With the advent of new IOL designs and in-the-bag placement of the lens and techniques, the incidence has reduced but not yet eliminated.

The incidence of after-cataract in children is very high and almost inevitable after pediatric cataract extraction due to the high proliferative capacity of the youngresidual lens fibres.

Clinically they are mainly divided into 3 types-

1) Fibrous type
2) Elschnig pearls
3) Sommering ring

FIBROUS TYPE OF PCO



Photograph 1: Fibrous type of PCO

The existence of residual lens epithelial cells adhered to anterior capsule's underside is the primary cause of this kind of PCO.

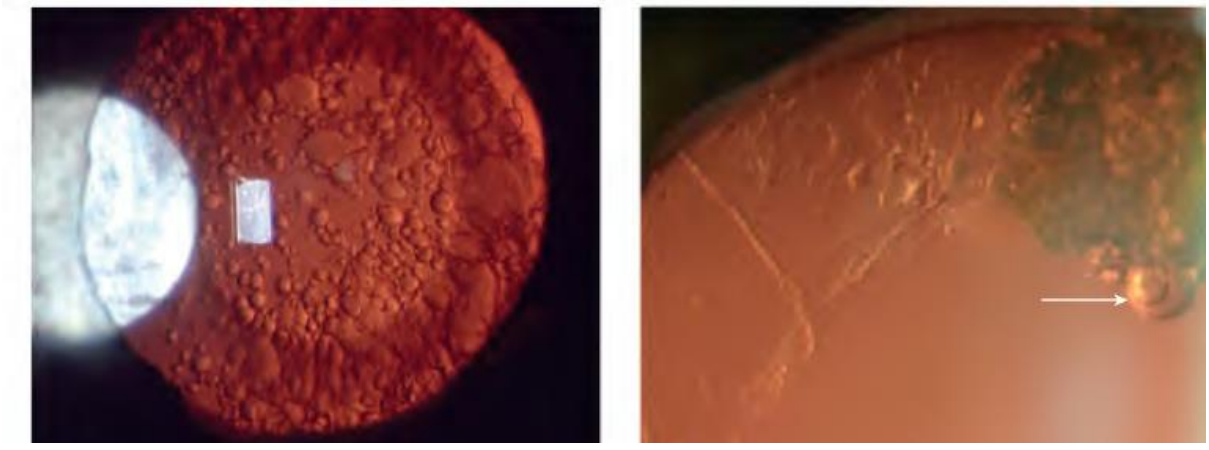
These remaining epithelial cells differentiate into cells with a contractile activity called spindle cells, which resemble fibroblasts. These fibroblastic cells multiply and move into the

posterior capsule to produce a cellular layer that secretes material resembling basal laminar tissue and components of the extracellular matrix. The contraction by this cellular layer results in the folds & wrinkling on the posterior capsule.

Early fibrosis tends to develop within 2-6months of cataract extraction, which can sometimes be clinically significant if it interferes with the visual axis. Late fibrosis develops after 6months which occurs due to further proliferation and multilayering of cells onto the posterior capsule. These cells lay down the extracellular matrix which is mainly composed of collagen type 1 and type 3 and proteoglycans like dermatan sulfate and chondroitin sulfate.

The basal laminar like material that is laid is composed of collagen type 4 and proteoglycans like heparin sulfate. Certain growth factors, such as acidic/basic fibroblast growth factor, epidermal derived growth factor, platelet-derived growth factor, and transforming growth factor beta, further promote this fibroblastic proliferation and cellular expansion.

ELSCHNIG PEARLS



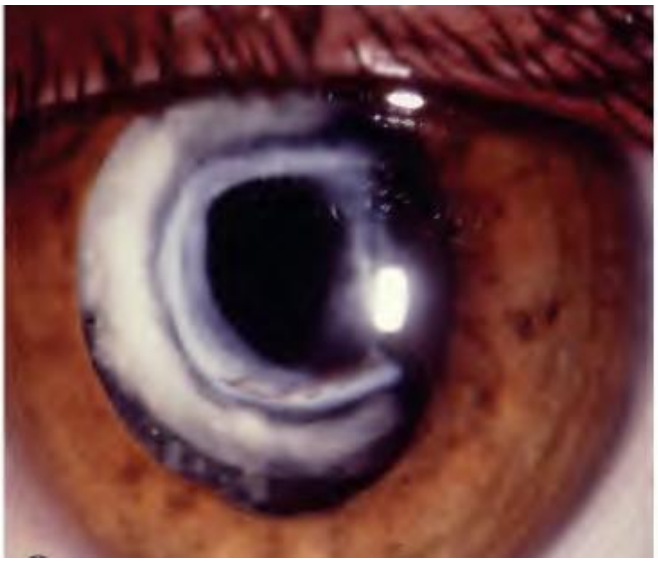
Photograph 2 : Elschnig pearl type of PCO

This type of PCO is mainly because of the residual wedl cells/bladder cells which originate from the equatorial lens epithelial cells which are involved in the formation of pearls.

After cataract extraction, there is no internal pressure inside the capsular bag which directs the newly formed lens fibres to align anteriorly/posteriorly, hence it results in formation of mass of cells which are large & globular that are loosely arranged and piled on each other to form pearls over the posterior capsule.

A single pearl is an epithelial cell that has tried to develop into a novel lens fibre with traits shared by both epithelial cells and fibres. When pearls build up in the posterior capsule's centre, it affects eyesight.

SOMMERING RING



Photograph 3 :Sommering type of PCO

Within the space between the anterior and posterior capsules, residual cortical fibres and epithelial cells are caught and sealed within this structure. The ability to divide and differentiate into lens fibres is still present in equatorial cells. Ring formation occurs as a result, however it is peripheral in nature and has no effect on vision.

PATHOGENESIS OF PCO

Anatomically there are two types of cells involved in the development of PCO. They are mainly of 2 types A-cells and E-cells. McDonell et.al have shown that lens epithelial cells undergo hyperplasia & differentiation into myofibroblast like cells within few days after surgery. Wedl cells, or balloon-like bladder cells, are formed by E-cell proliferation and migration onto the posterior capsule in Elschnig pearls.

A-cells proliferation is involved in the development of anterior capsular opacification and fibroblastic changes of posterior capsular opacification.

Both autocrine and paracrine mechanisms were found to influence the formation of PCO. These mechanisms were found to be influenced by several cytokines increased lens epithelial cells mitosis and collagen synthesis.

There are different types of lens remnants as per Duke-elder:

1) Capsular remnants
2) Capsule-lenticular remnants
3) Pigmentary, hemorrhagic or inflammatory fibrous elements.

The cuboidal epithelium lines the anterior capsule, which transform into fibrocyte like cells by metaplasia which undergo proliferation but do not migrate.

The germinal equatorial lens epithelial cells which have mitotic activity migrates to form epithelial pearls on the posterior capsule. Lentoid bodies are hyaline deposits which are closely packed and arranged lens fibres which cause optical distortion.

The lens cortical remnants following cataract surgery undergo lysis by the aqueous.

The anterior capsular edge folds inwards to reach the posterior capsule to form a doughnut shaped structure within the walls of which the epithelial cells proliferate due to regenerative cells within the walls.

METHODS OF ASSESMENT OF PCO-

1) Subjective tests
2) Objective tests

Subjective assessment of PCO is mainly based on the visual function tests.

Hayashi.et.al,2003[42]**, Aslam & patton**[43], 2004 and **Meacock**[44] et.al 2003 have shown that visual acuity and contrast sensitivity are the 2 main visual functions that are affected by PCO. They have linked the development of glare disability and forward light scatter also to PCO development.

Objective assessment is mainly based on slit lamp and classified differently.

Kruger et.al[45] graded the PCO of central 3mm as

0= Absent

1= Very mild

2= Moderate

3=Dense white

Sellman[46] **and lindstorm** graded PCO on a four point scale which uses diagrams to illustrate the various grades of both fibrous as well as pearl type of PCO.

1= No or slight PCO without reduced red reflex /no pearls on the IOL edge

2=Mild PCO reducing the red reflex / Elschnig pearls on the IOL edge

3=Moderate fibrosis/ pearls inside the IOL edge but clear visual axis

4=Severe fibrosis /pearls covering the visual axis reducing the red reflex

Congdon's study[47]**:** GRADING OF PCO:

Grade 0: without PCO, no opacity appeared only on peripheral capsule.

Grade 1: Wrinkling or opacity of the capsule limited in a circle 4 mm in diameter and centered on the visual axis, but the posterior polar retina could be viewed clearly.

Grade 2: Central/paracentral opacity worse than grade 1 that affected the detailed observation of macula slightly but had no effects on the observation of cup/disc ratio.

Grade 3: central/paracentral opacity worse than grade 2 and making cup/disc ratio difficult to ascertain.

Grade 4: central/paracentral opacity as defined above but making fundus observation difficult or impossible.

Legler.et.al[48]used the visibility of posterior segment structures including optic nerve head, retinal blood vessels, retinal nerve fibre layer with indirect ophthalmoscope.

Grade 1= RNFL &blood vessels seen clearly

Grade 2= optic nerve head visible, RNFL & blood vessels are hazily seen

Grade 3= optic nerve head hazy.

Sangeeta.et.al[49] used the visibility of posterior segment for grading the PCO

Mild= Fundus seen clearly with direct ophthalmoscope

Moderate= Fundus seen clearly with indirect ophthalmoscopy

Severe=No view of fundus or details hazy

In our study we used the same technique to grade the PCO as in **Congdon's study**

Though these techniques of grading are easy to apply clinically, they have inter-observer variability and lack precise quantification.

Imaging systems

1. Scheimpflug system It makes use of densitometry, which gauges the intensity of light

scattered. It is highly reproducible, but IOL material significantly affects the scattered light density measurement.

2. Digital photography A number of groups of computer based systems exist, and each one uses a certain approach to measure it. According to Wang et al., an analysis system based on brightness that grades PCO depending on the grey value of an image pixel

Heidelberg imaging, which uses retro-illumination photos to score PCO (Tetz et al) Density map system by friedman.et.al.

3. TRACEE, an upgraded computer programme for image analysis that includes modules for PCO grading.

RISK FACTORS FOR THE DEVELOPMENT OF PCO

1. Young age: The rate of LEC proliferation is age –dependent and the rate in patients <40yrs is 3 times faster than that of rate in >60yrs.The development of severe PCO is more common in diabetics than in non-diabetics. It was hypothesized that myopic eyes will develop PCO more frequently.

When compared to other cataract types, steroid-induced cataracts are linked to a higher prevalence of PCO development after surgery. Patients with myotonic dystrophy are more likely to experience posterior capsular opacification, which increases the requirement for repeated Nd-YAG capsulotomies. Compared to the general population, patients with retinitis pigmentosa have a higher risk of developing post-operative PCO. Nearly 92% of patients with traumatic cataracts are at risk of developing PCO, according to a three-year follow-up research.

2.Surgical procedure- Minimal surgical manipulation is always a pre-requisite for a favorable post-operative outcome Excessive manipulation would interfere with the blood-aqueous barrier and lead to inflammatory cells in the anterior chamber. Protein-rich environment favors proliferation of the LEC, thus increasing the risk of PCO development.

A proper cortical cleaving hydrodissection facilitates the removal of cortical matter together with E-cells from the equatorial region which are the culprit cells for the development of PCO.

Though contradicted by some studies[50] phacoemulsification with cortical irrigation & aspiration lead to less residual LEC concentration than with ECCE with manual nuclear expression[51] and meticulous vacuuming of the capsule using ultrasound endocapsular cataract extraction greatly reduced the need for laser capsulotomy[52].

3.IOL related factors Many IOL related factors are considered which are thought toinfluence the incidence of PCO. The biocompatibility of the material that is used is a factor that significantly influences the incidence. It has been proved that the hydrophilic lenses are associated with more incidenceof PCO than hydrophobic lenses[53].

Apart from the material of the IOL used, there are many other factors such as overall length of the IOL, optic edge design, optic diameter which influence the incidence.

Square truncated edge is a desirable optic edge design which prevents the migration of the equatorial lens epithelial cells to the visual axis, provided the IOL is placed in the bag. This advantage of the edge design is almost absent if it is not placed entirely in the bag.

The round edge design would leave potential space for the proliferative cells to reach the posterior capsule even if it is placed in the bag.

Implants like capsular tension rings are also advocated by some surgeons before implanting the IOL to achieve the stretching of the capsule and engaging the equator, thus preventing the migration of equatorial cells.

4.Pharmacological alterations Immunotoxin (MDX-A) is an important pharmacological factor that is found to be associated with reduced PCO rates[54,55].

Use of heparin is also advocated to reduce the incidence of the PCO either in the form of irrigating solution (or) with the usage of IOL coated with heparin (or) as topical eye drops[56].

TECHNIQUES TO PREVENT PCO FORMATION ARE AS FOLLOWS:

Surgical strategies modifications

1. Cortical- cleaving hydro-dissection
2. Meticulous capsular polishing
3. Controlled capsulorrhexis aimed at maintaining the size of the opening slightly smaller than the IOL optic size to bring a sealing effect.
4. In-the bag IOL placement

Intra-ocular lens related changes

1. Preference to use a bio-compatible IOL made of hydrophobic acrylic material
2. IOL optic edge design should be square- truncated.
3. Maintaining adequate touch of the IOL with posterior capsule.

MANAGEMENT OF POSTERIOR CAPSULAR OPACIFICATION-

1. Surgical management
2. Laser management.

SURGICAL MANAGEMENT

In remote areas where there is no laser infrastructure or if the thickness of the PCO is not amenable to laser, surgical approach can be adopted, however the results are inferior compared to laser.

The technique requires a 26-gauze needle which is bent at the tip to make a capsular nick either through a pars-plana approach (or) through a limbal approach. The procedure should be always combined with anterior vitrectomy. In cases where the PCO is a thick membrane and is accompanied by cortical matter, a proper sized membranectomy should be done to clear thevisual axis and combined with a pars plana anterior vitrectomy.

Surgical capsulotomy is of 2 types which basically involve the same technique, but adopted at different times

1. Primary posterior capsulotomy:

   Congenital cataracts which are expected to show almost a 100% incidence of posterior capsular opacification within a 2year duration can be managed in the same sitting by a planning a posterior capsulorhexis followed by anterior vitrectomy with either a in-the-bag placement (or) by an IOL-optic capture.

   Even with primary posterior capsulorrhexis it might not be possible to prevent the visual axis opacification due to the opacification of the hyaloid face.

   The dye used for staining the posterior capsule are 0.5% indocyanine green, 0.1% trypan blue.

2. Secondary surgical capsulotomy:

It is done in cases of thick posterior capsular opacities which are not amenable to YAG capsulotomy.

After a peribulbar block, the approach can either be through pupil or through pars-plana. Pars-plana approach is preferred and is achieved with a bent needle which traverses the vitreous or surgical discission with a zeiglers knife.

Complications associated with surgical treatment of PCO-

1) Anesthesia related complications.
2) Loss of vitreous
3) Retinal detachment
4) Cystoid macular oedema
5) Endophthalmitis

LASER MANAGEMENT

NEODYMIUM YTTRIUM ALUMINIUM GARNET LASER (Nd-YAG)

CAPSULOTOMY

LASER- It is acronym for light amplification by stimulated emission of radiation.

Principle of Nd-YAG laser- It works on the principle of photo disruption,a infrared beam of 1064nm is produced which concentrates sufficient energy required to produce a plasma effect at a focal point of 11microns which disrupts the tissues adjacent to it acoustically.

COMPLICATIONS OF LASER CAPSULOTOMY

1. IOP elevation- It is a major post-YAG complication. It was estimated that early rise in IOP occurs during the first 3-4hr following laser. Studies proved that IOP returns to its pre treatment level in 89% of cases within 1 week. An IOP rise of up to 30mmHg was seen in

majority of cases. A persistent IOP rise was reported in 1% of cases.

Several thoughts were put forward by certain studies showing that Nd-YAG could cause persistently elevated IOP[57-59].

There are several factors which contribute to the IOP rise which include pre-existing glaucoma, total laser energy used, capsulotomy size, placement of intra-ocular lens.

2.IOL related compllications-These include pitting, cracks and decentration of the IOL.

3.Cystoid macular oedema. It is estimated to occur in patients from 0.5% - 2.5%, because of disruption of anterior hyaloid face, & vitreous which is caused by the laser capsulotomy. An inflammatory response is produced due to the release of capsular debris which releases prostaglandins and leukotrienes[60,61].

4.Retinal detachment, holes and tears

One potential mechanism leading to increased occurrence of retinal complications after capsulotomy was reported by Osterlin in 1971.Opening made to the capsule by any means allows hyaluronic acid to diffuse anteriorly out of vitreous causing vitreous instability which causes posterior vitreous detachment and its complications like retinal tear,retinal detachment and macular hole [62,63]**.**

5.Iritis, **Hyphaema, Endophthalmitis**, **Secondary closure of capsulotomy**

CONTRAINDICATIONS FOR ND-YAG LASER CAPSULOTOMY

A. Absolute

a. Macular opacity / corneal leucoma.

b. Corneal surface disorders.

c. Corneal oedema.

It is quite difficult to see the target well in the aforementioned circumstances, and performing a Nd:YAG capsulotomy leads to inconsistent optical breakdown.

d. Inability to fixate eye steadily.

e. Uncooperative or unwilling patients

B. Relative.

a) Glass Intraocular Lens-Optics of glass IOLs are very prone to fracture.

b) Diagnosed Cystoid Macular Oedema (CME)

c) Eyes with very active inflammation

d) High risk group of patients for rhegmatogenous retinal detachment

– Patients with previous history of rhegmatogenous retinal detachment (RRD)

– Treated prophylactically.

– Myopia.

– Having peripheral retinal degenerations, silent holes, etc

In the a forementioned circumstances, the smallest amount of laser energy per pulse and the fewest number of laser pulses should be used to form a sufficient capsular window or opening for peripheral retinal viewing.

In a descriptive study, Pandey SK et al. examined the parameters that prevent posterior capsular opacification in surgical procedures and IOL selection, as well as the etiopathogenesis of the condition and experimental and clinical investigations that support it [64].

Abay R. Vasavada conducted an analytical investigation to investigate whether posterior subcapsular cataract patients undergoing cataract procedures may have their posterior capsule

plaques predicted prior to surgery and it concluded that 88.6% of the predictions are plausible[65].

The study by Stephen D. McLeod provided detailed information on the lens design and prevention methods as well as the intraoperative risk factors for PCO [66].In-depth information about the etiopathogenesis, risk factors, and therapy of posterior capsular opacification is provided in the paper by Shetal M. Raj et al [67].

The article by IC Lloyd et al. provides a brief overview of the problems associated with managing cataracts in children, particularly the amblyopia that results from posterior capsular opacification. Additionally, it discusses how to manage capsules to prevent this issue[68].The work by T M Aslam et al. elaborates on the techniques for grading posterior capsular opacification by illustrating the vast range of systems of analysis of posterior capsular opacification [69].

The research conducted by Praveen MR et al. compares the evolution of PCO in steroid-treated cataracts to that of other cataracts without steroid treatment. In a one-year follow-up research, it was found that the incidence of post-operative capsular opacification was greater in posterior subcapsular cataracts caused by steroids than in cataracts not caused by steroids[70].

According to K. Hayashi et al.'s study, the kind of intraocular implant is crucial in the development of posterior capsular opacities. When compared to hydrophilic PMMA lenses, hydrophobic intraocular lenses lessen the growth of posterior capsular opacities [71].According to a study by W. R. Meacock et al., the size and kind of intraocular lenses in the capsulorrhexis affect the development of posterior capsular opacity[72].

MATERIALS & METHODS

MATERIAL AND METHODS

SOURCE OF DATA:

A total of 71 patients experiencing diminished vision, who were diagnosed with cataractous lens changes and met the inclusion criteria, were included in this cross-sectional study. These patients underwent surgery and were monitored over a period of 6 months. The study was conducted in the outpatient department of Ophthalmology at R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar, which is affiliated with Sri Devaraj Urs Medical College.

STUDY DESIGN:

Prospective cross sectional observational study

STUDY PERIOD:

September 2022 to December 2023

INCLUSION CRITERIA:

1. Patients of all age groups.
2. All patients with cataract.
3. Patients with history of diabetes.
4. Patients with history of oral steroid intake.

EXCLUSION CRITERIA:

1. Patients with ocular diseases affecting anterior structures such as lens subluxation , ocular trauma, corneal scar and Uveitis

2. Patient with history of retinal vascular disorders such as central retinal vein occlusion (CRVO),branch retinal vein occlusion (BRVO)
3. Poor pupil mydriasis (less than 6 mm)

ETHICAL CLEARANCE:

Before the study began, approval was obtained from the Institutional Ethics and Research Committee of Sri Devaraj Urs Medical College, Kolar.

INFORMED CONSENT:

All patients who met the selection criteria were informed about the nature of the study. Written informed consent was obtained from each participant prior to enrolment (Annexure II and III).

METHOD OF COLLECTION OF DATA

Patients underwent comprehensive assessments utilizing a variety of methods. Initial clinical assessments included detailed history taking, visual acuity evaluation using Snellen's chart for distant vision and Jaeger's chart for near vision, intraocular pressure measurement with Goldmann Applanation tonometer, duct patency assessment, slit lamp bio microscopy for anterior segment examination, and fundus examination via direct and indirect ophthalmoscopy, utilizing a 90 D lens.

Additionally, biochemical parameters such as HbA1c levels, random blood sugar (RBS), fasting blood sugar (FBS), and postprandial blood sugar (PPBS) were measured, along with routine blood investigations. Axial length was measured using A-scan biometry. The study also involved collecting additional patient data, including age, history of diabetes mellitus, cataract grading, history of steroid usage, type of surgery performed, and the position of the intraocular lens during surgery. Furthermore, all patients who underwent cataract surgery

were followed up for 6 months. During these follow-up visits, patients were assessed for the development of PCO. Data collected from these assessments were systematically documented for analysis.

SURGICAL TECHNIQUE:

ANAESTHESIA:

Local anesthesia was used during surgery on adult patients. A peribulbar block was given. To do this, 5 ml of an anesthetic combination containing 2% lignocaine, 1:200000 adrenaline, and 75 units of hyaluronidase had to be given. There were two places for it. 3 ml injected at a depth of 2.5 cm over the meeting point of the lower lid's medial and lateral one third. The remaining 2 milliliters were injected 2.5 centimeters deep, inferomedial to the supraorbital notch. After the block, a little digital massage was given.

General anesthesia was administered to children in the pediatric age range following an anesthesiologist's assessment and clearance of fitness. Anaesthesia via inhalation has been suggested.

PRE OPERATIVE MYDRIASIS:

Phenylephrine (5%) + Tropicamide (1%) eye drops were administered to dilate the pupils. For every case, Flurbiprofen (0.03%) eye drop was also utilized.

SURGERY:

All surgeries were performed by single surgeon only.

The eye was draped under sterile conditions. Topical ocular drops containing 5% betadine were used. Eye speculum inserted and superior rectus bridle suture applied. Two approaches

were used most often when performing surgeries.

1. PCIOL implantation with small incision cataract surgery

2. PCIOL implantation with phacoemulsification.

1. SMALL INCISION CATARACT SURGERY TECHNIQUE:

A superior fornix based conjunctival flap was created. Using 15no blade, a frown shaped scleral incision is made around 5-6mm.

This incision was made around 2 mm from the limbus.

Crescent blade was used to make the sclerocorneal tunnel. A 15 degree lancet blade was used to make a side entry at 9'o clock position and anterior capsule was stained with tryphan blue dye.

Keratome was used to intend the major entering wound.

In each case, a bent 26 G needle was used for the capsulotomy utilizing the continuous curvili near capsulorhexis approach. A balanced salt solution was used for hydrodissection, and it was injected one or two times beneath the anterior lens capsule. The cortex and nucleus are separated by this process. After that, the nucleus was carefully rotated into the anterior chamber and prolapsed into AC using Sinsky's hook.

Subsequently, the nucleus was delivered via the sandwich procedure, which involved the use of vectis and Sinsky's hook or viscoexpression. 22 G Simcoe cannula was then used to administer cortical wash. PCIOL was inserted into the bag after AC was created using visco elastics. In the event that problems prevented in-bag implantation, PCIOL was inserted into the sulcus. After washing the visco elastics, a balanced salt solution side port hydrated to form AC. Gentamycin 20 mg with Dexamethasone 2 mg injection was applied sub conjunctivally after the procedure was done.

PHACOEMULSIFICATION TECHNIQUE:

A corneal incision was made here, using a keratome 2.8 mm corneal incision made and two side ports created. Continuous Curvilinear Capsulorhexis method was used to perform an anterior capsulotomy using a bent 26 G needle.

Hydro delineation and hydro dissection done. Next, using a phaco probe, nucleus trenching was carried out. Chop and Stop was the final technique used. Care was taken to avoid disrupting the anterior hyaloid phase.

Through suction and irrigation, cortical wash done. Foldable PCIOL was inserted into the capsular bag after the AC was filled with visco elastics. Later visco elastics were thoroughly Aspirated With a balanced salt solution side ports hydrated and AC formed. 2 mg of dexamethasone and 20 mg of gentamycin were injected subconjunctively.

POST OPERATIVE ANALYSIS:

Every patient underwent examination during the initial post-operative phase. Snellen's chart was used to record visual acuity. Records were made both with and without a pinhole.

A thorough Slit Lamp examination was performed to check for iridocyclitis, striate keratopathy. Dilated slit lamp examination was performed to examine the posterior capsule in detail and determine the location of the IOL. The patient experienced this every day while they were in the hospital for 7 days, all patients got systemic antibiotics and anti-inflammatory medications in addition to topical antibiotic-steroid drops six times a day. The 45-day course of antibiotic-steroid drops was gradually tapered on weekly basis.

FOLLOW UP:

During the first month all patients were monitored weekly and after that, every month for the

next six months.

Both the BCVA and the UCVA were recorded at each visit. With the pupil dilated, a thorough Slit Lamp Examination was performed to check for symptoms of iridocyclitis, assess the location of the IOL, and thoroughly examine the posterior capsule. Any PCO noted it is graded by Congdon's study. A thorough examination of the fundus was done.Antibiotic steroid drops were given for 45 days with tapering dosage.

STATISTICAL ANALYSIS

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test or Fischer's exact test** (for 2x2 tables only) was used as test of significance for qualitative data.

Continuous data was represented as mean and standard deviation. **Independent t test** was used as test of significance to identify the mean difference between two quantitative variables.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs

P value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data

RESULTS

RESULTS

Table 2: Distribution of subjects according to age group

Age Group	Frequency	Percent
<40	7	9.9
41-60	31	43.7
61-80	31	43.7
>80	2	2.8
Total	71	100

In our study 9 . 9 % of patients were < 40 years, 43.7% patients were in the age range of 41-60 years, 43.7% patients were in the range of 61-80 years, 2.8 % are over > 80 years.

Graph 1: Bar diagram showing Distribution of subjects according to age group

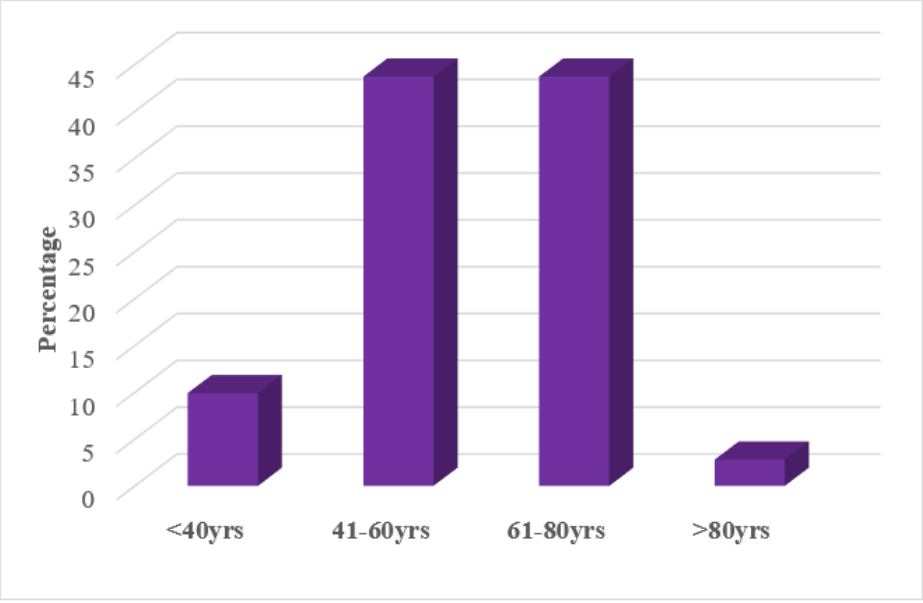


Table 3: Distribution of subjects according to sex

Sex	**Frequency**	**Percent**
Female	43	60.6
Male	28	39.4
Total	71	100

In our study of 71 patients enrolled in the study, 39.4% patients were males and 60.6% were females.

Graph 2: Pie diagram showing Distribution of subjects according to sex

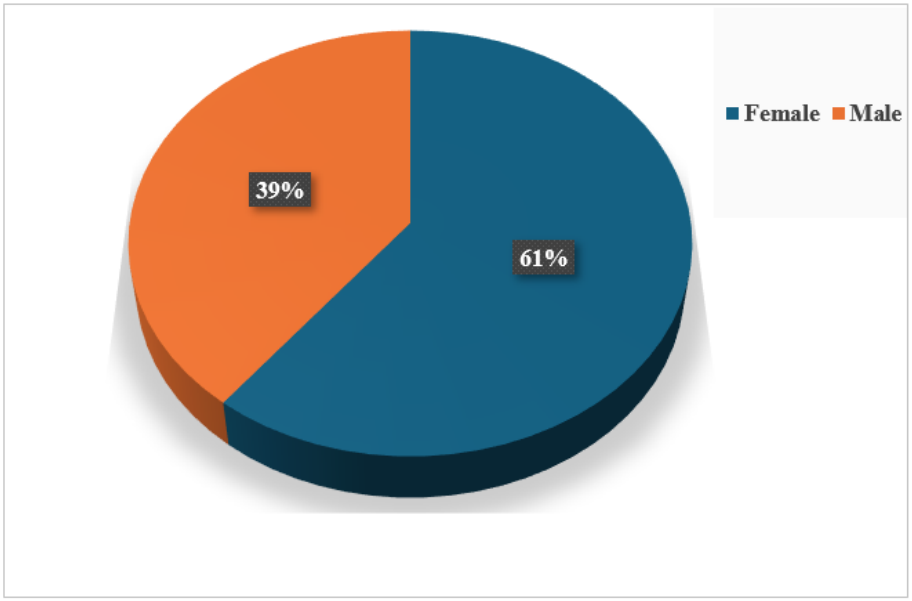


Table 4: Distribution of subjects according to laterality

Laterality	Frequency	Percent
Left	32	45.1
Right	39	54.9
Total	71	100

In our study 54.9% were right eye and 45.1% were left eye.

Graph 3: Pie diagram showing Distribution of subjects according to laterality

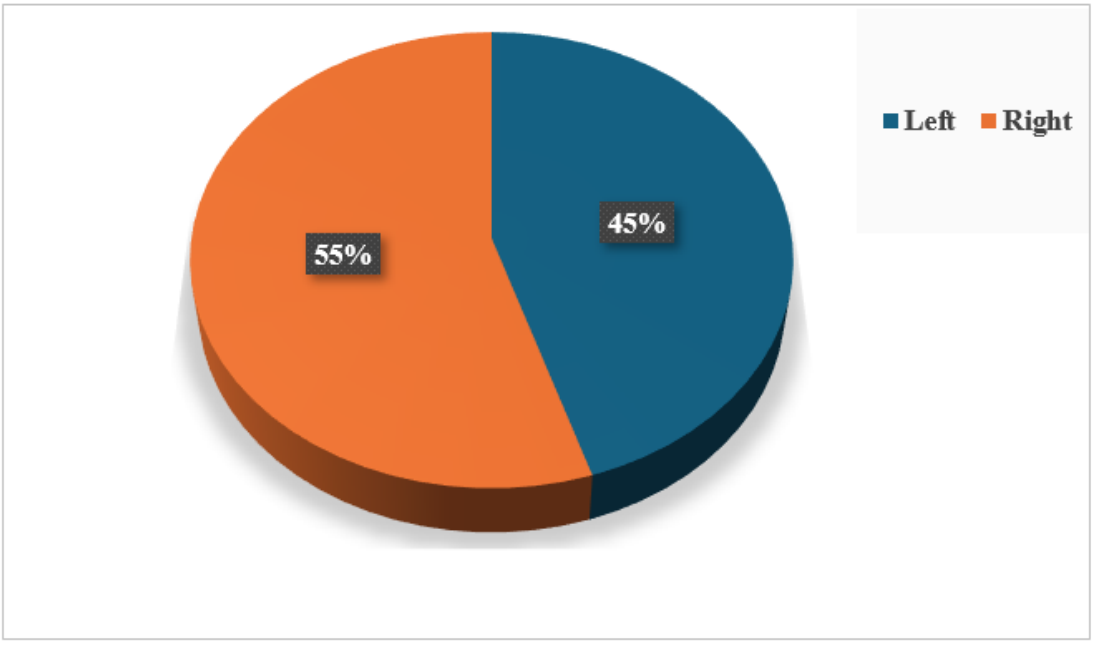


Table 5: Distribution of subjects according to grading of cataract

Type of cataract	Frequency	Percent
Mature	17	23.9
NS	5	7.0
PPC	4	5.6
PSC	45	63.4
Total	71	100

In our study, MATURE cataracts constitute 17 cases (23.9%), NS (Nuclear Sclerotic) cataracts are present in 5 cases (7.0%), and PPC (Posterior Polar Cataract) account for 4 cases (5.6%). The most common type is PSC (Posterior Subcapsular Cataract), with 45 cases (63.4%)

Graph 4: Pie diagram showing Distribution of subjects according to grading of cataract

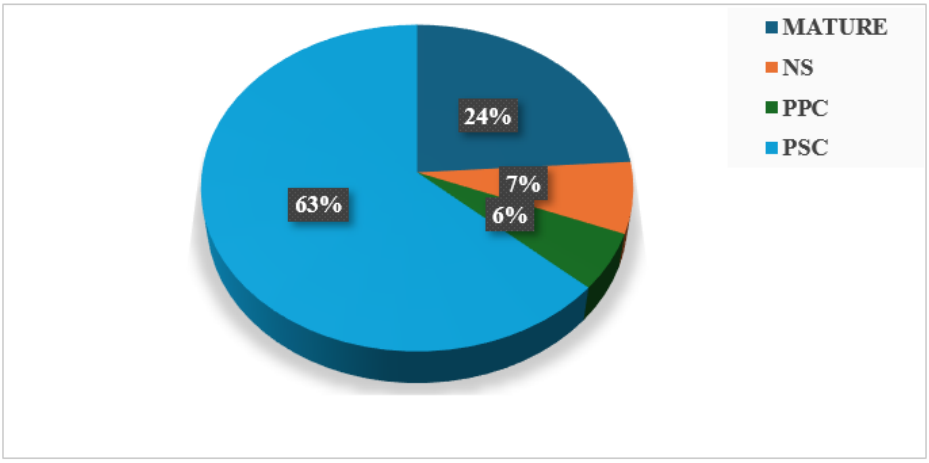


Table 6: Distribution of subjects according to grade of PCO

Grade of PCO	Frequency	Percent
Grade 0	14	19.7
Grade 1	24	33.8
Grade 2	22	31
Grade 3	11	15.5
Total	71	100

In our study on the grade of PCO, Grade 0 was observed in 14 cases (19.7%), Grade 1 in 24 cases (33.8%), Grade 2 in 22 cases (31.0%). Grade 3 in 11 cases (15.5%).

Graph 5: Pie diagram showing Distribution of subjects according to grade of PCO

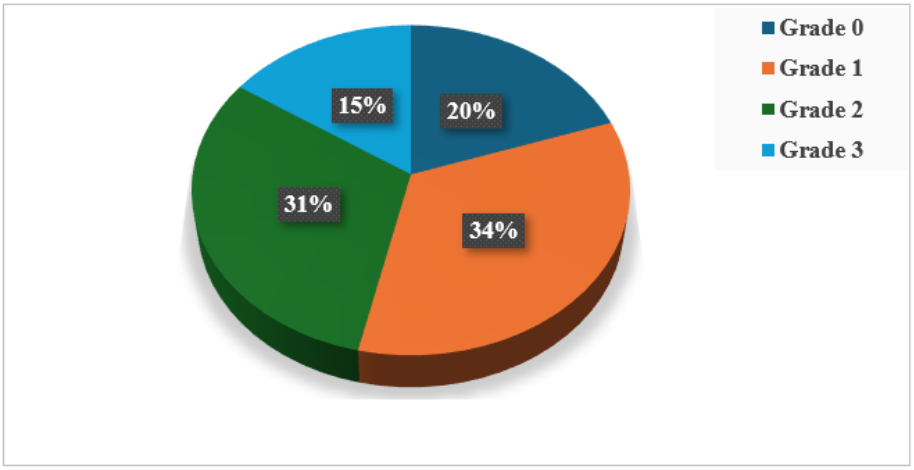
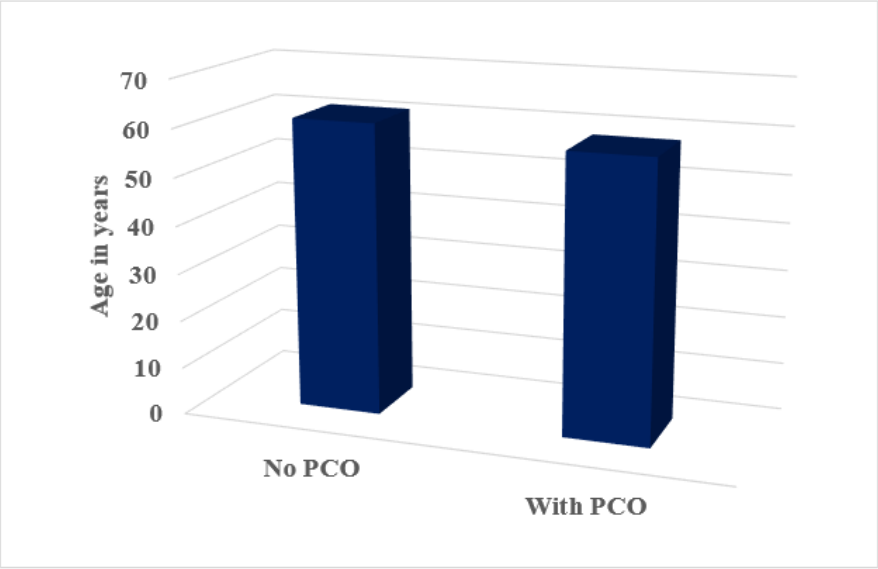


Table 7: comparison of mean age among subjects with PCO and without PCO

	Mean	Std.Deviation	P value
No PCO	61.14	9.734	0.545
With PCO	58.3	16.750	

Graph 6: - Bar diagram showing Comparison of mean age among subjects with PCO and without PCO.



Graph 7: Bar diagram showing Distribution of subjects according to sex and PCO

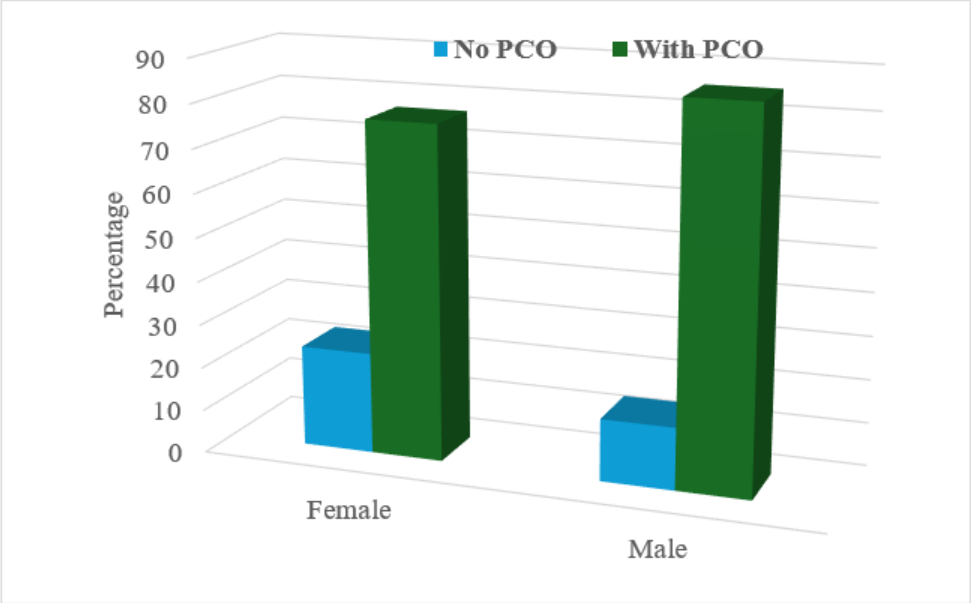


Table 8:- Distribution of subjects according Sex and PCO

Sex	No PCO		PCO	
	N	%	N	%
Female	10	23.3	33	76.7
Male	4	14.3	24	85.7

In our study, subjects based on gender and the presence of PCO. Among females, 10 subjects (23.3%) showed no signs of PCO, while 33 subjects (76.7%) exhibited PCO. Among males, 4 subjects (14.3%) had no PCO, while 24 subjects (85.7%) displayed PCO.

Table 9:- Distribution of subjects according to Diabetes and PCO

Diabetes	No PCO		With PCO	
	N	%	N	%
No	11	19.6 %	45	80.4%
Yes	3	20%	12	80 %

In our study, subjects were categorized based on their diabetes status and the presence of Posterior Capsule Opacification (PCO). Among those without diabetes, 11 subjects (19.6%) did not develop PCO, while 45 subjects (80.4%) did. Conversely, among subjects with diabetes, 3 (20.0%) did not develop PCO, while 12 (80.0%) did.

Figure 8: - Bar diagram showing Distribution of subjects according to diabetes and PCO

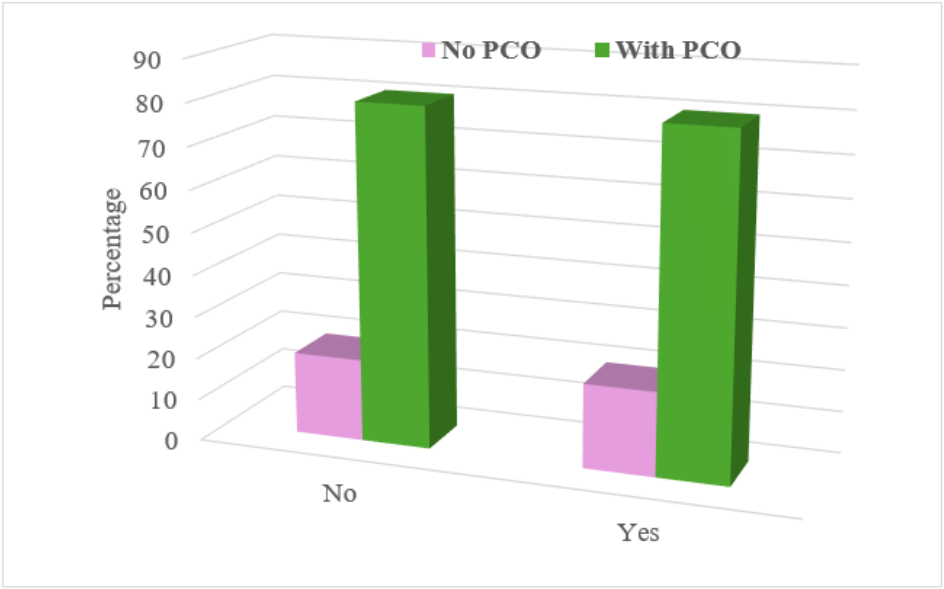


Table 10:- Distribution of subjects according to Steroid intake and PCO

Oral Steroid intake	With out PCO		With PCO	
	N	%	N	%
No	14	22.6	48	77.4
Yes	0	0	9	100

In our study, subjects were studied based on oral steroid intake and the presence of PCO. Among subjects who did not take oral steroids, 14 (22.6%) did not develop PCO, while 48 (77.4%) did. Conversely, among those who took oral steroids, none (0.0%) showed no signs of PCO, while all 9 subjects (100.0%) developed PCO.

Graph 9: Bar diagram showing Distribution of subjects according to steroid intake and PCO

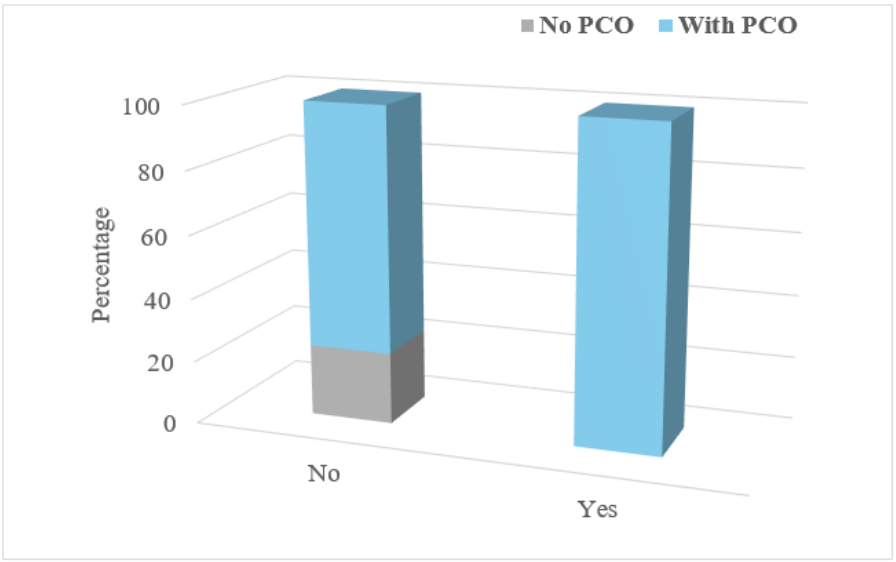


Table 11: Distribution of subjects according to type of cataract and PCO

Type of cataract	With out PCO		With PCO	
	N	%	N	%
Mature	7	41.2	10	58.8
NS	1	20	4	80
PPC	1	25	3	75
PSC	5	11.1	40	88.9

In our study, subjects were categorized according to the type of cataract and the presence of PCO. Among subjects with MATURE cataracts, 7 (41.2%) did not show signs of PCO, while 10 (58.8%) did. For NS cataracts, 1 subject (20.0%) did not exhibit PCO, while 4 subjects (80.0%) did. Similarly, for PPC cataracts, 1 subject (25.0%) did not develop PCO, while 3 subjects (75.0%) did. Lastly, among subjects with PSC cataracts, 5 (11.1%) did not show PCO, while 40 (88.9%) did.

Graph 10: Bar diagram showing Distribution of subjects according to type of cataract and PCO

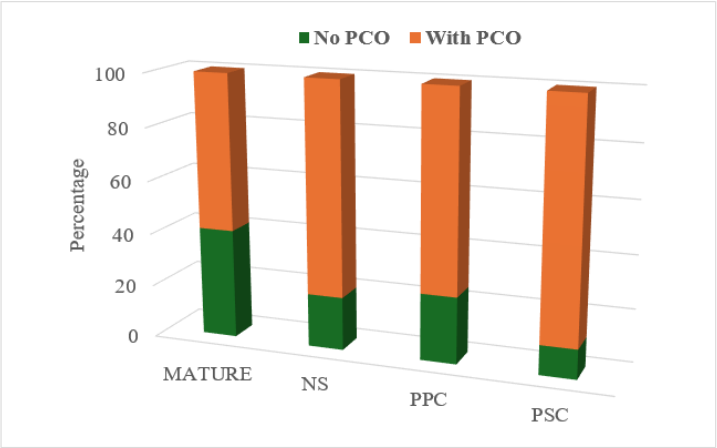


Table 11: Distribution of subjects according to IOL Type and PCO

Type of IOL	With out PCO		With PCO	
	N	%	N	%
Acrylic	0	0	4	100
PMMA	14	20.9	53	79.1

In our study, we examined subjects based on the type of intraocular lens (IOL) used and the presence of PCO. Among subjects implanted with Acrylic IOLs, none (0.0%) showed signs of PCO, while all 4 subjects (100.0%) developed PCO. In contrast, among those with PMMA IOLs, 14 subjects (20.9%) did not exhibit PCO, while 53 subjects (79.1%) did.

Graph 11: Bar diagram showing Distribution of subjects according to IOL Type and PCO

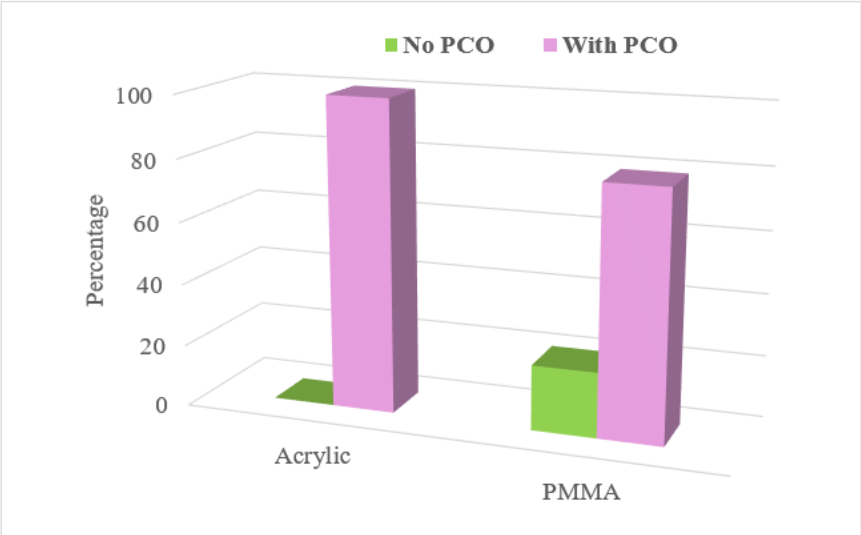


Table 13 : Distribution of subjects according to IOL position and PCO

IOL Position	With out PCO		With PCO	
	N	%	N	%
In Bag	13	21.7	47	78.3
Sulcus	1	9.1	10	90.9

In our study, we categorized subjects according to the position of the intraocular lens (IOL) and the presence of PCO. Among subjects with the IOL placed in the capsular bag, 13 subjects (21.7%) did not exhibit PCO, while 47 subjects (78.3%) did. Conversely, for subjects with the IOL placed in the sulcus, only 1 subject (9.1%) showed no signs of PCO, while 10 subjects (90.9%) developed PCO.

Graph 12: Bar diagram showing Distribution of subjects according to IOL position and PCO

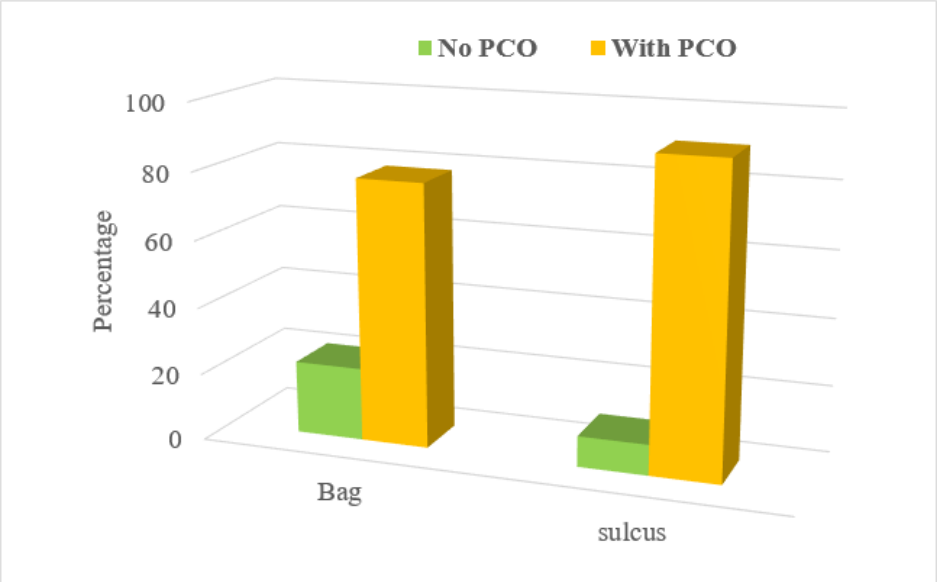


Table 14: Distribution of subjects according to type of surgery and PCO

Surgery type	With out PCO		With PCO	
	N	%	N	%
SICS	14	23.3	46	76.7
Phacoemulsification	0	0	11	100

In our study, we evaluated subjects based on the type of surgery performed and the incidence of PCO. For those who underwent Small Incision Cataract Surgery (SICS), 14 subjects (23.3%) did not develop PCO, while 46 subjects (76.7%) did. In contrast, for subjects who underwent Phacoemulsification, none (0.0%) showed no signs of PCO, while all 11 subjects (100.0%) developed PCO.

Graph 13: Bar diagram showing Distribution of subjects according to type of surgery and PCO

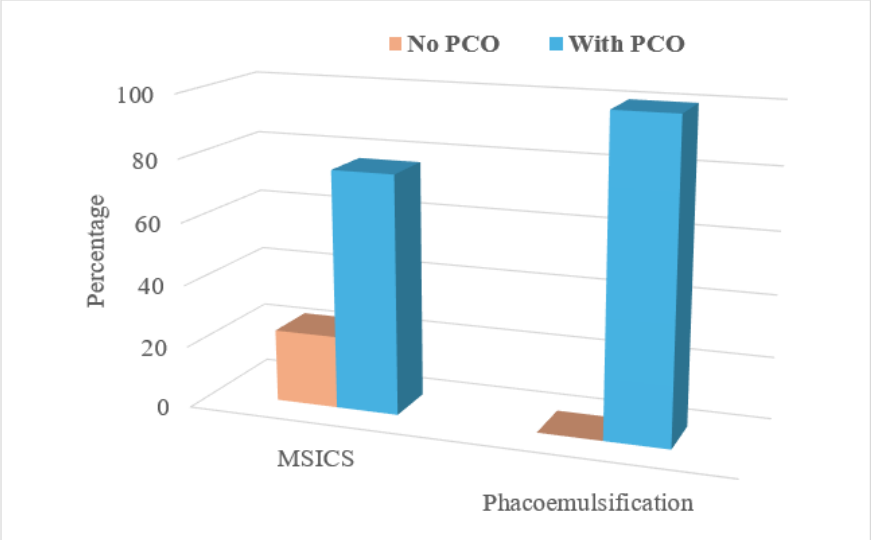
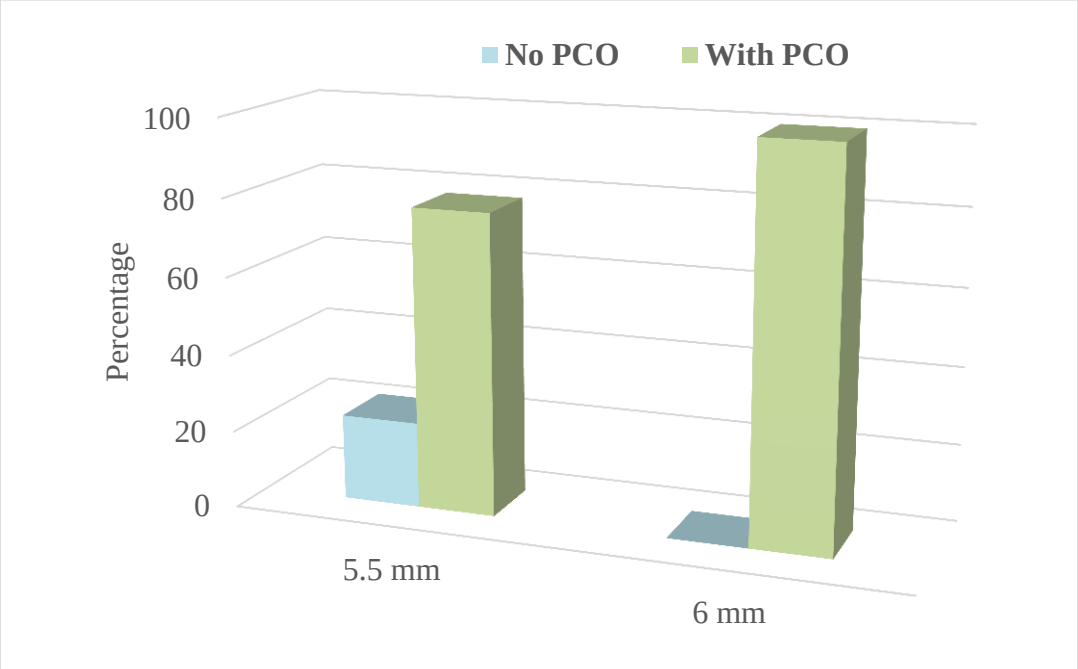


Table14: Distribution of subjects according to capsulorrhexis size and PCO

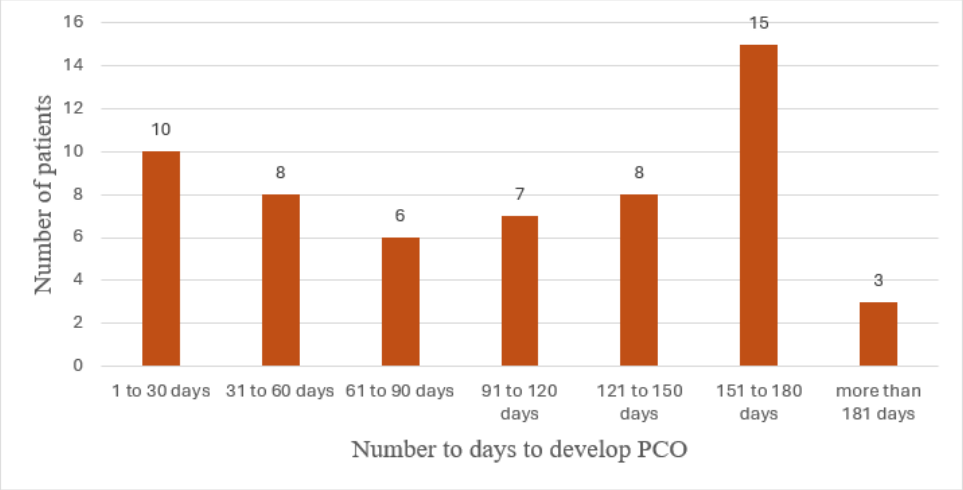
Rhexis size	With out PCO		With PCO	
	N	%	N	%
5.5 mm	6	22.2	21	77.8
6 mm	8	18.2	36	81.8

In our study, we assessed subjects according to the size of the capsulorhexis and the incidence of PCO. For the 5.5 mm capsulorhexis size, 6 subjects (22.2%) showed no signs of PCO, while 21 subjects (77.8%) developed PCO. For the 6 mm capsulorhexis size, 8 subjects (18.2%) had no PCO, while 36 subjects (81.8%) developed PCO.

Graph 14: Bar diagram showing Distribution of subjects according to CAPSULORHEXIS size and PCO



Graph 15: Bar diagram showing Distribution of subjects according to number of days to develop PCO



DISCUSSION

DISCUSSION:

This prospective study, conducted at R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar, aims to identify the preoperative and intraoperative risk variables related to PCO in 71 cases of cataract.

Opacification of the lens proteins is known as a cataract. IOL implantation and cataractous lens extraction have grown increasingly skilled thanks to contemporary advancements in ocular surgery practise. Following surgery, this has aided patients in regaining clear vision. However, the posterior capsule becoming opacified following surgery, which can take from months to years, has become a significant barrier to patients visual rehabilitation.

Patients' quality of life is negatively impacted by PCO development, which substantially decreases their visual acuity and contrast sensitivity. The patient's ability to get around and manage in low light and night is greatly reduced. Nd YAG capsulotomy is a means of treating this posterior capsular opacification. It affects the patient's confidence and adds to their financial stress for them to receive a laser therapy after cataract surgery.

These days, lowering the prevalence of posterior capsular opacification has become important. A number of established risk factors for PCO development have been identified. Age, thick posterior subcapsular cataracts, and cataract in people on long-term steroid consumption are a few of them. The size of the capsulorhexis intraoperatively, the kind of IOL inserted, and the IOL's centration all affect the formation of PCO after surgery. In contrast to rhexis size less than that of optic size, rhexis size greater than that of optic size increases the incidence of PCO. PCO development is more common with PMMA IOLs than hydrophobic acrylic.

Since cataracts are prevalent in all age groups, this study was conducted on participants

ranging in age from 13 to 84. Different trends can be observed in our study based on the age distribution of patients and the incidence of PCO. Three individuals under the age of twenty-one acquired PCO, suggesting a 100% incidence rate, while none of the patients in this age group showed no evidence of the condition. In the same manner, all three patients in the 20–39 age range had PCO, indicating a 100% incidence rate. A considerable percentage of PCO cases were seen in the 40–59 age group, where 6 individuals (21.4%) did not develop PCO and 22 patients (78.6%) did. Of the individuals over 60, 29 patients (78.4%) developed PCO, while 8 patients (21.6%) did not. According to these findings, patients over of younger age have a much higher chance of having PCO.

The findings from Sundelin et al.'s [73] in vitro investigation, indicating a higher incidence of PCO in younger patients, align closely with our clinical study results. our study revealed a markedly elevated incidence of PCO among patients under 40 years old, with all individuals in this age group developing PCO.

Tokko et al.'s [74]study, which shows a correlation between age and the requirement for YAG capsulotomy, confirms our findings that younger patients have a higher risk of PCO. Their finding that those who had PCO were much younger than those who did not supports the idea that age plays a significant role in determining PCO risk and progression.

The combined findings from our study and the study by V. Vinod Mootha et al. [75] underscore the importance of cataract maturity and morphology in predicting the risk of PCO following cataract surgery. The significantly higher frequency of residual capsule opacities in eyes with mature cataracts highlights the need for heightened vigilance and tailored management strategies in this patient population. Moreover, the identification of specific risk factors, such as the severity of posterior subcapsular cataract (PSC) and nuclear sclerosis (NS), provides valuable insights into the factors contributing to PCO development.

Based on evidence of posterior capsule opacification (PCO) and the subjects' diabetes status, we categorised the participants in our study. Of those without diabetes, 80.4% developed PCO and 19.6% did not. On the other hand, of the participants who had diabetes, 80.0% developed PCO and 20.0% did not.

According to research by Ebihara Y et al.[76] and Kiziltoprak H et al.[77], people with diabetes also had a greater incidence of PCO. Specific percentages may differ throughout studies, but overall trends across all three show that patients with diabetes had a higher risk of developing PCO after cataract surgery than people without diabetes.

There is a significant association between oral steroid intake and the risk of PCO after cataract surgery, according to both our study and the study by Mamidipudi R et al[78]. The results of our study showed that subjects who took oral steroids had a higher incidence of PCO than those who did not, and that nearly all of the oral steroids subjects developed PCO. Similarly, at the 1-year follow-up, Mamidipudi R et al. discovered that steroid-induced PSC was linked to an increased risk for PCO.

The significance of taking oral steroid intake into account as a risk factor for PCO development in individuals undergoing cataract surgery is made obvious by these consistent findings. In order to reduce the risk of PCO formation and improve patient outcomes, clinicians should carefully assess the usage of oral steroids and take alternate methods for treatment into consideration.

Based on the type of IOL that each patient had been using and whether or not they had posterior capsule opacification (PCO), we investigated each individual in our study. After receiving an acrylic IOL implant, 0% of the patients displayed any symptoms of PCO, whereas 100% of the four subjects experienced PCO development. 79.1% of patients with PMMA IOLs had PCO, compared to 20.9% who did not.

According to a study by Suresh K. Pandey et al.[79], **Ye Rin Kwon et al** [80] hydrophobic Acrylic IOLs had the lowest PCO generation out of nine distinct rigid and foldable lens designs.

In our study, people were grouped into categories based on the IOL location and whether or not PCO was present. Of the participants whose IOL was inserted into the capsular bag, 47 subjects (78.3%) showed signs of PCO, while 13 subjects (21.7%) did not. On the other hand, of the participants who had the IOL positioned in the sulcus, 10 (90.9%) experienced PCO, while only 1 (9.1%) did not exhibit any of the signs of PCO

Most of the patients in our study had undergone phacoemulsification and SICS after undergoing traditional ECCE. In order to decrease the probability of PCO, the methods used during SICS and phacoemulsification such as cortical cleaving hydro-dissection and bimanual irrigation/aspiration strive to eliminate as many lens fibers and epithelial cells at the capsular bag's equator.

The findings of our investigation are also in line with the reported PCO instances among 150 patients in the study by Moulick et al[81] ,Sahu p et al[82]. By successfully eliminating equatorial lens cells and fibers, the methods outlined in both studies cortical cleaving hydro-dissection and bimanual irrigation/aspiration probably contributed to the reported decrease in PCO incidence.

We analyzed the participants in our study based on the incidence of PCO and the size of the c apsulorhexis.

It was determined that 18.2% of participants with a 6 mm capsulorhexis and 22.2% of those with a 5.5 mm capsulorhexis had no PCO. “In contrast, a research by Langwińska found that 68.18% of patients with a large capsulorhexis had moderate or severe PCO, compared to 86.79% of patients with a minor capsulorhexis who had nil or mild PCO.Wośko E. and

others[6]. PCO was absent or minimal in 89.4% of patients with central capsulorhexis and 75.75% of patients with paracentral capsulorhexis.

Additionally, 86.44% of patients with an anterior capsulorhexis that was consistently rimmed had little or minor PCO, compared to 69.04% of patients with an irregular capsulorhexis rim had moderate or severe PCO [47].

Table15 :Distribution of subjects according to duration to develop PCO

Duration to develop PCO	Number of patients
1 - 30 days	10
31 - 60 days	8
61- 90 days	6
91- 120 days	7
121-150 days	8
151 - 180 days	15
More than 181 days	3

Understanding the evolution of PCO is contingent upon knowing how long it takes to develop after cataract surgery. Our results show that patients' PCO development occurred on a different timescale, starting as early as one month and continuing for almost six months after surgery.

With the highest number of cases reported between 151 and 180 days postoperatively, the distribution shows that a significant proportion of patients acquired PCO within the first six months following surgery. This shows that the first few months after surgery are when PCO

development usually happens, emphasizing the importance of close observation during this time.

The comparatively smaller number of cases that continued past 180 days after surgery would suggest that PCO development has stabilized or that some people had delayed onset. To clarify the long-term course of PCO development and its implications for patient management, additional longitudinal research is necessary.

Clinicians can monitor postoperative patients and take prompt action to treat and stop the course of this common complication by having a better understanding of how long PCO takes to develop.

CONCLUSION

CONCLUSION:

This study emphasizes the importance of preoperative and intraoperative factors in the development of PCO following cataract surgery. Significant research indicates that younger people, particularly those under 40, are more likely to have PCO. Significant pre-operative risk variables such as oral steroid use, diabetes, and cataract grade are uncontrollable; however, intra-operative risk factors such as size of capuslorrhexis, appropriate hydrodissection, and thorough cortical wash are modifiable. According to recent research, placing an IOL in a bag lowers PCO formation when compared to sulus implantation.

57 out of 71 cases had developed PCO within 6 months of time, while out of 10 cases, 2 cases had significant PCO with in short period of 1 month. Previous studies had shown PCO as a late result, but our research demonstrated that it can occur as early as 1 month after surgery.

All things considered, the findings of this study provide useful information to improve patient outcomes after cataract surgery and reduce the occurrence of PCO. Clinicians can apply specific approaches to stop PCO from developing and raise the standards of care for patients undergoing cataract surgery by considering risk factors both before and after surgery.

SUMMARY

SUMMARY:

This study involved 71 patients diagnosed with cataractous lens changes and monitored over six months post-surgery at R. L. Jalappa Hospital and Research Centre. The research aimed to identify preoperative and intraoperative risk factors for PCO after cataract surgery. The findings revealed that younger patients (under 40) had a 100% incidence of PCO, with those aged 40-59 showing a 78.6% incidence, and those over 60 showing a 78.4% incidence. These results align with other studies indicating a higher PCO incidence in younger patients. Furthermore, 80.4% of non-diabetic and 80.0% of diabetic patients developed PCO, corroborating previous research that suggests a higher PCO risk in diabetic patients. Additionally, a higher incidence of PCO was noted in patients taking oral steroids, reinforcing the significance of steroid use as a risk factor.

The study also examined the type of IOL implanted. Patients with non-acrylic IOLs showed a 100% incidence of PCO, whereas those with acrylic IOLs did not develop PCO. This supports findings from other studies that hydrophobic acrylic IOLs have the lowest PCO rates. The position of the IOL also influenced PCO development, with 78.3% of patients having IOLs in the capsular bag and 90.9% with IOLs in the sulcus developing PCO. Surgical techniques like phacoemulsification and SICS were found to be effective in reducing PCO by removing lens fibers and epithelial cells.

Moreover, the size and placement of the capsulorrhexis played a role in PCO incidence. Patients with a 6 mm capsulorhexis had an 18.2% incidence of no PCO, while those with a 5.5 mm capsulorhexis had a 22.2% incidence. A smaller, well-centered capsulorhexis was associated with a lower incidence of PCO. The study also highlighted the timeline of PCO development, which ranged from one month to six months post-surgery, with the highest

number of cases occurring between 151-180 days. These findings underscore the importance of close postoperative monitoring during the first six months.

In our study, 9.9% of patients were under 40 years, 43.7% were aged 41-60 years, 43.7% were aged 61-80 years, and 2.8% were over 80 years. Of the 71 patients, 39.4% were males and 60.6% were females, with 54.9% being right-eye cases and 45.1% left-eye cases. MATURE cataracts constituted 23.9% of the cases, NS cataracts 7.0%, PPC 5.6%, and PSC 63.4%. Regarding PCO grades, 19.7% were Grade 0, 33.8% Grade 1, 31.0% Grade 2, and 15.5% Grade 3.

Among the patients, those without diabetes had an 80.4% incidence of PCO, while diabetic patients had an 80.0% incidence. All patients on oral steroids developed PCO, compared to 77.4% of those not on steroids. For different cataract types, 58.8% with MATURE cataracts, 80.0% with NS cataracts, 75.0% with PPC, and 88.9% with PSC developed PCO. None of the patients with acrylic IOLs developed PCO, whereas 79.1% with PMMA IOLs did. For IOL positions, 78.3% with IOLs in the capsular bag and 90.9% with IOLs in the sulcus developed PCO. All patients who underwent phacoemulsification developed PCO, compared to 76.7% of those who underwent MSICS. Capsulorhexis sizes of 5.5 mm and 6 mm had PCO incidences of 77.8% and 81.8%, respectively.

This study emphasizes the importance of preoperative and intraoperative factors in the development of PCO following cataract surgery. Significant research indicates that younger people, particularly those under 40, are more likely to have PCO. Significant pre-operative risk variables such as oral steroid use, diabetes, and cataract grade are uncontrollable; however, intra-operative risk factors such as size of capsulorhexis, appropriate hydrodissection, and thorough cortical wash are modifiable. According to recent research, placing an IOL in a bag lowers PCO formation compared to sulcus implantation. 57 out of 71

cases developed PCO within six months, with some cases occurring as early as one month post-surgery. Previous studies had shown PCO as a late result, but our research demonstrated that it can occur early. These findings provide useful information to improve patient outcomes after cataract surgery and reduce the occurrence of PCO. Clinicians can apply specific approaches to stop PCO from developing and raise the standards of care for patients undergoing cataract surgery by considering risk factors both before and after surgery.

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ANNEXURE

ANNEXURE I - PATIENT PROFORMA

CASE PROFORMA	
Group:	Case no:
Name:	Date:
Age:	IP no:
Sex:	Date of examination:
Occupation:	

Address:

Chief complaints:

History of Presenting illness:

Past history:

DM/HTN/BA/Epilepsy/TB/BA

Family history:

Personal history:

Appetite– Sleep – Bowel–

Diet– Habits – Bladder –

GPE:

Pallor / Edema /Icterus / Cyanosis / Clubbing / Lymphadenopathy

Vital signs:

a. Pulse– c) RR –

b. BP – d) Temp –

Systemic examination:

a. CVS– c. RS–

b. PA– d. CNS–

OCULAR EXAMINATION		
	RE	**LE**
1. Headposture 2. Ocularposture 3. Facialsymmetry		
4. Ocular movements		
5. VisualAcuity Distant PH Near		
6. Anterior Segment		
7. Fundus (Slit Lamp +90D)		
8 IOP		
9. LS		

VISUAL ACUITY	PREOPERATIVE	POD1	POD7	POD30

INTRAOPERATIVE COMPLICATIONS	

	DAY1	DAY7	DAY30
POSTOPERATIVE COMPLICATIONS			

ANNEXURE - II

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH , TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Case no:

IP no:

TITLE:

ASSESSMENT OF PREOPERATIVE AND INTRAOPERATIVE RISK FACTORS OF POSTERIOR CAPSULAR OPACIFICATION

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of personal information as outlined in this consent form.

I understand the purpose of this study, the risks and benefits of the technique and the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

I have had the opportunity to ask questions regarding the various aspects of this study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw the participation from this study at any time and this will not change the future care.

Participation in this study does not involve any extra cost to me.

Name	Signature	Date	Time
Patient:			
Witness:			
Primary Investigator/ Doctor:			

ANNEXURE - II

ಶ್ರೀದೇವರಾಜ್ಅರಸ್ವೈದ್ಯಕೀಯಕಾಲೇಜು, ಟಮಕ, ಕೋಲಾರ

ತಿಳಿವಳಿಕೆಯಸಮ್ಮತಿನಮೂನೆ

ಕೇಸ್ಸಂಖ್ಯೆ:

ಐಪಿಸಂಖ್ಯೆ:

ಶೀರ್ಷಿಕೆ: ಹಿಂಭಾಗದ ಕ್ಯಾಪ್ಸುಲರ್ ಅಪಾರದರ್ಶಕತೆಯ ಪೂರ್ವಭಾವಿ ಮತ್ತು ಇಂಟ್ರಾಪರೇಟಿವ್ ಅಪಾಯದ ಅಂಶಗಳ ಮೌಲ್ಯಮಾಪನ

ನಾನು, ಅಂಗೀಕರಿಸಿದ,
ಈಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳಲುಮತ್ತುಈಸಮ್ಮತಿಯರೂಪದಲ್ಲಿವಿವರಿಸಿರುವಂತೆವೈಯಕ್ತಿಕಮಾಹಿತಿಯಸಂಗ್ರಹಮತ್ತುಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನುಅನುಮೋದಿಸಲುಒಪ್ಪುತ್ತೇನೆ.

ಈಅಧ್ಯಯನದಉದ್ದೇಶ,
ತಂತ್ರದಅಪಾಯಗಳುಮತ್ತುಪ್ರಯೋಜನಗಳನ್ನುಮತ್ತುಅಧ್ಯಯನದಸಮಯದಲ್ಲಿಸಂಗ್ರಹಿಸಲ್ಪಟ್ಟಿರುವಮತ್ತುಬಹಿರಂಗಪಡಿಸುವಮಾಹಿತಿಯಗೌಪ್ಯತೆಗೆನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.
ಸಂಗ್ರಹಿಸಿದಮಾಹಿತಿಯನ್ನುಸಂಶೋಧನೆಗೆಮಾತ್ರಬಳಸಲಾಗುತ್ತದೆ.

ಈಅಧ್ಯಯನದವಿವಿಧಅಂಶಗಳನ್ನುಕುರಿತುಪ್ರಶ್ನೆಗಳನ್ನುಕೇಳಲುನನಗೆಅವಕಾಶವಿದೆಮತ್ತುನನ್ನತೃಪ್ತಿಗೆನನ್ನಪ್ರಶ್ನೆಗಳಿಗೆಉತ್ತರನೀಡಲಾಗಿದೆ.

ಈಅಧ್ಯಯನದಿಂದಭಾಗವಹಿಸುವಿಕೆಯನ್ನುಯಾವುದೇಸಮಯದಲ್ಲಿನಾನುಹಿಂಪಡೆಯಲುಮುಕ್ತವಾಗಿರುತ್ತೇನೆಮತ್ತು ಇದುಭವಿಷ್ಯದಕಾಳಜಿಯನ್ನುಬದಲಿಸುವುದಿಲ್ಲಎಂದುನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸುವಿಕೆನನಗೆಯಾವುದೇಹೆಚ್ಚುವರಿವೆಚ್ಚಒಳಗೊಳ್ಳುವುದಿಲ್ಲ.

ಹೆಸರು	ಸಹಿ/ಹೆಬ್ಬೆಟ್ಟಿನಗುರುತು	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿಯಹೆಸರು			
ಸಾಕ್ಷಿಗಳಹೆಸರು			
ಪ್ರಾಥಮಿಕಸಂಶೋಧಕರು/ ವೈದ್ಯರು			

ANNEXURE – III
SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.
PATIENT INFORMATION SHEET

TITLE: "**ASSESSMENT OF PREOPERATIVE AND INTRAOPERATIVE RISK FACTORS OF POSTERIOR CAPSULAR OPACIFICATION**".

This information is to help you understand the purpose of the study. You are invited to take part voluntarily in this research study, it is important that you read and understand the purpose, procedure, benefits and discomforts of the study.

The purpose of this study is to find out the risk factor associated with the development of posterior capsular opacification. There is absolutely no risks associated with various investigations involved in this study such as Visual acuity, Subjective refraction, slit lamp examination etc.

Participation in this research study may not change the final outcome of your eye condition. However, patients in the future may benefit as a result of knowledge gained from this study. You will not be charged extra for any of the procedures performed during the research study. Your taking part in this study is entirely voluntary.

You may refuse to take part in the study or you may stop your participation in the study at any time, without any penalty or loss of any benefits to which you were otherwise entitled before taking part in this study.

CONFIDENTIALITY

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. Your original records may be reviewed by your doctor or ethics review board. For further information, clarification please contact below mentioned, SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR – 563101

CONTACT DETAILS:

DR. HASAREEN SHAIK, MBBS, (MS)

1st YEAR RESIDENT

DEPARTMENT OF OPHTHALMOLOGY,

SDUMC, KOLAR – 563101

Contact no: 8463976061

ANNEXURE – III

ಶ್ರೀದೇವರಾಜ್ಅರಸ್ಉನ್ನತಶಿಕ್ಷಣಮತ್ತುಸಂಶೋಧನಾಸಂಸ್ಥೆ,

ಟಮಕ, ಕೋಲಾರ - 563101.

ರೋಗಿಯಮಾಹಿತಿಪತ್ರ

ಈ ಮಾಹಿತಿಯು " ಹಿಂಭಾಗದ ಕ್ಯಾಪ್ಸುಲರ್ ಅಪಾರದರ್ಶಕತೆಯ ಪೂರ್ವಭಾವಿ ಮತ್ತು ಇಂಟ್ರಾಪರೇಟಿವ್ ಅಪಾಯದ ಅಂಶಗಳ ಮೌಲ್ಯಮಾಪನ".

ಈ ಮಾಹಿತಿಯು ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳಲು ನಿಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ. ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಪಾಲ್ಗೊಳ್ಳಲು ನಿಮ್ಮನ್ನು ಆಹ್ವಾನಿಸಲಾಗಿದೆ, ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಹಿಂಭಾಗದ ಕ್ಯಾಪ್ಸುಲರ್ ಅಪಾರದರ್ಶಕತೆಯ ಬೆಳವಣಿಗೆಗೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯಕಾರಿ ಅಂಶವನ್ನು ಕಂಡುಹಿಡಿಯುವುದು.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಒಳಗೊಂಡಿರುವ ವಿವಿಧ ತನಿಖೆಗಳೊಂದಿಗೆ ಸಂಪೂರ್ಣವಾಗಿ ಯಾವುದೇ ಅಪಾಯಗಳಿಲ್ಲ ದೃಷ್ಟಿ ತೀಕ್ಷ್ಣತೆ, ವಸ್ತುನಿಷ್ಠ ವಕ್ರೀಭವನ, ಸ್ಲಿಟ್ ಲ್ಯಾಂಪ್ ಪರೀಕ್ಷೆ.

ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆಯು ನಿಮ್ಮ ಕಣ್ಣಿನ ಸ್ಥಿತಿಯ ಅಂತಿಮ ಫಲಿತಾಂಶವನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ. ಆದಾಗ್ಯೂ, ಈ ಅಧ್ಯಯನದಿಂದ ಪಡೆದ ಜ್ಞಾನದ ಪರಿಣಾಮವಾಗಿ ಭವಿಷ್ಯದಲ್ಲಿ ರೋಗಿಗಳು ಪ್ರಯೋಜನ ಪಡೆಯಬಹುದು. ಸಂಶೋಧನಾ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಡೆಸಿದ ಯಾವುದೇ ಕಾರ್ಯವಿಧಾನಗಳಿಗೆ ನಿಮಗೆ ಹೆಚ್ಚುವರಿ ಶುಲ್ಕ ವಿಧಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನೀವು ಭಾಗವಹಿಸುವುದು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ.

ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಮೊದಲು ನೀವು ಅರ್ಹರಾಗಿದ್ದ ಯಾವುದೇ ಪ್ರಯೋಜನಗಳ ಯಾವುದೇ ದಂಡ ಅಥವಾ ನಷ್ಟವಿಲ್ಲದೆಯೇ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಗೌಪ್ಯತೆ

ನಿಮ್ಮ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯನ್ನು ಅಧ್ಯಯನ ವೈದ್ಯರು ಮತ್ತು ಸಿಬ್ಬಂದಿ ಗೌಪ್ಯವಾಗಿಡುತ್ತಾರೆ ಮತ್ತು ಸಾರ್ವಜನಿಕವಾಗಿ ಲಭ್ಯವಾಗುವಂತೆ ಮಾಡಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮೂಲ ದಾಖಲೆಗಳನ್ನು ನಿಮ್ಮ ವೈದ್ಯರು ಅಥವಾ ಎಥಿಕ್ಸ್ ರಿವ್ಯೂ ಬೋರ್ಡ್ ಪರಿಶೀಲಿಸಬಹುದು. ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ, / ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ದಯವಿಟ್ಟು ಶ್ರೀ ದೇವರಾಜ್ ಯುಆರ್ಎಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನೆ ಅಕಾಡೆಮಿ, ತಮಕ, ಕೋಲಾರ - 563101 ಅನ್ನು ಸಂಪರ್ಕಿಸಿ.

ಸಂಪರ್ಕ ವಿವರಗಳು:

DR. ಹಸರೀನ್ ಶೇಕ್, MBBS, (MS)

1 ನೇ ವರ್ಷದ ನಿವಾಸಿ

ನೇತ್ರವಿಜ್ಞಾನ ವಿಭಾಗ,

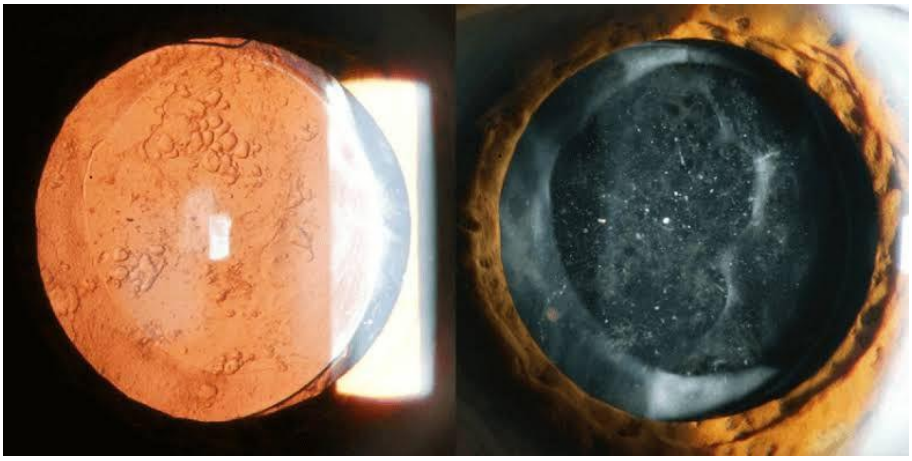
SDUMC, ಕೋಲಾರ - 563101

ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 8463976061

ANNEXURE-IV

Photograph 4: slit lamp examination

Photograph 5: Indirect ophthalmoscope examination



Photograph 6: Posterior capsular opacification

MASTER CHART

KEY TO MASTER CHART

M:MALE

F: FEMALE

RE:RIGHT EYE

LE:LEFT EYE

SHMC: SENILE HYPERMATURE CATARACT

SMC: SENILE MATURE CATARACT

NS:NUCLEAR SCLEROSIS

PPC:POSTERIOR POLAR CATARACT

PSC:POSTERIOR SUBCAPSULAR CATARACT

PCIOL:POSTERIOR CHAMBER INTRA OCULAR LENS

PMMA:POLYMETHYLMETHACRYLATE

SICS:SMALL INCISION CATARACT SURGERY

					PRE OPERATIVE RISK FACTORS			INTRA OPERATIVE RISK FACTORS						
S.NO	UHID	AGE	GENDER	LATERALITY	DIABETES	STEROID INTAKE	CATARACT GRADING	IOL TYPE	IOL POSITION	TYPE OF SURGERY	CAPSULORRHEXIS SIZE	CORTICAL WASH	DURATION IN DAYS	GRADE OF PCO
1	142289	65	M	RE	NO	NO	PSC+PPC+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient		GRADE 0
2	223203	54	F	RE	Yes	NO	PSC+NS 3+ CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	12	GRADE 1
3	242489	53	M	LE	NO	YES	PSC+NS 1+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	30	GRADE 1
4	246512	55	M	LE	NO	NO	PSC+NS 3+ CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	20	GRADE 1
5	281893	67	F	RE	yes	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	26	GRADE 1
6	113296	60	F	LE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient		GRADE 0
7	113293	65	F	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	178	GRADE 1
8	286795	76	M	LE	NO	NO	SMC	PMMA, PCIOL	Sulcus	SICS	5.5 mm	sufficient	30	GRADE 1
9	222266	48	F	RE	NO	NO	Presenile Mature cataract	PMMA, PCIOL	Bag	phacoemulsification	5.5 mm	sufficient	25	GRADE 2
10	242477	13	F	LE	NO	NO	NS 1+Cortical	PMMA, PCIOL	Bag	phacoemulsification	5.5 mm	sufficient	40	GRADE 2
11	236711	65	F	RE	NO	YES	PSC+NS 3+CORTICAL Cataract	PMMA, PCIOL	Bag	phacoemulsification	5.5 mm	sufficient	60	GRADE 2
12	209434	48	F	LE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	phacoemulsification	6 mm	sufficient	60	GRADE 2
13	233809	73	M	LE	NO	NO	SHMC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0
14	241047	66	F	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	phacoemulsification	6 mm	sufficient	25	GRADE 2
15	216765	22	M	RE	yes	NO	PSC+NS 3+CORTICAL Cataract	Acrylic, PCIOL	Bag	phacoemulsification	5.5 mm	sufficient	45	GRADE 2
16	157331	70	F	RE	yes	NO	PSC+NS 3	Acrylic, PCIOL	Bag	phacoemulsification	5.5 mm	sufficient	45	GRADE 2
17	196480	55	M	LE	yes	NO	PSC+NS 2	Acrylic, PCIOL	Bag	SICS	5.5 mm	sufficient	30	GRADE 3
18	294118	55	F	RE	NO	YES	NS 2 + CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	180	GRADE 3
19	284012	75	M	LE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	30	GRADE 3
20	89872	77	M	LE	NO	YES	PSC+NS 3+ CORTICAL Cataract	Acrylic, PCIOL	Bag	SICS	5.5 mm	sufficient	180	GRADE 3
21	275183	78	M	RE	NO	YES	PSC+NS 2 +CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	60	GRADE 1
22	244392	51	F	RE	NO	NO	SHMC	PMMA, PCIOL	sulcus	SICS	6 mm	sufficient	180	GRADE 3
23	150626	65	F	RE	NO	NO	SHMC	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient		GRADE 0
24	138774	45	M	LE	NO	NO	Presenile Hyper mature Cataract	PMMA, PCIOL	sulcus	SICS	5.5 mm	sufficient	80	GRADE 2
25	180919	59	F	LE	NO	NO	PSC+NS 2 +CORTICAL Cataract	PMMA, PCIOL	Sulcus	SICS	5.5 mm	sufficient		GRADE 0

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26	150617	77	F	LE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	sulcus	Phacoemulsification	5.5 mm	sufficient	150	GRADE 3
27	145023	84	M	LE	NO	NO	SHMC	PMMA, PCIOL	sulcus	Phacoemulsification	6 mm	sufficient	120	GRADE 1
28	145020	75	M	RE	NO	NO	PSC+NS 3+CORTICAL Cataract	PMMA, PCIOL	sulcus	Phacoemulsification	6 mm	sufficient	140	GRADE 2
29	145008	55	F	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	sulcus	Phacoemulsification	6 mm	sufficient	125	GRADE 3
30	163936	60	F	LE	NO	NO	PSC+NS 1+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0
31	163930	72	F	LE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	sulcus	SICS	6 mm	sufficient	80	GRADE 1
32	185820	60	M	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	165	GRADE 2
33	185812	66	M	RE	NO	YES	PSC+NS 1+ CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	145	GRADE 1
34	180922	68	F	RE	yes	NO	SMC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	180	GRADE 1
35	185824	60	F	LE	NO	NO	SMC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	185	GRADE 2
36	180924	77	M	RE	NO	NO	PSC+NS 3+CORTICAL Cataract	PMMA, PCIOL	sulcus	SICS	6 mm	sufficient	90	GRADE 2
37	200163	76	F	LE	NO	NO	PSC+NS 2	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	182	GRADE 1
38	190991	67	M	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	100	GRADE 3
39	209638	75	M	LE	NO	NO	PSC+NS 3+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0
40	209636	58	F	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	137	GRADE 2
41	190972	15	F	LE	NO	NO	NS 3	PMMA, PCIOL	sulcus	SICS	6 mm	sufficient	60	GRADE 3
42	233803	44	F	LE	NO	YES	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	100	GRADE 3
43	235345	65	F	LE	NO	NO	PSC+NS 2	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	180	GRADE 2
44	235349	58	F	LE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	20	GRADE 2
45	235328	67	M	RE	yes	NO	NS 4	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	179	GRADE 1
46	242509	50	M	RE	yes	NO	PSC+NS 3+CORTICAL cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	126	GRADE 2
47	244382	83	M	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	180	GRADE 3
48	244393	50	F	RE	NO	NO	SMC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0
49	244392	51	F	LE	NO	YES	SMC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	175	GRADE 1
50	246783	55	F	RE	NO	NO	PSC+NS 1+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	160	GRADE 2
51	246777	40	F	LE	NO	NO	PSC+NS 1+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	120	GRADE 1

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52	380685	54	M	RE	yes	NO	PSC+NS 1+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	90	GRADE 1
53	381632	69	F	LE	yes	NO	PSC+NS 1+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient	185	GRADE 2
54	244384	18	M	RE	NO	YES	PPC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	80	GRADE 2
55	142289	65	M	RE	NO	NO	PPC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	148	GRADE 1
56	153777	36	F	LE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	84	GRADE 2
57	145020	75	M	LE	NO	NO	PSC+NS 3+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	156	GRADE 1
58	153772	74	F	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	168	GRADE 1
59	153769	70	F	RE	yes	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	180	GRADE 2
60	158100	43	F	RE	NO	NO	Presenile Mature Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	96	GRADE 2
61	158130	77	F	LE	NO	NO	PSC+NS 1+ CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	120	GRADE 1
62	151477	70	M	RE	NO	NO	SMC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0
63	161475	35	F	RE	NO	NO	Presenile Mature Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	120	GRADE 1
64	163938	42	F	LE	NO	NO	PPC	PMMA, PCIOL	Bag	SICS	5.5 mm	Insufficient	143	GRADE 1
65	163928	74	F	RE	NO	NO	NS 4	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0
66	177683	48	M	RE	yes	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient	178	GRADE 1
67	174116	54	F	LE	yes	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient		GRADE 0
68	184944	58	F	LE	NO	NO	SHMC	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0
69	195724	56	M	RE	NO	NO	PSC+NS 2+CORTICAL Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	In sufficient	45	GRADE 1
70	186196	48	F	RE	yes	NO	Presenile Mature Cataract	PMMA, PCIOL	Bag	SICS	5.5 mm	sufficient		GRADE 0
71	235329	45	F	RE	yes	NO	Presenile Mature Cataract	PMMA, PCIOL	Bag	SICS	6 mm	sufficient		GRADE 0