A DISSERTATION ON

# "A STUDY ON THE VISUAL OUTCOME IN PATIENTS WITH LENS INDUCED GLAUCOMA FOLLOWING SMALL INCISION CATARACT SURGERY"

Submitted to

**THE TAMIL NADU DR. M. G. R. MEDICAL UNIVERSITY**

In partial fulfilment of the requirements

For the award of degree of

**M.S. ( Branch III ) --- OPHTHALMOLOGY**


**GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI, TAMIL NADU**

**MAY 2020**

## CERTIFICATE

This is to certify that the study entitled **"A STUDY ON THE VISUAL OUTCOME IN PATIENTS WITH LENS INDUCED GLAUCOMA FOLLOWING SMALL INCISION CATARACT SURGERY"** is the result of original work carried out by **DR. PANIMALAR R**, under my supervision and guidance at **GOVERNMENT STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of M.S Degree in Ophthalmology, course from 2017 to 2020 at Government Stanley Medical College, Chennai.

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## DECLARATION

I hereby declare that this dissertation entitled **"A STUDY ON THE VISUAL OUTCOME IN PATIENTS WITH LENS INDUCED GLAUCOMA FOLLOWING SMALL INCISION CATARACT SURGERY"** is a bonafide and genuine research work carried out by me under the guidance of **PROF.DR.THANGERANI RAAJASEHARAN,M.S.,D.O.,** Unit chief and Head of the Department, Department of Ophthalmology, Government Stanley Medical college and Hospital, Chennai – 600001.

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## ACKNOWLEDGEMENT

I express my immense gratitude to The **Dean, Prof. Dr. SHANTHIMALAR, M.D., D.A.,** Government Stanley Medical College for giving me the opportunity to work on this study.

I wish to express my heartfelt and profound indebtness to my teacher and guide **Prof. Dr. THANGERANI RAAJASEHARAN,M.S.,D.O.,** Head of Department, Department of Ophthalmology, Government Stanley Medical College, Chennai-01, for her invaluable feedback, suggestions and guidance throughout the completion of the study.

I would like to thank **Prof. Dr. K. VINAYAGAMURTHY M.S.,** for his valuable guidance and suggestions for the study.

I am grateful to **Prof. Dr. M.S. GOKILA., M.S., D.O.,** for her timely support and guidance.

I would like to thank **Prof.DR K S T LATHA, M.S.,** for her valuable guidance and suggestions for the study.

My heartfelt thanks to my teachers and Assistant Professors, **Dr. S.VENKATESH M.S., DR.SIVAKUMAR MS., Dr. VINOTHINI M.S., D.O.,** for their supervision and assistance during the progression of my study.

I specially thank My Guide **Dr S.HEMAPRIYA DNB., DO.,** for the valuable suggestion and supervision throughout my thesis work.

My sincere thanks to **Mr T.Maria Jusler Kalsingh MBE.,** for his timely help with the statistics.

My thanks to all my colleagues, seniors and juniors for their support.

My special thanks to all the patients, for their cooperation and patience shown during the study without whom this study would not have been complete.

No words can express my sincere gratitude to my husband, parents and my brother for their encouragement, motivation and timely help during the course of this study.

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**INSTITUTIONAL ETHICS COMMITTEE**

Title of the Work : A STUDY ON VISUAL OUTCOME IN PATIENTS WITH LENS INDUCED GLAUCOMA FOLLOWING SMALL INCISION CATARACT SURGERY

Principal Investigator : DR. R.PANIMALAR,
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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 21.12.2017 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

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## TABLE OF CONTENTS

## LIST OF ABBREVIATIONS

| | |
|---|---|
| ❖ AC | Anterior Chamber |
| ❖ ACIOL | Anterior Chamber Intra Ocular Lens |
| ❖ BCVA | Best Corrected Visual Acuity |
| ❖ IOP | Intra Ocular Pressure |
| ❖ K.P's | Keratic Precipitates |
| ❖ LA | Local Anaesthesia |
| ❖ LIG | Lens Induced Glaucoma |
| ❖ mmHg | millimetres of Mercury |
| ❖ PCIOL | Posterior Chamber Intra Ocular Lens |
| ❖ PCR | Posterior Capsular Rent |
| ❖ POD | Post Operative Day |
| ❖ SFIOL | Scleral Fixation Intra Ocular Lens |
| ❖ SICS | Small Incision Cataract Surgery |
| ❖ VL | Vitreous Loss |

# INTRODUCTION

The human crystalline lens is a transparent, biconvex intraocular structure. It is present in the anterior segment of the eye and is suspended radially at its equator by zonular fibres to the ciliary body, between the iris and vitreous body. A capsular bag encloses the lens which is present in the patellar fossa.

In India, there is a backlog of 12 million(1) cases of cataract annually, and the disease bureden is increasing at a rate of 3.8 million (2)With such large number of cases treated with intraocular lens, lens induced glaucoma(LIG) is widely prevalent among the Indian population(3,4).

In the developing nations, one of the leading causes of secondary glaucoma is LIG due to hyper mature cataract. A steady improvement in life expectancy rate and population explosion, increase the backlog of untreated cataract. Adding to the burden, there is a low productivity due to the underutilization of surgical services. This is more pronounced in the rural population where the eye care services are less availed.

LIG increases the pre-operative and post-operative morbidity compared to simple uncomplicated cataract. Irrespective of the surgical intervention visual outcome are affected to some extent and the prognosis guarded. Hence timely intervention is important in achieving good results.

The management of each patient varies according to the type of LIG. It begins with identifying the underlying cause and followed by control of intraocular pressure. Once these goals are achieved, it is preferable to perform the extraction of cataract with intraocular lens (IOL) implantation.

## NEED FOR STUDY

The importance of patient education, early diagnosis of cataract, and treatment of all cases of cataract before they go for complications necessitates the need for this study.

This study was undertaken to emphasis that the timely intervention in cases of lens induced glaucoma could yield a good vision to the patients thereby improving their quality of life.

Hence this study was designed to study the various clinical aspects of LIG, the approach towards the patients, the appropriate management, post operative results analyzing the visual outcome and intraocular pressure control as practiced in the department of Ophthalmology, Government Stanley Medical College Hospital, Chennai.

## CRYSTALLINE LENS:

## ANATOMY, PHYSIOLOGY AND BIOCHEMICAL ASPECTS:

The lens is a transparent structure which alters the refraction of light entering the eye. The lens is an oblate spheroid structure. It is avascular and lacks nerves and connective tissue. It is located posterior to the iris with its anterior surface in contact with the aqueous and the posterior surface in contact with the vitreous in the patellar fossa. Berger described that the lens is separated from the hyaloid membrane by a slit like space called as Berger's space filled with primitive vitreous.

The lens is suspended by the zonular fibers. They arise from the ciliary epithelium and insert 1–2 μm into the capsule at its outer part.

## EMBRYOLOGY:

The crystalline lens is ectodermal in origin. During the seventh week of intrauterine life, the cells of the surface ectoderm, initially in contact with the optic vesicle, invaginate to form the lens placode and the lens vesicle (Figure 1). It consists of a single layer of cells covered by basal lamina. The lens develops by formation of primary and secondary lens fibers that are derived from the lens epithelium. The lens capsule is a membranous non cellular envelope that surrounds the lens. At birth, the lens is approximately 6 mm in diameter and continues to grow throughout life.

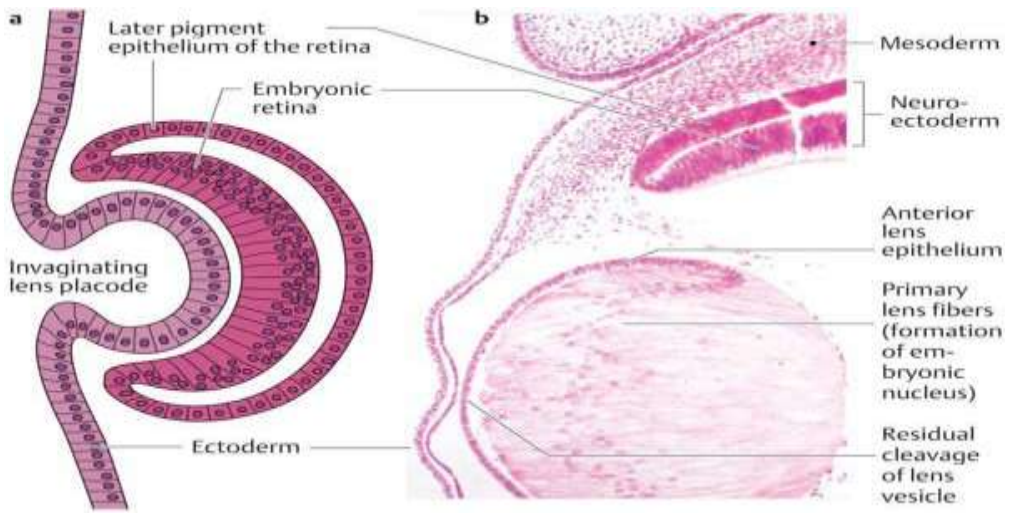



**FIGURE 1: FIRST MONTH OF FETAL DEVELOPMENT:**

a) The ectoderm invaginates and is isolated in what becomes the opticcup.

b) The lens vesicle is completely invaginated.

Histologically the lens consists of three major components: the capsule, epithelium, and lens substance.

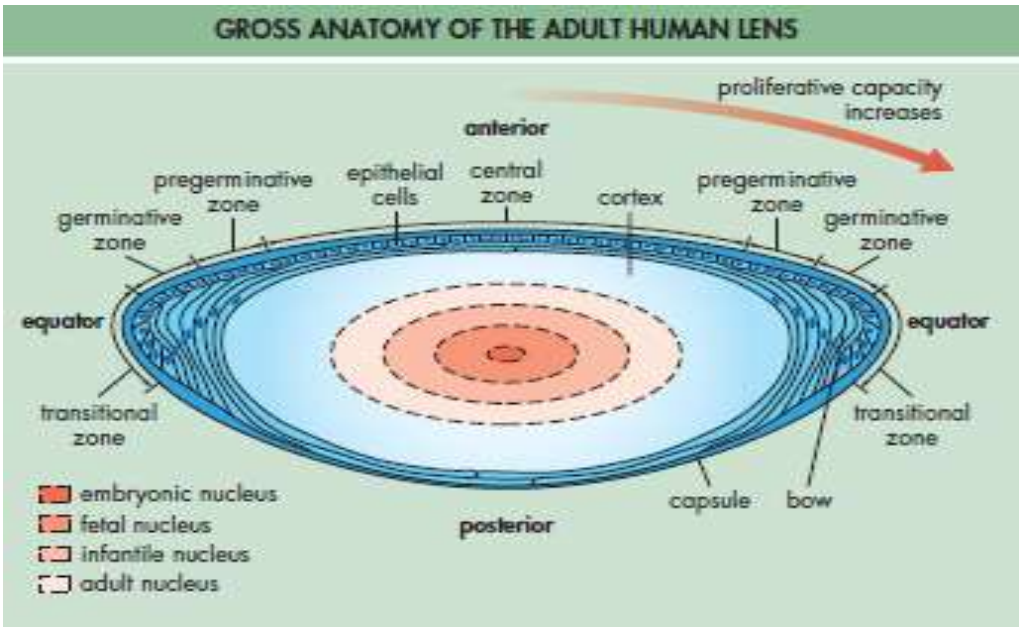



**FIGURE 2: GROSS ANATOMY OF HUMAN CRYSTALLINE LENS**

## CAPSULE:

The lens capsule is an acellular envelope that is continuously synthesized by the lens epithelium anteriorly and fiber cells posteriorly. It is true basement membrane formed by basal laminae material deposited by lens epithelium. It is the thickest basement membrane of the body. It is composed of a number of stacked lamellae which contain major structural proteins, type IV collagen and fibronectin.

The lens epithelium is a single layer of cuboidal cells approximately 10 μm high and 15 μm wide. It is located beneath the anterior capsule that extends to the equatorial lens bow. Their basal surface adheres to the capsule, whereas their anterior surface abuts the newly formed elongating lens fibers. The proliferative capacity of epithelial cells is greatest at the equator. The cells in the germinative zone are dividing constantly. Here, newly formed cells are forced into the transitional zone where they elongate and differentiate and forms the fiber mass of the lens.The bulk of the lens is composed of the nucleus and cortex that comprise densely packed lens cytoplasm(‘fiber cells’) with very little extracellular space.

The thickness of lens capsule varies according to the location (Figure 3). It allows the passage of molecules both into and out of the lens. It is much thicker at the anterior capsule and the anterior and posterior capsules are much thicker at the equator where the zonules are attached. The zonules run from the ciliary processes and fuse into the outer layer of capsule. Capsule thickening increases anteriorly with age with little change posteriorly reflecting that the secretory epithelium is situated anteriorly.

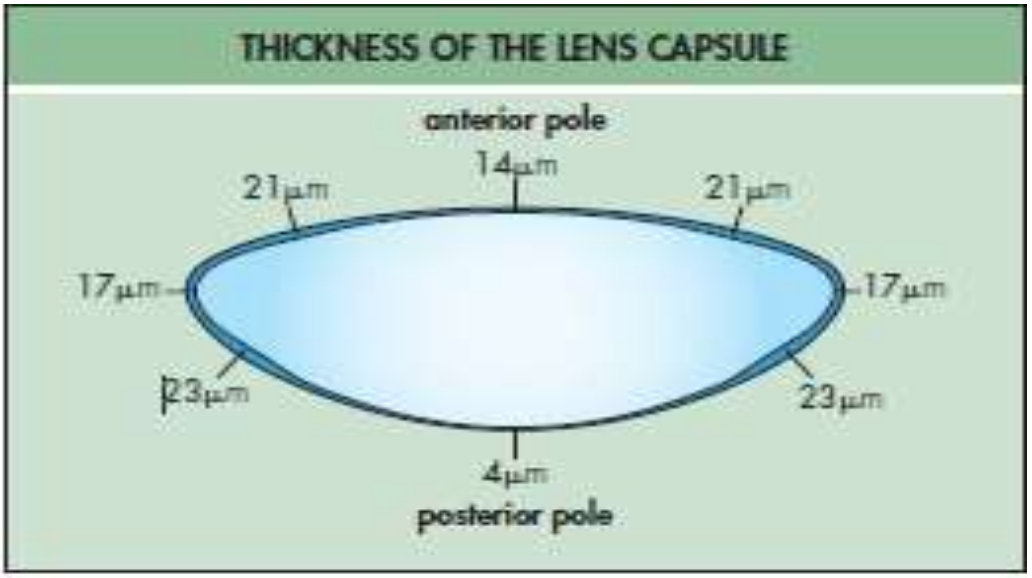



**FIGURE 3: THICKNESS OF LENS CAPSULE.**

## ANTERIOR EPITHELIUM (SUBCAPSULAR EPITHELIUM):

Functionally divided into 2 zones:

- Equatorial zone
- Nonequatorial zone

The equatorial zone is actively dividing and differentiating into lens fibres. The cells of non equatorial zone is involved in basal laminae secretion and movement of solutes between the lens. This layer produces lens fibres. Gradually, the cuboidal cells which are present in the anterior epithelium become columnar and they elongate towards the equator. Eventually, they converted into lens fibres. As new fibres are formed the older fibres are pushed in. The older fibres occupy a more central portion while newer fibres form the periphery.

## LENS SUBSTANCE:

The lens substance, the bulk of the lens, is composed of densely packed lens cells with very little extracellular space.

The lens Divided into

- Cortex
- Nucleus

The nucleus accounts for 84% of bulk of the adult human lens and cortex accounts for remaining 16%. The primary lens fibres are elongation of cells of posterior wall of lens vesicle and forms the embryonic nucleus. So the posterior aspect of lens is devoid of lens epithelium. The secondary lens fibres are formed from the anterior lens epithelium at the equatorial region. The secondary lens fibres are laid down concentrically.

## CORTEX

Only the young lens fibers have normal cellular organelles which subsequently disintegrate uponaging. Newly formed cortical fibers elongate with one end of the cell moving anteriorly and the other endposteriorly.

## NUCLEUS

It is the central part of the lens. It is divided into four zones

a) **Embryonic nucleus**: it is formed at 1 to 3 months of gestation and it is the inner most part

b) **Fetal nucleus** – corresponds to the lens from 3 months of intrauterine life till birth

c) **Infantile nucleus** –corresponds to lens from birth to puberty

d) **Adult nucleus** – corresponds to lens in adultlife.

These are derived from the equatorial lens epithelium. These fibres are laid down in concentric layers, the outermost of which lies in the cortex of the lens and the innermost in the core or nucleus. The lens fibres are strap like or spindle shaped cells which arch over the lens in concentric layers from front to back.

## SUTURES

Sutures are found at both the anterior and the posterior poles. They are formed by the overlap of ends of secondary lens fibers in each growth shell. No sutures are found between the primary fibers in the embryonic nucleus.

Each growth shell of secondary fibres formed before birth has an anterior suture shaped as an ‘erect Y’ and a posterior suture shaped as an ‘inverted Y’(Figure 4). The formation of sutures enables the shape of the lens to change from spherical to a flattened biconvex sphere.

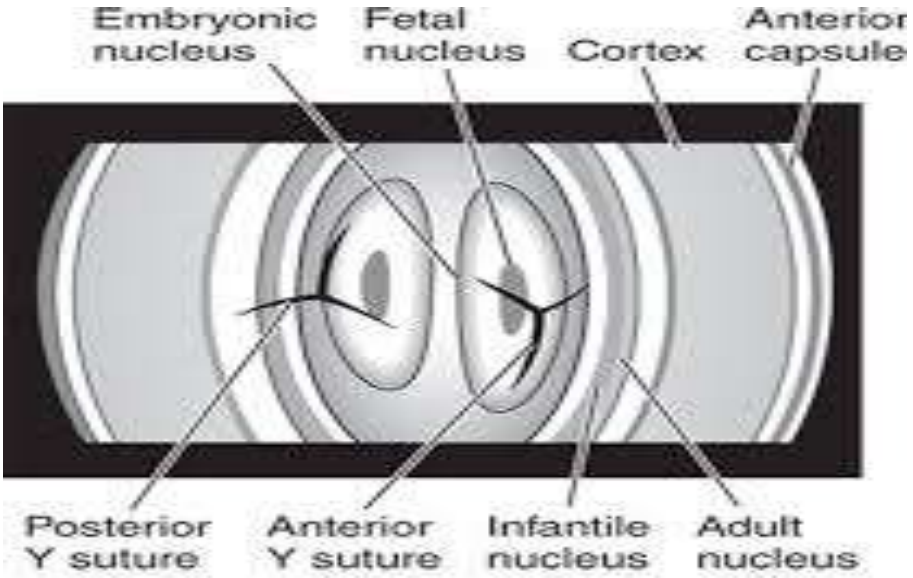



**FIGURE 4: LENS SUTURES**

## CILIARY ZONULES:

The ciliary zonules consists of multiple fibres which arise from the ciliary body and run towards the lens. The lens is held in place by the ciliary body and the ciliary muscles, which attach to the lens, act on it. The ciliary muscles get attached at the equator to the lens capsule.(Figure 5)

The zonular fibres are classified as:

- Main fibres
- Auxillary fibres.

## MAIN FIBRES

| Fibers | Origin | Insertion |
|---|---|---|
| **Orbiculoposterior capsular** | Ora serrata | Posterior capsule |
| **Orbiculoanterior capsular** | Pars plana | Anterior capsule |
| **Cilioposteriorcapsularfibres** | Valley and side of ciliary process | Posterior capsule |
| **Cilioequitorialfibres** | Ciliary valleys | Equator of the lens |

## AUXILLARYFIBRES:

Auxillary fibres add strength to the main fibres and anchor individual portions of the zonules. Some fibers also hold the ciliary body together. These fibers are very fine and run from without inwards and forwards.

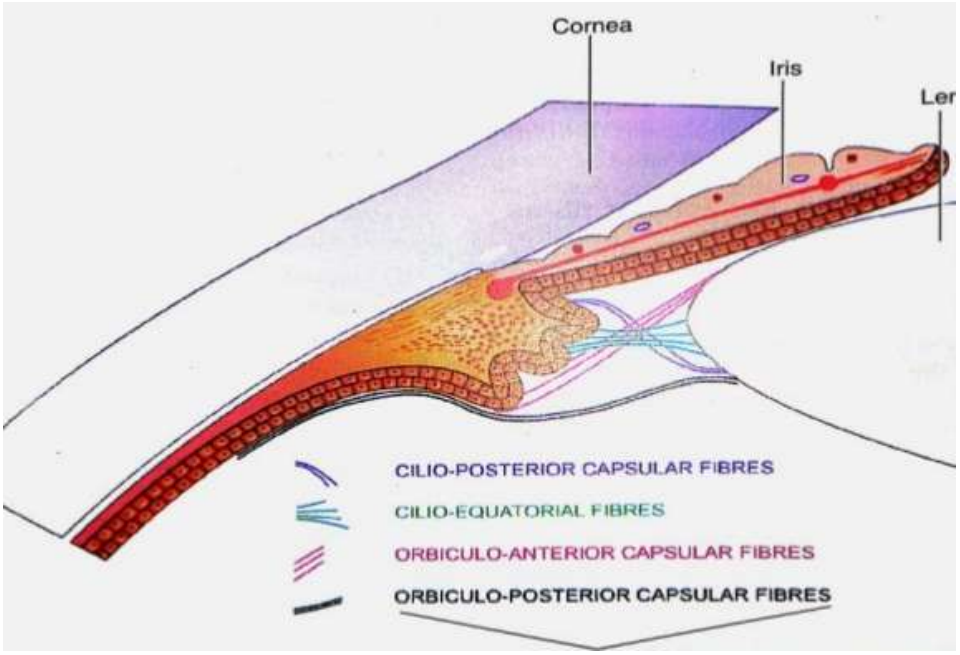



**FIGURE 5: CILIARY ZONULES**

## PHYSIOLOGY OF LENS:

After involution of the hyaloid blood supply to the lens, the metabolic needs are met by the aqueous and vitreous humor. The capsule of lens is freely permeable to water, ions, other small molecules, and proteins with a molecular weight of up to 70 kDa. There are a number of channels, pumps, and transporters in the epithelium that enable transepithelial movement to and from the extracellular milieu.

The lens acts as a spectral filter absorbing long UV-B (300–315 nm) and most of the UV-A (315–400 nm) wavelengths.

During the early stages of embryonic development the lens is opaque due to the presence of hyaloid vasculature. The lens becomes transparent as development continues and the hyaloid vascular supply is lost. Transparency of the lens is due to the absence of chromophores able to absorb visible light and the presence of a uniform structure that scatters light minimally. Light scatter is

minimized in lens fiber cells once the fibers have elongated and their organelles have degenerated.

The refractive index of the lens increases from 1.386 in the peripheral cortex to 1.41 in the central nucleus of the lens. Both the curvature and refractive index of the lens increase from the periphery toward the center. Each successive layer of fibers has more refractive power and, therefore, can bend light rays to a greater extent.

## BIOCHEMISTRY OF LENS:

### Carbohydrate and energy metabolism:

The lens is entirely dependent on the glucose metabolism for energy production. Glucose crosses the lens capsule and enters the lens substance by simple diffusion and partly facilitated diffusion. In here, glucose is rapidly metabolized by glycolysis that results in low level of glucose in the lens i.e, <1/10 of the level of glucose in aqueous

### Glucose metabolism occurs in fourpathways:

1. **Anaerobic glycolysis:** 85% glucose gets metabolized by this major pathway, it is responsible for 70% of the energy. It results in
    - 85% of glucose gets metabolized bythis major pathway.
    - Gives more than 70% of energy forlens.
    - 2 moles ofATP are generated for every mole of glucose.
    - Lactate generated undergoes 2 pathways of metabolism
        - Further metabolism via Kreb's cycle
        - Diffusion from lens into aqueous

i. Aerobic metabolism (Krebs cycle)

- It is limited to epithelium.
- 1 mole of glucose gives 38 moles of ATP.
- Only 3% of lens glucose is metabolized by this pathway.
- 20% of total ATP needed for the lens is generated.

ii. Hexose monophosphate shunt

- Accounts for 5% of glucose metabolized.
- Produces NADPH required for metabolic pathways like glutathione reductase and sorbitol pathway.

iii. Sorbitol pathway

- Glucose is converted to sorbitol by aldose reductase then to fructose by polyol dehydrogenase.
- Accounts for 5% of glucose metabolism by lens.
- When sorbitol accumulates within cells of lens, it sets up an osmotic gradient which induces influx of water and lens swelling, and ultimate loss of lens transparency.

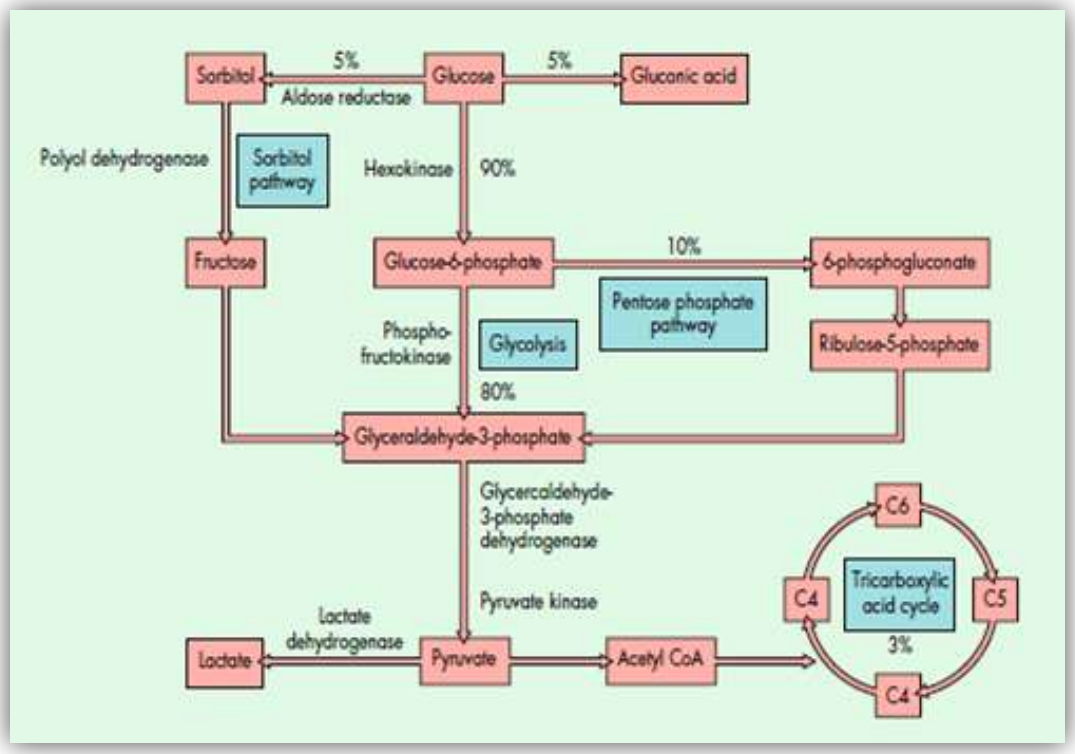



**FIGURE 6: OVERVIEW OF THE MAJOR PATHWAYS OF GLUCOSE METABOLISM IN THE LENS.**

## BIOCHEMICAL STRUCTURE OF LENS PROTEINS

### 1. Water soluble lens crystallins:

They form 90% the lens protein and are of three types

- Alpha crystalline
- Beta crystalline
- Gamma crystalline

### 2. Water insoluble proteins:

- Membrane proteins which are urea insoluble
- Cytoskeletal proteins and crystallin aggregates which are urea soluble

## PATHOGENESIS OF CATARACTOUS LENS

The lens is avascular structure and lies suspended in the intraocular fluid so its pathology is simpler than that of most other tissues. Therefore, primary inflammatory processes do not occur in the lens.

**The epithelial cells and the capsule:**

There can be thickening of the lens capsule as in some cases of senile cataract, or a thinning of the capsule in intumescent or mature cataracts. Occasionally the capsule spontaneously rupture, with the possible development of anaphylactic uveitis and secondary glaucoma.

The subcapsular epithelium may proliferate owing to toxic influences or injury. They may also suffer metaplasia into fibroblasts sometimes with the formation of a new hyaline capsule, which may encircle the mass of the epithelial cells. Alternatively, cloudy swelling, cytoplasmic vacuolation, pyknosis and death of the subcapsular cells occur as degenerative process.

**The lenticular fibres:**

Two types of change occur; a slow process of sclerosis and the more rapid development of coagulation and necrosis, either in the cortex or in a localized foci.

**1. Sclerosis:**

- The human lens normally undergoes changes with age.
- Older lens fibres in the center of lens become dehydrated and compact.
- The cross linking of soluble to insoluble proteins by coagulation in the nucleus, results in increase in optical density; decrease in transparency;

there is also development of index myopia; this is clinically known as nuclear sclerosis.

- It is accompanied by increased pigmentation with changes in colour from dark yellow, yellow brown to sometimes black.

**2.Proteolysis and necrosis**:

- It occurs primarily in the cortex and may be associated with the development of nuclear sclerosis.
- Loss of glutathione, the earliest event in the formation of cortical cataract, leads to electrolyte imbalances and decrease in protein synthesis, which eventually leads to protein and lipid aggregation and insolubilization.

This electrolyte imbalance leads to hydropic swelling of the fibres and an increase in the osmotic pressure, resulting in lens hydration called intumescent cataract.

At this stage lens shows water vacuoles and clefts between the fibres. It is a reversible change. If the metabolic upset can be controlled, as in case of early diabetic cataract or after a contusion it can be reversible.

- If the necrosis proceeds, the changes may result in the appearance of spokes, wedge shaped or dot shaped opacities or lamellar separation of the fibres. Sometimes it is localized or may proceed for complete opacification. As necrotic changes proceed the nuclei disappear from the fibres, vacuoles coalesce into large spaces and eventually the fibres break down into round globules called the Morgagnian globules. At later

stages, the lens is filled with waste products of the original fibres, clear fluid morgagnian globules, albuminous coagula of insoluble proteins, fatty droplets, the detritus of partially disintegrated fragments of fibres, and calcareous and crystalline deposits of various types.

- There is loss of water by diffusion and the lens become inspissated, wrinkled and shrunken. It becomes flat and yellowish with scattered calcification, such cataract is termed as hypermature cataract.
- Owing to thickening of the capsule and proliferation of epithelial cells, permeability is abolished. The cortex is replaced by milky fluid with small homogenous shrunken nucleus, which sinks to the bottom of the capsular sac and is called Morgagnian cataract.

## THE ANTERIOR CHAMBER ANGLE(ACA)

### EMBRYOLOGY:

The development of the ACA starts between the $12^{th}$ and $14^{th}$ week of intrauterine life. The formation of aqueous and its drainage is evident by the $8^{th}$ week. The ACA is well formed by the $8^{th}$ month of fetal life. By this time the sclera spur can also be identified. The average depth of the AC in newborn is 2.6mm, however it may range between and 2.4 mm to 2.9 mm.

### ANATOMY:

THE ANTERIOR CHAMBER(AC):

The AC of the eye is bounded anteriorly by the posterior surface of the cornea and posterior boundary is formed by the anterior surface of iris, the pupil

and the pupillary portion of the lens. The ACA borders it in the periphery. The ACA in turn is formed by trabecular meshwork, scleral spur, ciliary body, body, and root of iris.

The AC is ellipsoidal (kidney, bean like) in shape on a cross section. Its diameter varies from 11.3 to 12.4 mm, which is roughly the same as the cornea diameter. Analyzing the position of ACA with the corneal periphery in the horizontal meridian and sagittal meridian, it lies 1 mm and 0.75 to 1 mm posteriorly in the respective planes. The AC is narrowest at the angle. However, as the direction of the iris changes when it inserts into the ciliary body, there is a slight widening of the angle in its furthest recess.

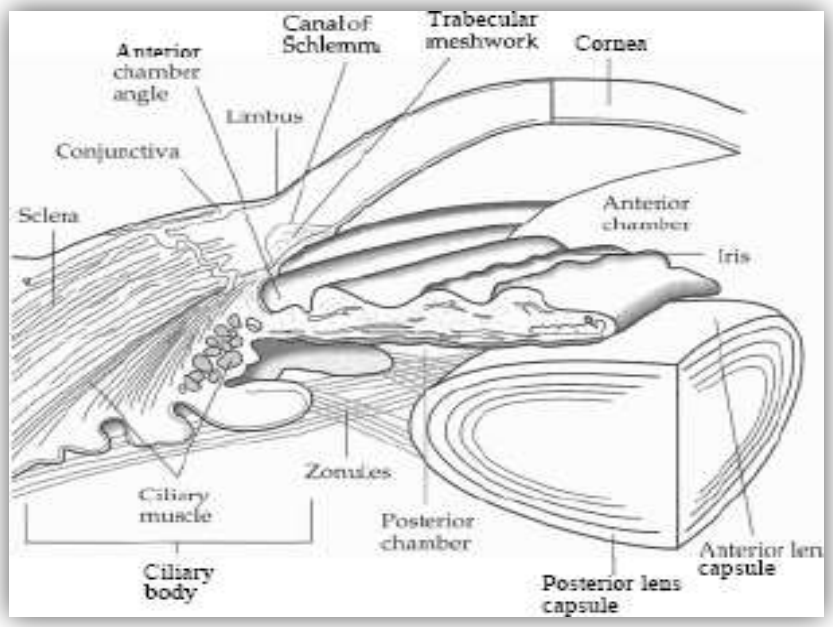



**FIGURE 7: ANATOMY OF THE ANGLE OF THE ANTERIOR CHAMBER**

## VARIATION OF ANTERIOR CHAMBER DEPTH:

The depth of the AC varied considerably. The factors affecting it are

- Age:

  The depth of the AC decreases with age, it may be attributed to thickening of the lens.

| Age in years | AC depth(mm) |
|---|---|
| <15 | 3.6-3.65 |
| 15-35 | 3-3.7 |
| 35-55 | 2.8-3.3 |
| >55 | 2.7-3.2 |

- Refractive error:

  Generally, in hypermetropia, the central depth of the anterior chamber ranges from 3 to 3.5 mm, in emmetropia it ranges from 3.1 to 3.6 mm, and in myopia it ranges from 3.3 to 3.8mm.

- Genetics.

## AQUEOUS HUMOUR:

The AC contains the aqueous humor. The specific gravity of this crystal clear fluid is between 1.0034 and 1.0036. The refractive index of this fluid is1.3336. It's refractive index is less than that of the lens. The AC with the aqueous humor it contains has an approximate volume of 0.25 cc, whereas the

volume of the posterior chamber and the aqueous humor it contains is 0.06 cc. The rate of production of the aqueous humor and its elimination is responsible for the intraocular pressure. In addition, the aqueous humor provides nutrients for the avascular lens and the cornea and the egress for waste products from these structures.

The aqueous fluid contains about 0.1% to 0.2% of the concentration of plasma protein and higher concentrations of amino acids than does plasma.

The aqueous has higher levels of lactate and ascorbate. The aqueous humor is produced at a rate of 2to 2.5 cc/min. Every minute,1% of the volume of aqueous humor of AC is and 3% of volume in the posterior chamber volume is replaced.

## ANTERIOR CHAMBER ANGLE:

The root of iris is 1.5 mm posterior to the corneoscleral margin. A variety of specialized channels lie between the iris root and the end of Descemet's membrane, they are the channels for the egress of aqueous fluid.

Numerous structures are visualized on gonioscopy, they include the root of iris, anterior surface of the ciliary body, scleral spur, iris processes, trabecular meshwork (external to which lie Schlemm's canal), Schwalbe's line, and the posterior cornea.

**1. Schwalbe's ring:**

Schwalbe's ring is just posterior to the peripheral termination of Descemet's membrane and contains of connective tissue arranged in concentric bands and elastic fibers.

**2. Trabecular meshwork (Trabeculum) :**

The trabeculum is a sieve like structure present at the angle of the AC. It converts the sclera sulcus into a circular channel called Schlemm's canal. It consists of two parts, a non-functional anterior nonpigmented part and a functional posterior pigmented portion. 90% of the aqueous humour leaves the eye through the trabecular meshwork.

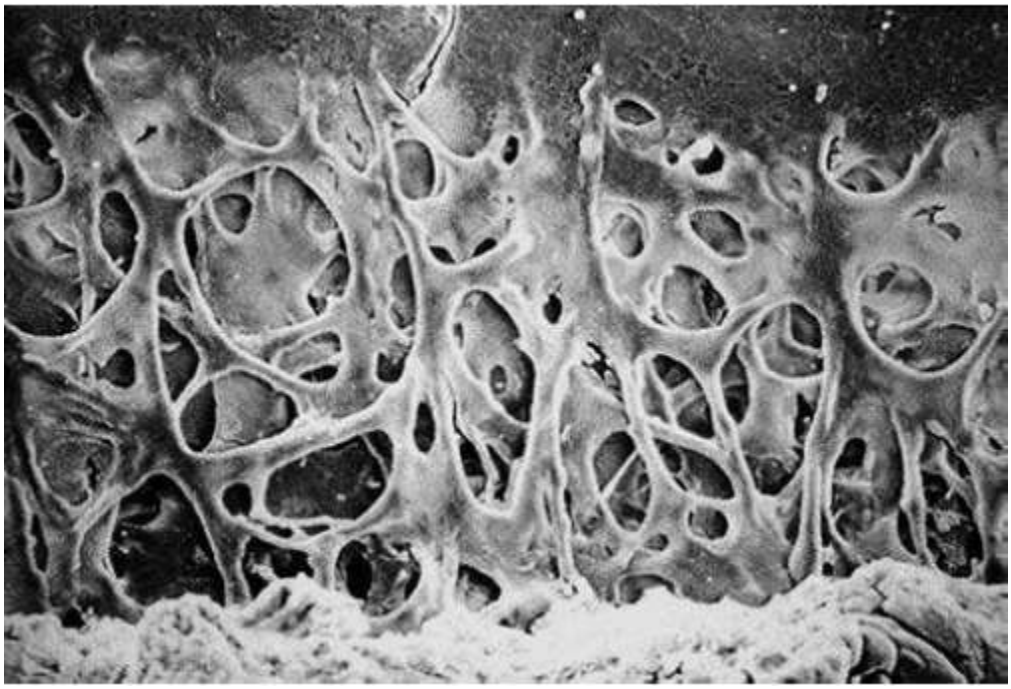


**FIGURE 8:SCANNING ELECTRON MICROGRAPH OF THE TRABECULAR MESHWORK**

The trabecular meshwork consists of a core of connective tissue which is surrounded by endothelium. It is divided into three portions:

- **Uveal meshwork**: Innermost portion which is adjacent to the aqueous humor in the AC and extends from the root of iris and ciliary body to the peripheral cornea. It consists of cord like endothelial cell-covered strands arising from the iris and the ciliary body stroma. Size of openings in this part varies from 25- 75microns. The intertrabecular spaces are relatively large and offer little resistance to the passage of aqueous.
- **Corneoscleral meshwork**: It lies external to the uveal meshwork. It is the thickest part of the trabecular meshwork. It is present between the sclera spur to the anterior wall of sclera sulcus. It consists of sheets of trabeculae perforated by openings which are elliptical in shape, these openings progressively reduce in size as the trabecular sheets get nearer to the Schlemm's canal. It is composed of layers of connective tissue strands with overlying endothelial-like cells. The intertrabecular spaces are smaller than those of the uveal meshwork, conferring greater resistance to aqueous flow.
- **Juxtacanalicular (cribriform) meshwork**: It is the outermost portion of meshwork and consists of a layer of connective tissue lined on either side by endothelium. The outer endothelial layer comprises the inner wall of schlemm's canal. It links the corneoscleral meshwork with the endothelium of the inner wall of the canal of Schlemm. The cells embedded in the dense extracellular matrix with narrow intercellular spaces, confers the major proportion of normal resistance to aqueous outflow.

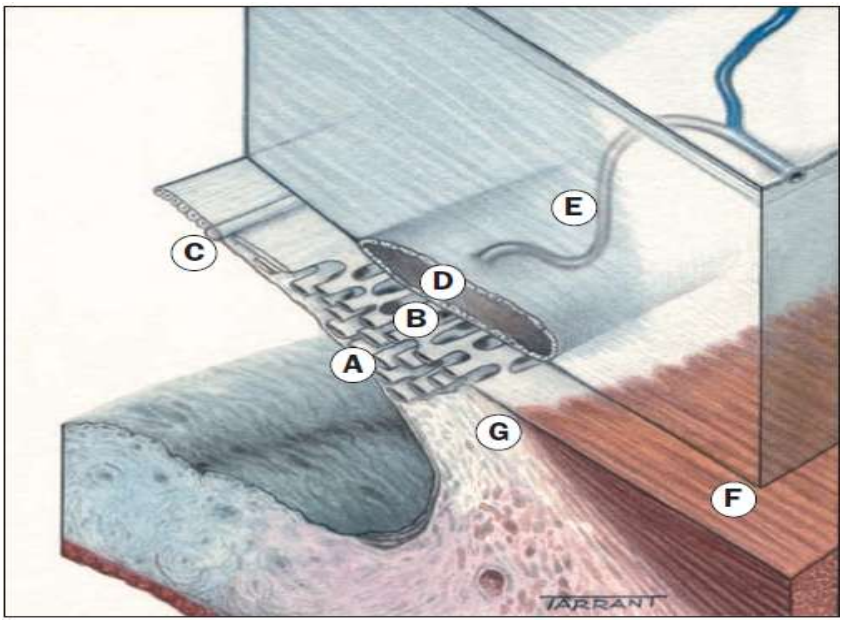



**Figure 9 : Anatomy of outflow channels: A, Uveal meshwork; B, corneoscleral meshwork; C, Schwalbe line; D, Schlemm canal; E, connector channels; F, longitudinal muscle of the ciliary body; G, scleral spur**

### 3. Schlemm's canal:

Schlemm's canal is a circumferential channel within the perilimbal sclera. In the most posterior recess in the internal scleral sulcus, it is present external to the trabecular meshwork. The posterior border is formed, respectively, externally and internally by the sclera and scleral spur. Anterior boundary is formed externally by the sclera and internally by the corneoscleral meshwork. It is a venous channel lined with a single layer of endothelial cells that measures 0.1 to 0.2 µm in thickness and 10 µm in diameter. It contains the openings of collector channels, which leave the canal at oblique angles and connect directly or indirectly with episcleral veins. Septa commonly divide the lumen into 2–4 channels. Its cross section is elliptic in appearance and ranges from 350 to 500 µm in width. When compared to adults it is narrower in children.

**4. Sclera spur:**

A group of fibers, the sclera roll, forms the posterior wall of the scleral sulcus. It runs parallel to the limbus and projects inwards to form the sclera spur. The longitudinal fibres of the ciliary muscle are attached to the posterior surface and helps in the drainage of aqueous.

**5. Ciliary body band:**

The portion of ciliary body that is visible in the AC as result of iris insertion into the ciliary body.

**6. Root of iris:**

This may sometimes be seen in a wide open angle.

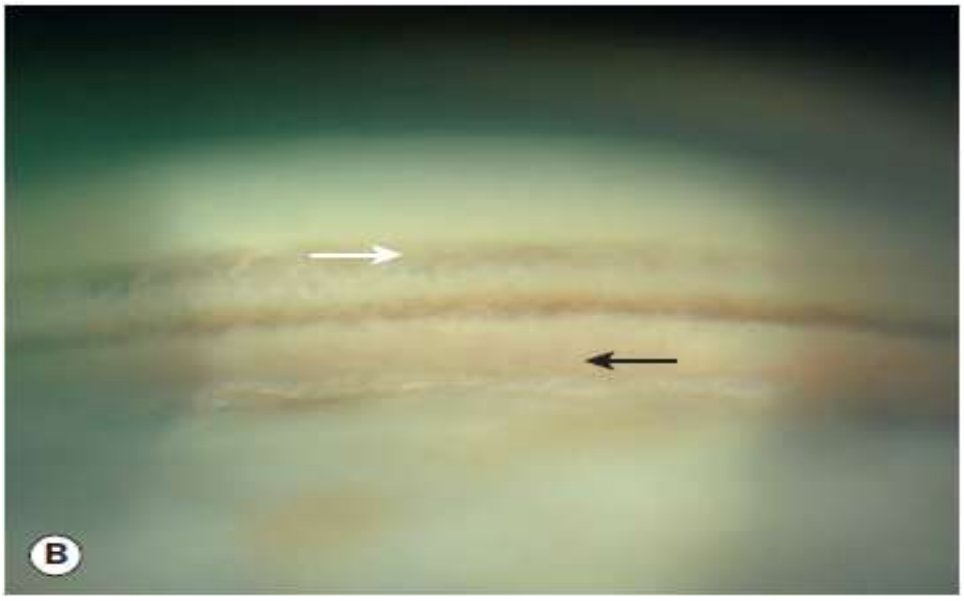



**Figure 10: Goniophotograph – a broad Schwalbe line is indicated by the white arrow, below which are the nonpigmented meshwork, the pigmented meshwork, the sclera spur and the ciliary body (black arrow) – the ciliary body is relatively lightly pigmented**

## DRAINAGE OF AQUEOUS HUMOR

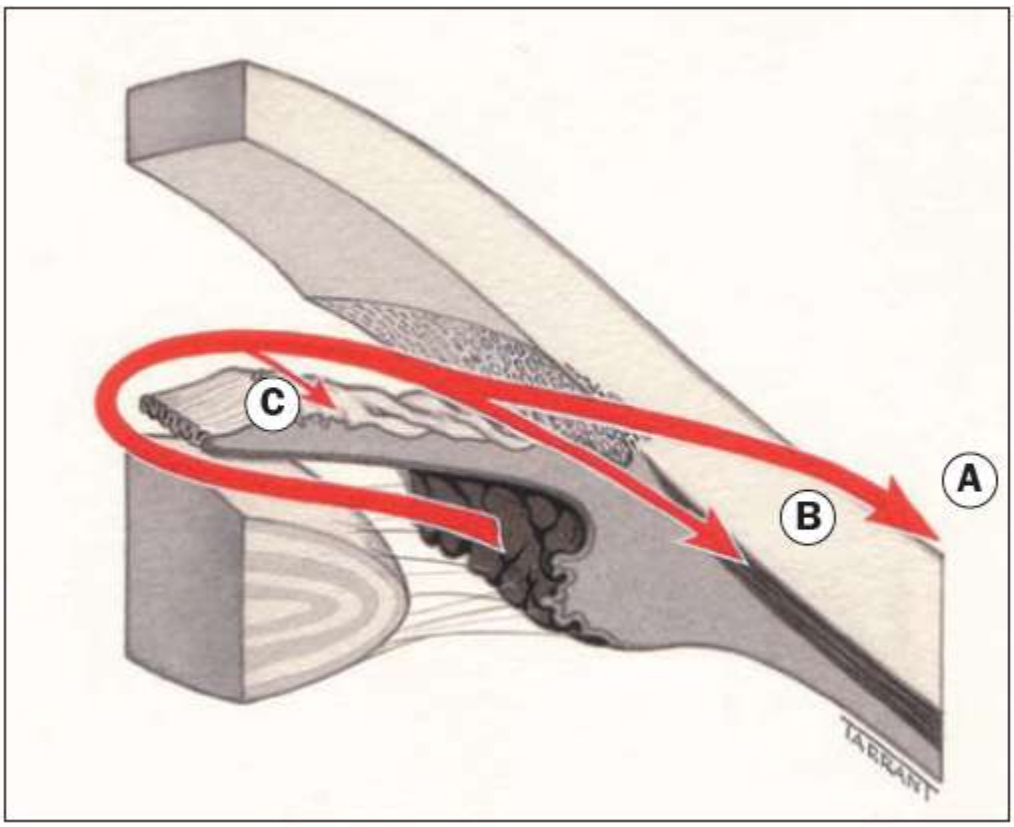



**FIGURE 11: ROUTES OF AQUEOUS OUTFLOW: A, TRABECULAR; B,UVEOSCLERAL; C, IRIS**

### 1. TRABECULAR OUTFLOW:

The trabecular outflow forms route for 90% of outflow. The aqueous humor enters the Schlem's canal after it passes through the trabecular meshwork finally it enters the collector channels. The Schlemm's canal is not a uniform vessel but is often divided in different parts by bridges orsepta.

The septa cross the lumen of the canal mostly in an oblique direction. The septa get fixed where the collector channels begin; i.e the outer wall of the canal. This results in septa forming twisted or spiral bands, which can be

considered to be guiding structures for aqueous humor toward the openings of the collector channels.

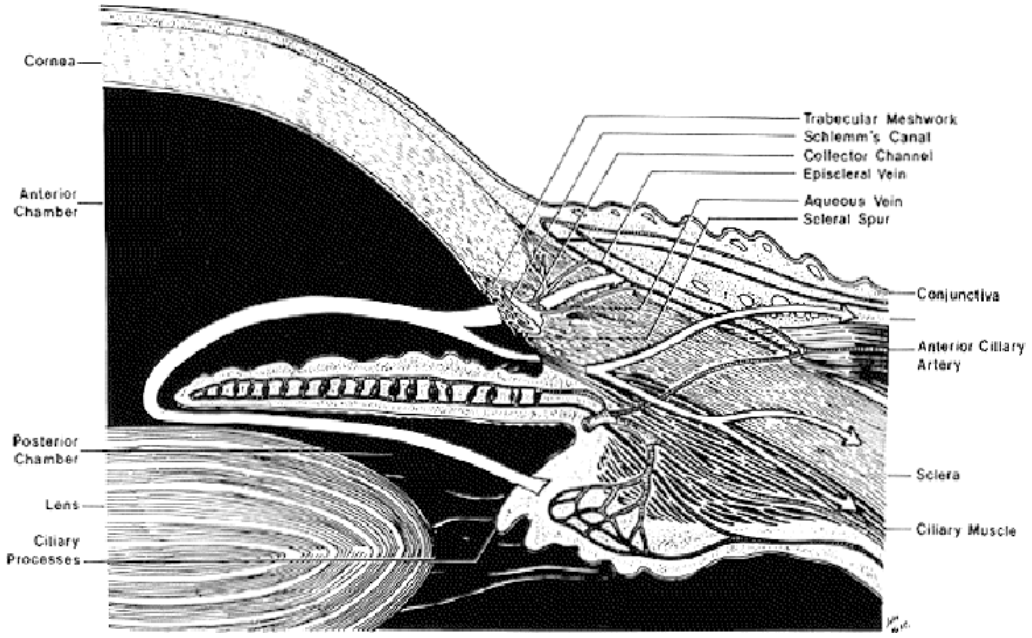



**FIGURE 12: AQUEOUS OUTFLOW**

The Schlemm's canal structure very much differs between the outer wall and the inner wall. It is formed by a single layered endothelial lining and a well developed basement membrane. Transcellular microchannels are absent in these cells. Collagenous and elastic-like fibers and fibroblasts are present in the stroma.

The aqueous humor drains towards the intrascleral or episcleral venous plexus through the collector channels which are about 25 to 35 in number. The collector channels can be distinguished as two types.

Direct channels run directly toward the episcleral plexus without any connections with the intrascleral vessels. The indirect collector channels, which are more numerous nasally than temporally, join the intrascleral venous plexus in various distances from Schlemm's canal.

2. **UVEO – SCLERAL OUTFLOW:**

Two pathways have been discriminated:

- **Uveoscleral:** The outflow occurs through the anterior uvea at the root of iris and the ciliary body (anterior surface) and then it flows through the suprachoroidal space into the episcleral tissues. It is drained by the venous circulation in the ciliary body, choroid and sclera.
- **Uveovortex:** into the iris vessels and vortex veins. More commonly seen in nanophthalmic eyes.

3. **IRIS :**

Some aqueous also drains via the iris.

# GLAUCOMA

It is a diverse group of disorders characterized by a progressive optic neuropathy with visual field loss and characteristic structural changes, including thinning of the retinal nerve fiber layer and excavation of the optic nerve head, and in which IOP is a key modifiable factor.

Glaucoma may be classified as

- Congenital (developmental)
- Acquired

On the basis of etiology-the underlying disorder that leads to an alteration in aqueous humor dynamics or retinal ganglion cell loss, glaucoma can be classified as

- Primary - no apparent contribution from other ocular or systemic disorder.
- Secondary - underlying, predisposing ocular or systemic events.

On the basis of mechanism by which the aqueous outflow is impaired with respect to the AC angle configuration the glaucoma can be classified as

- Open angle
- Closed angle
- Developmental anomalies of the ACA

## LENS-INDUCED GLAUCOMA

The lens induced glaucoma (LIG) is a heterogeneous group of secondary glaucoma inwhich may develop either through an open-angle or angle-closure mechanisms secondary to disorders of the lens.

**TABLE 3: Lens-lnduced Glaucomas**

| Entity | Angle Status | Mechanism of Glaucoma |
|---|---|---|
| Phacolytic glaucoma | Open | Outflow obstruction by lens protein and macrophages with phagocytosed lens material. |
| Lens particle glaucoma | Open | Outflow obstruction by lens particles, possibly inflammatory cells. |
| Glaucoma associated with Phacoanaphylactic uveitis | Open or closed | Outflow obstruction due to inflammation; pupillary block. |
| Phacomorphic glaucoma | Closed | Pupillary block; rarely direct compression of angle by intumescent lens |
| Glaucoma secondary to Ectopialentis | Closed | Pupillary block |

It is essential to identify the underlying mechanism causing elevation in intra ocular pressure in suspected LIG in order to provide an appropriate management.With sole cliinical examination, it is different to differentiate

between phacolytic, phacoanaphylactis and infectious procress(5,6). The cytological,, histopathological, and microbiological examination of ocular fluids or tissues can contribute greatly to the management of affected eyes and fellow eyes as well.

## PHACOLYTIC GLAUCOMA

The term phacolytic should be reserved for the sudden onset of open angle glaucoma caused whenhigh molecular weight lens protein leak through a relatively intact capsule in mature or hypermature cataract (4).

Usually, the patients presents with the acute onset pain and redness eye in an eye that had poor vision for a long period of time. They present with hyperemia of the conjunctiva with the raise in intraocular pressure and the ACA is open.

In such patients, the anterior segment examination is difficult due to corneal odema.The response of AC inflammatory cells vary considerably, flare is typical, and is often associated with iridescent or hyperrefringent particles. However, it is less common to present with extensive keratic precipitates or hypopyon. Posterior synechiae seldom form.

In the AC, white flocculent materials can be seen, they may also been in the vitreous cavity on case of posterior displacement of the lens. In phacolytic glaucoma, patches of white material are seem adherent to the lens capsule. These diagnostic values of these clinical findings, in patients with poterior dislocation of lens have been emphasized by Epstein.

Liquefaction of the lens cortex along with thinning of the lens capsule (posterior capsule) are seen in the histopathological examination of eyes enucleated in patients with phacolytic glaucoma (a rare outcome today).(4)

The lens capsule may rupture more frequent than what is clinically suspected. The calcium oxalate crystals noted in 18% enucleated specimens correlate with the occasional clinical observation of iridescent particles in the aqueous humor (4,7).

The inflammatory cell are mostly formed by bloated macrophages that appear to have engulfed the lens material. The phacolytic glaucoma has significantly lesser number of numbers of lymphocytes, plasma cells, and polymorphonuclear leukocytes as compared to other forms of intraocular inflammation. Confirmatory evidence may be obtained by observation of characteristic macrophages, whereas phacolytic glaucoma may not be ruled out when these cells are not identified.(8)

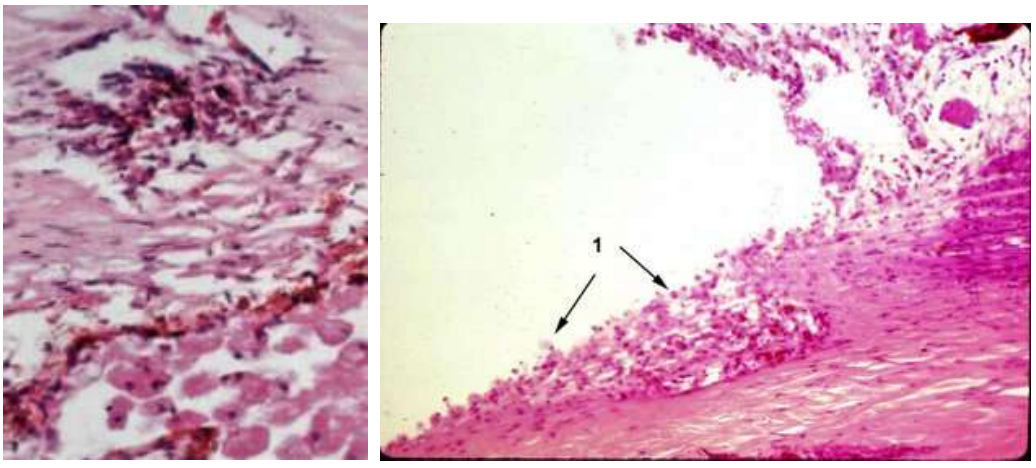



**Figure 13: The proteinaceous material and macrophage accumulate in the angle structures and trabecular meshwork (Arrow 1)**

With developing liquefaction of the lens cortex, its postulated p that heavy-molecular-weight protein becomes soluble. These proteins mostly diffuse through an intact lens capsule into the AC. A spontaneous or traumatic rupture of the lens capsule may result in formation of phacolytic glaucoma.(9).

The inflammatory cells in phacolytic galucoma may be attributed to greater chemotactic attraction of the monocytes and macrophage cell lines, compared to polymorphonuclear cell, by the lens protein(10).

Although the elevation of introacular pressure has been attributed to macrophage infiltration of the aqueous outflow pathway, Epstein and colleagues, clinically and experimentally provided evidence that the main contribution in outflow is in mainly by high molecular weight soluble lens protein.(10,11).

Cataract extraction is the definitive treatment for phacolytic glaucoma, it eliminates the obstruction of the aqueous outflow pathway by the prime source-the lens protein.

Preoperatively, intraocular pressure may be reduced by medical treatment with topical ß-blockers, systemic carbonic anhydrase inhibitors, and hyperosmotic agents. In the past, intracapsular cataract extraction was advocated due to concerns that extracapsular cataract surgery may lead to perpetuation and exacerbation of the disease when lens material are not cleared completely(12)(5).

However existing reports of cure attained in phacolytic glaucoma with extracapsular cataract extraction with or without posterior chamber intraocular lens implantation suggest that this may be a useful alternative to intracapsular surgery. (12,13)

## LENS PARTICLE GLAUCOMA

Intraocular pressure elevation with uveitis may occur subsequent to release of lens material into the AC through a surgically or traumatically created opening in the lens capsule (4,8).

Because this can occur years after an incomplete extracapsular cataract extraction, at a time when soluble lens protein has presumably been absorbed, it has been inferred that insoluble lens particles are responsible for outflow obstruction in this entity. The observation that the irrigation of the AC to remove lens particles helps control the glaucoma adds support to this theory.

A cellular role in outflow obstruction is suggested by the report of bloated macrophages in the aqueous humor of a patient who developed this syndrome 67 years after incomplete extracapsular cataract extraction.

In cases in which relatively small amounts of lens material are involved, medical therapy with topical corticosteroids, topical ß-blockers, and systemic carbonic anhydrase inhibitors may stabilize the eye long enough for absorption of the lens material to occur.

However, if the amount of dispersed lens substance is great (e.g., recent perforating injury to the lens) or the pressure elevation and inflammation are severe or protracted, surgical removal of lens material by either irrigation/aspiration, or extraction, is indicated.

## PHACOMORPHIC GLAUCOMA

Increasing lens thickness due to growth of the lens cortex is a well-recognized factor in the development of primary angle closure glaucoma.(14).

Other factors, such as short axial length of the globe, preexisting individual differences in anatomy of the ACA, and zonular relaxation may also contribute to variable extents. When angle-closure glaucoma develops due to an intumescence of the lens that can be distinguished from normal lens growth, the term phacomorphic glaucoma is applied (15).

Among the more readily identifiable situations in which angle closure may be attributable to lens swelling are rapidly developing mature cataracts and cataracts caused by trauma or inflammation.

The diagnosis of phacomorphic glaucoma should be entertained when unilateral or asymmetrical cataract is associated with shallowing of the ACA not explained by other factors (e.g., miotic therapy, lens subluxation, or uveal effusion).

In some cases, the differentiation of phacomorphic glaucoma from primary angle-closure glaucoma may be less clear-cut. Fortunately, both conditions respond to iridectomy (unless extensive peripheral anterior synechiae exist), indicating a common mechanism of pupillary block. The very rare development of phacomorphic glaucoma despite a patent iridectomy (16) suggests that in extreme cases of lens enlargement in very small eyes, the peripheral iris may be directly pushed against the trabecular meshwork without pupillary block.

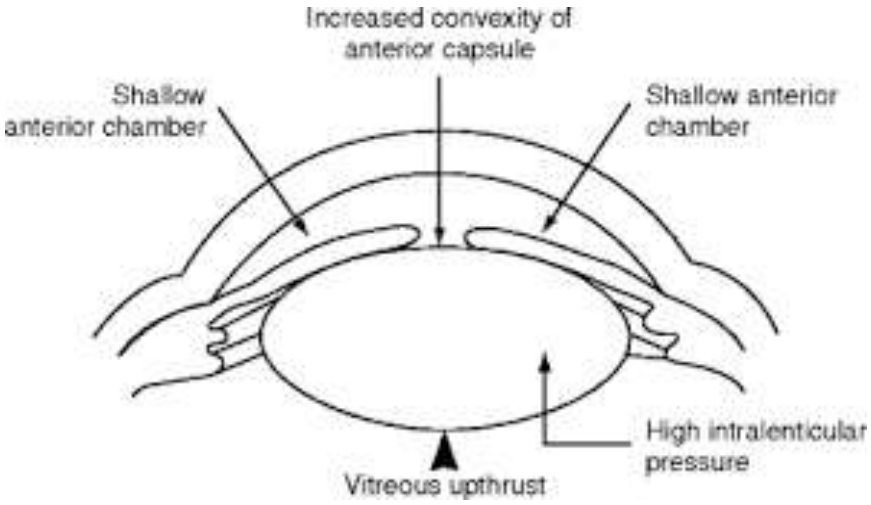



**FIGURE 14: PATHOGENESIS OF PHACOMORPHIC GLAUCOMA**

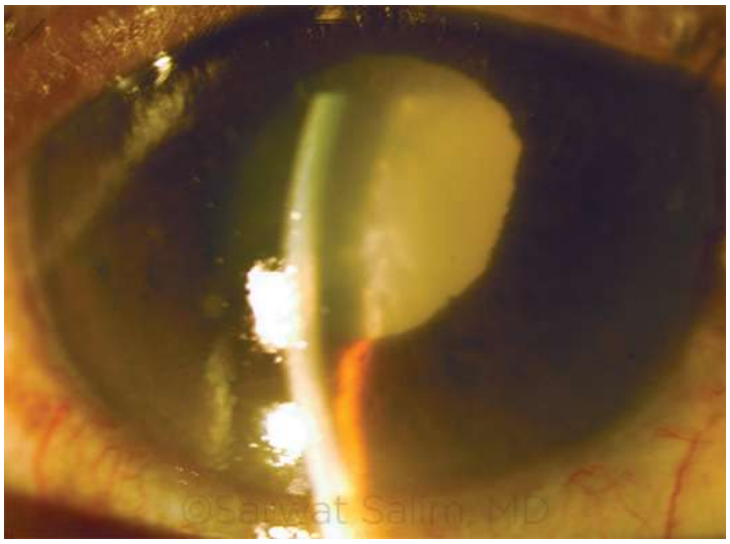


**FIGURE 15 : PHACOMORPHIC GLAUCOMA**

The definitive treatment of phacomorphic glaucoma in eyes with the potential for visual improvement is cataract surgery.

Just as angle closure can result from crowding of the ACA by an enlarged lens, a similar process can occur with an average-sized lens in an extraordinarily small (nanophthalmic) eye.

## GLAUCOMA SECONDARY TO ECTOPIA LENTIS

When the lens is displaced from its normal position, it is considered subluxated if it remains in the pupillary area and luxated or dislocated if it is completely displaced from the pupil (separation of all zonular attachments).

**Etiology:** Lens displacement may be divided into three types

- Traumatic,
- Hereditary and
- Spontaneous.

a. Traumatic ectopia lentis:

Trauma is the most common cause of a displaced lens, more than 50%. In addition to the lens dislocation, hyphaema, iridodialysis, angle recession, rupture of the vitreous face, scleral rupture, vitreous hemorrhage, retinal oedema, hemorrhage or detachment may be present. Lens may be subluxated or dislocated, anteriorly or posteriorly which may lead to pupillary block glaucoma by lens itself or by vitreous herniation.

b. Hereditary ectopia lentis:

Hereditary forms may be associated with other systemic or ocular anomalies or appear as isolated anomaly.

Marfan's Syndrome:

In 1896, Marfan described this autosomal dominant disorder which is characterized by tall, slender individual with long distal limbs (arachnodactyly), ectopia lentis, poorly developed

musculature and cardiopathy. The most characteristic ocular anomaly is ectopia lentis, found in 70% to 80% of the cases, which is bilateral, partial and rarely progressive with characteristic displacement superiorly and temporally. It is usually seen early in life at 5 years in 50% of patients. Glaucoma, which is present in about 8% of eyes with ectopic lenses, may result from the lens dislocation into the pupil or from angle anomaly.

Homocystinuria:

These patients resemble those with Marfan's syndrome in habitus and ocular problems, but differ by an autosomal recessive inheritance and frequent mental retardation. The lens dislocation is more often inferiorly. High incidence of complete dislocation and pupillary block glaucoma.

Weill Marchesani Syndrome:

This syndrome also called spherophakia, is much more rare than Marfan's syndrome and is also the antithesis of it with respect to habitus in that the patients are short and stocky. The principle features are short fingers (brachydactyly) and small, round lenses (microspherophakia). Lens dislocation occurs later in life, with more common glaucoma due to anterior dislocation or pupillary block.

Other hereditary conditions:

Include Ehlers Danlos syndrome, hyperlysinemia, sulfite oxidase deficiency, Sturge weber syndrome, Crouzon's syndrome, ectopialentis at pupillae, aniridia etc.

c. Spontaneous ectopialentis:

It is slightly more common than the heritable varieties and is related to mechanical stretching of the zonule. In some middle aged or older individuals, the lens gradually tilts backward as the upper zonules give way and eventually becomes dislocated into the vitreous cavity, usually in association with cataract formation. It may be seen in high myopia, in endophthalmitis, also occurs after treatment of retinal detachment by diathermy, in Eale's disease, chalcosis and so on. An acute dislocation may follow perforation of a corneal ulcer when the lens iris diaphragm is suddenly thrust forward. It may be associated with other ocular disorders such as buphthalmos, megalocornea and coloboma of the iris and choroid.

## MECHANISM OF GLAUCOMA IN DISPLACED LENSES:

Secondary glaucoma is a common occurrence due to displacement of lens, which may be due to:

i. 'Pupillary block' by the lens or vitreous or both.
ii. Development of permanent 'Peripheral anterior synechiae.
iii. Phacolytic glaucoma.

iv. Neovascularisation of the iris and angle.

v. Due to repeated attacks of iridocyclitis.

vi. In traumatic cases in addition to all the above factors, angle recession is an important cause.

vii. In some hereditary conditions it may occur due to associated dysgenesis of the angle of AC.

## REVIEW OF LITERATURE

There are clinically recognisable several forms of glaucoma that may occur in association with the formation of cataracts.

In 1900, Gifford and Von Reuss described a form of glaucoma which is of open angle mechanism due to hypermature cataract(17). Fewer terms were subsequently suggested for this condition and were called phacogenic glaucoma(18) and lens-induced uveitis(19).

**Flocks et al**.(20) reported that the histological findings suggesting that the glaucoma-inducing mechanism was a macrophagic response to lens material. Hence it was called as phacolytic glaucoma.

**Epstein et al**.(10) has studied the evidence of high molecular weight proteins responsible for the obstruction of aqueous outflow. He termed it to be lens particle glaucoma.(11)

In eyes with advancement in cataract formation, the lens may become intumescent or swollen, which cause progressive reduction in the anterior chamber angle leading to the formation of angle closure glaucoma. This is termed as phacomorphic glaucoma(8)

**Tomey KF et al**. in his study of cases with phacomorphic glaucoma, the attack of angle closure was relieved in all cases by the procedure of laser iridectomy(21).It brought the pressure under control before the cataract surgery.

It was thought that a primary toxicity of cataractous lens material was

responsible for the inflammatory reaction called phacotoxic uveitis there were associated glaucoma in few cases. Subsequent studies by Muller H. have not supported the concept that liberated lens material is toxic(22). It is due to the liberation of lens particles and debris after disruption of the lens capsule and it is termed as lens particle glaucoma(8).

In 1922, **Verhoeff and Lemoine**(23) reported that a few cases were hypersensitive to lens protein and the rupture of lens capsule leads to an intraocular inflammationand is called endophthalmitis phacoanaphylactica. Although they are rare, Rahi AHS et al.(24) showed that there is evidence of true phacoanaphylaxis which occured in response to lens protein antigen, with inflammation. Few of them had a feature of open angle glaucoma.sss

Dislocation/ Subluxation of the lens may be associated with a number of clinical manifestations, which leads to the formation of glaucoma by various mechanisms. The most common cause being trauma causing displacement of lens(25). **Jarrett WH**(26) in a series of 166 cases reported that injury accounted for 53% of the total group.

Pseudoexfoliation syndrome can be associated with spontaneous or traumatic lens subluxation/ dislocation. Dislocation of the lens may occur without associated ocular or systemic abnormalities as a congenital anomaly or as a spontaneous disorder later in life(27,28).

There are several studies(27,28)[21,22] which showed that Ectopia lentis et pupillae is a condition with a slit shaped pupil and subluxation of lens in the opposite direction.

**Cross HE, Jensen AD**(29) have reported that the incidence of lens subluxation is increased in Marfan's, Homocystinuria and Weil-Marchesani syndrome(30). Several studies have found increased association of lens dislocation with rare congenital disorders like Ehlers-Danlos syndrome, hyperlysinemia, sulphite oxidase deficiency and aniridia(31–33).

**Ellant. J. P, Obstbaum.S**(34) in their study concluded that the crystalline lens is found to be the causative element in producing several forms of glaucoma. Etiologically they representss diversity in the presentation of the glaucomatous process. These conditions include glaucoma related to: lens dislocation (ectopia lentis), lens swelling (intumescent cataract), classical pupillary block, aqueous misdirection--ciliary block, phacoanaphylaxis, lens particle, and phacolytic glaucoma. The management of elevated intraocular pressure often requires altering the intraocular relationship of anatomic structures surrounding the lens or lens removal. They also reviewed the entities that produce these lens-induced glaucomatous conditions and suggested a rational approach to their diagnosis and treatment.

According to **Filipe. J. C et al**(35) the pathogenesis of lens-induced uveitis and phacolytic glaucoma is still not fully understood. The authors report a case of a 62-year-old white female, with bilateral lens dislocation into the vitreous, who

presented clinical and pathological features of phacolytic glaucoma in the left eye. The ultrastructural study of aqueous and vitreous aspirates showed lenticular fragments and macrophages with lipofuscin granules and phagocytic vacuoles containing lens proteins. Immunocytochemistry revealed foamy macrophages immunoreactive for CD68 and HLA-DR. One year later the right eye disclosed a mild anterior granulomatous uveitis with corneal mutton-fat keratic precipitates that remained unchanged in the course of sixteen months follow-up without further treatment. These findings corroborate the possibility that, besides their mechanical and inflammatory roles in the impairment of the outflow system of the (exciting) left eye, phacolytic macrophages might also have been involved in the afferent phase of the mild chronic uveitis of the (fellow) right eye.

Several studies on lens induced glaucoma were done in India. A few of the studies and the conclusions are listed below.

**Prajna. N V, Ramakrishnan**(4). R, in their study "Lens induced glaucoma-visual results and risk factors for final visual acuity" came to the conclusion that lens induced glaucomas are a common occurrence in India. An attempt was made to understand the clinical modes of presentation and post operative visual results in 93 patients with lens induced glaucoma, 49 phacomorphic and 44 phacolytic, attending their institute during 1994. All these patients were subjected to a planned extracapsular cataract extraction. Forty four percent had a posterior chamber intraocular lens implantation following surgery. Fifty seven percent eyes with phacomorphic glaucoma and 61% with phacolytic glaucoma recovered visual acuity of 6/12 or better. There was no significant difference in the final visual acuity

between those patients who had an intraocular lens implanted and those who did not (P = 0.18).

Univariate analysis was performed for selected risk factors such as age, sex and duration of glaucomatous process as predictors of final visual acuity and odds ratios with 95% confidence intervals were calculated. Patients with age more than 60 years (OR = 2.7, 95% CI = 1.04-6.93) and in whom the glaucoma was present for more than 5 days (OR = 3.1, 95% CI = 1.21-8.13) had a significantly higher risk of poor visual outcome post-operatively.

**Mandal. A. K et al**(36) in their study titled "Intraocular pressure control and visual outcome in patients with phacolytic glaucoma managed by extracapsular cataract extraction with or without posterior chamber intraocular lens implantation." observed that ECCE with or without primary PC IOL implantation is safe and curative in patients with phacolytic glaucoma. The addition of trabeculectomy to cataract extraction seems to be superfluous in the control of IOP in patients with phacolytic glaucoma operated on within 2 to 3 weeks of the onset of symptoms. Light perception without projection is not a contraindication for cataract surgery in phacolytic glaucoma.

**Pradhan.D, Hennig. A, Kumar. J**(37), **et al** and **Sujatha et al**(38), made a prospective study of 413 cases of LIG in Nepal and the results highlight the importance of early diagnosis and treatment of visually disabling cataract.

## AIM OF THE STUDY

- To assess the final visual outcome in different types of lens induced glaucoma after surgical management.

## OBJECTIVE

- To study the various modes of presentation of different lens induced glaucoma.

- To assess the factors influencing the final visual outcome.

## MATERIALS AND METHODS

A prospective observational study was carried out in GOVERNMENT STANLEY MEDICAL COLLEGE, Chennai. 98 patients diagnosed with LIG who attended ophthalmology Outpatient department (OPD) were included in the study during the year January 2018 to June 2019. A proper informed consent was obtained and complete ophthalmic examination was carried out. All the patients were admitted and underwent manual small incision cataract surgery after IOP control. The patients were followed up and complete ophthalmic examination was done.

### INCLUSION CRITERIA:

The following cases were included in our study.

1. Phacomorphic glaucoma.
2. Phacolytic glaucoma
3. Phacotoxic uveitis/ lens particle glaucoma.
4. Glaucoma secondary to ectopia lentis.

### EXCLUSION CRITERIA:

The following cases were excluded from the study.

1. Cases of primary open angle or angle closure glaucoma associated with cataract were excluded from the study.
2. Aphakic or pseudophakic glaucoma
3. Other associated glaucomas with cataract such as pseudoexfoliation glaucoma, pigmentary glaucoma.

4. Congenital /developmental glaucoma
5. Cases with significant posterior segment pathology.

## PATIENT EVALUATION:

All the 98 patients after clinical diagnosis were admitted and a detailed history and examination were done.

The details of history included the duration of diminution of vision and its progression, the onset of pain, redness, watering and photophobia in the affected eye. The associated symptoms such as headache, nausea and vomiting were also taken. The patient was enquired about history of ocular trauma and previous surgery done in the eye.

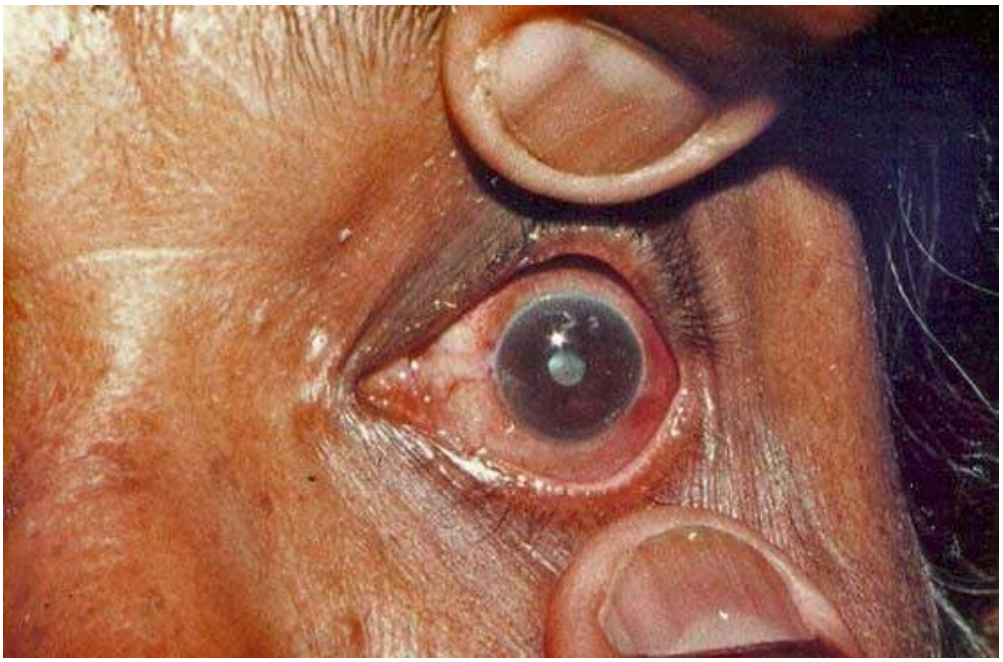


**FIGURE 16 : A CASE OF PHACOMORPHIC GLAUCOMA**

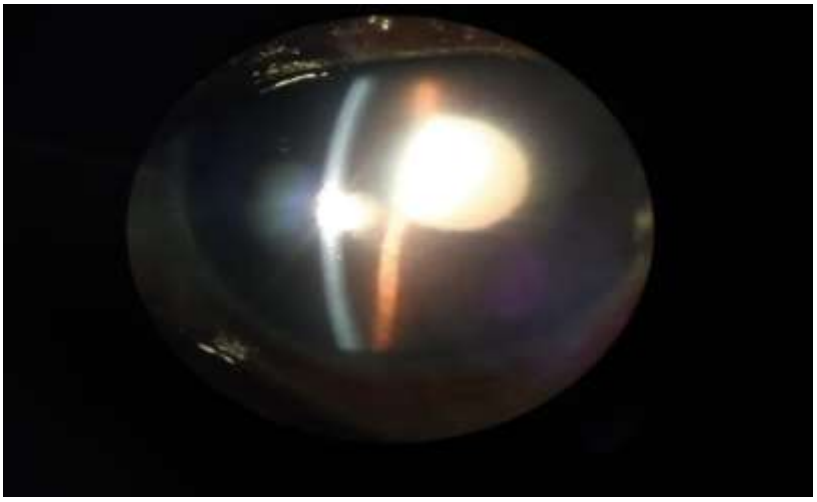


**FIGURE 17 : A CASE OF PHACOLYTIC GLAUCOMA**

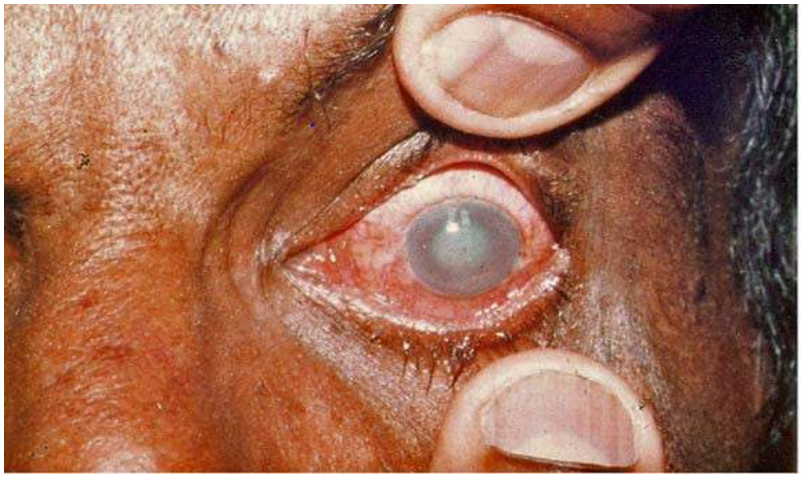


**FIGURE 18 : A CASE OF LENS PARTICLE GLAUCOMA**

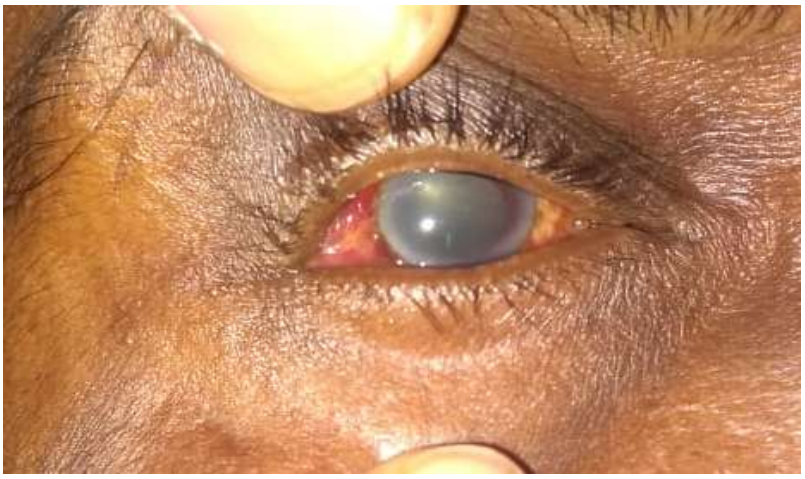


**FIGURE 19 : CASE OF TRAUMATIC ANTERIOR SUBLUXATION OF LENS AND 2ACG DUE TO PAPILLARY BLOCK.**

The physical examination of all these 98 patients included a thorough general and systemic examination. Examination of the globe and adnexa was also done. A detailed proforma of the case sheet has been shown later (Pg no 97)

In the affected eye, visual acuity was recorded and retinal function was assessed by the perception of light and projection of rays.

The intraocular pressure was measured using applanation tonometer. When corneal edema was present rebound tonometry was used.

Complete ocular examination was done and the diagnosis of the type of lens induced glaucoma is done.

The examination of the other eye was done in detail. The status of the other eye such as clear or cataractous lens or pseudophakia was noted. The visual acuity in other eye was noted. If operated for the catarctous lens earlier, the duration in years before which the surgery was done is also noted.

Other routine tests like lacrimal syringing, A scan biometry, blood sugar, blood urea, serum creatinine, urine sugar, blood pressure, ECG recording were done.

**MANAGEMENT:**

The management of the cases included pain relief and bringing down the raised intraocular pressure. Apart from these, subsidence of the uveal inflammation was also done. Ultimately removal of the cataractous lens is the definitive treatment.

## MEDICAL MANAGEMENT:

Medical management consisted of analgesics, cycloplegics in cases of open angle mechanism, topical steroids, anti- glaucoma medication and drugs for reducing associated symptoms.

Cycloplegics were used in cases of open angle mechanism of lens induced glaucomas. Topical Atropine Sulphate 1% eye ointment helped to reduce uveal inflammation and also reduced pain due to its cycloplegic action.

Topical 1% Prednisolone eye drops was preferred steroids and wascapplied hourly which helps to bring down the inflammation. In cases with severe iridocyclitis, Tab. Prednisolone 1gm/kg body weight/day was used.

To reduce the IOP, Inj. Mannitol 20%, 1 g/kg was given IV over 30 minutes BD. Along with that oral Acetazolamide 500mg stat followed by 250 mg $6^{th}$ hourly is given as maintainence dose. This was supplemented with topical antiglaucoma medications like beta-blockers preferably 0.5%Timolol maleate eye drops bd or topical alpha adrenergic receptor agonist like brimonidine 0.2% eye drops bd.

Supportive management consisted of antiemetics such as injection ondansetron 4 mg IV to reduce vomiting. Diclofenac Sodium was used as analgesic in oral or intramuscular forms to reduce pain.

## PREOPERATIVE MANAGEMENT:

Prior to the surgery, IOP was recorded in all patients and if the IOP was still raised, Inj. Mannitol 20%, 1g/kg body weight IV was given over a period of 30 min one to one and half hours before surgery along with oral Tab. Acetazolamide 500mg stat and 250 mg QID maintenance.

Pre-operatively pupils were dilated with combination of Tropicamide (0.08%) and Phenylephrine(5%) eyedrops, instilled every 15min until the pupils dilated. one drop of Flurbiprofen(0.03%) eyedrops were instilled 1/2hourly three times preoperatively to prevent miosis intraoperatively.

**SURGICAL MANAGEMENT:**

Following control of IOP, patient was operated for lens extraction and Intraocular Lens implantation(IOL). The preferred surgery was Manual Small Incision Cataract Surgery with IOL implantation under local (peribulbar) anaesthesia. Suitable modifications in the surgical steps were made according to the complications encountered.

**TECHNIQUE OF SURGERY:**

- A Peribulbar injection of infiltrative anaesthesia of 2% Lignocaine and 0.5% bupivacaine with hyaluronidase was given using a 24G needle under aseptic precautions.
- The eyeball and periorbital region was painted and draped with 10% betadine solution.
- A universal wire speculum was applied to keep the lids apart during surgery.
- A superior rectus bridle suture was placed. A fornix based conjunctival flap was made and wet field cautery was used to arrest the bleeding vessels if necessary.
- A straight incision was made around 2mm away from the superior limbus using a No. 15 Surgical blade and the length of the incision made was 6mm.
- By using an angled cresent blade, a sclerocorneal tunnel was made upto 1.5mm into the clear cornea.

- A sideport was made by using a $15^0$ side port entry blade. Depending on the right eye or left eye, it is made at the 9 0'clock or the 3 0'clock limbus respectively.

- The Anterior Chamber (AC) is formed with air and the capsule was stained using Trypan blue.
- The capsulotomy was done either by continuous curvilinear capsulorrhexis or can opener technique depending on the situation.

- The AC is formed with viscoelastic substance and the entry into the AC is made through the main tunnel done using a 3.2mm angled keratome and the extension is made using a 5.2mm angled keratome.
- The nucleus is prolapsed into the AC and delivered out by visco expression or irrigating wire vectis.
- By continous irrigation and aspiration method the remaining cortical matter was removed.

- The AC was formed with viscoelastics and the capsular bag was distended.
- The IOL was implanted in the capsular bag or in the sulcus.
- If any intraoperative complications are encountered like zonular dialysis and vitreous loss due to posterior capsular rent, making Posterior Chamber Intraocular Lens (IOL) implantation difficult, iris claw lens or scleral fixated IOL or Anterior chamber IOL was placed. Peripheral iridectomy was performed wherever necessary. Anterior vitrectomy was done in cases of vitreous loss following posterior capsular rupture.

- Minimal AC wash was done to remove the viscoelastic material.
- The side port wound was closed by hydration.
- Subconjuctival injection of dexamethasone and gentamycin was given at the end of the procedure.
- Any complications that occurred during the surgery were noted down.
- The SICS procedure is combined with trabeculectomy in cases of long standing glaucoma causing peripheral anterior synechiae formation.

**POSTOPERATIVE MANAGEMENT:**

The eye pad was removed the next day in the first postoperative day . A detailed slit lamp evaluation was done and looked for any post-operative complications. Vision with pin hole was tested on the first day.

All the patients were given topical antibiotic and steroidal drops to be intilled 8-10 times daily for a period of 6 weeks on tapering dose. Topical non steroidal anti inflammatory drugs were added 4 times a day.

Patients were followed up at the time of discharge at day 3 and 4 weeks later. At each visit a detailed slit lamp examination, applanation tonometery and Best corrected visual acuity (BCVA) were recorded.

The above data were compiled using a standardized proforma and at the end of the study the data was analysed statistically using the Chi-Square method and the Probabaility value. The stastistical analysis was done using the 'MINITAB for Windows' software.

# OBSERVATIONS AND RESULTS

## STUDY SUBJECTS

98 patients diagnosed with lens induced glaucoma were included in this study.

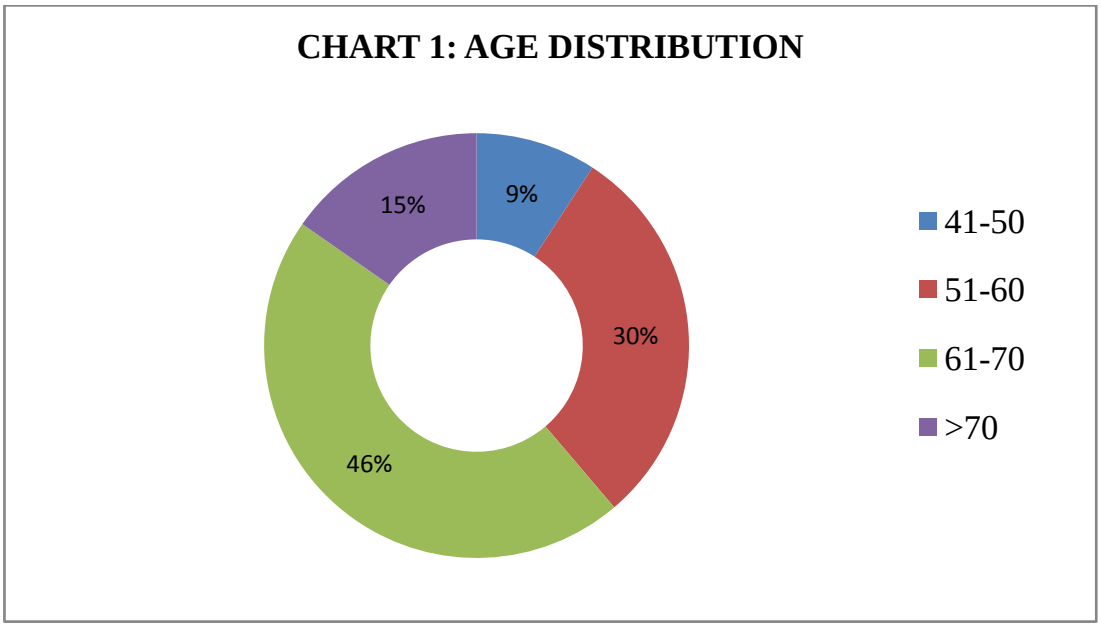
The collected data were analysed with IBM.SPSS statistics software 23.0 Version. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference in the multivariate analysis the one way ANOVA with Tukey's Post-Hoc test was used. To find the significance in categorical data Chi-Square test was used. In all the above statistical tools the probability value .05 is considered as significant level.

## AGE

The following table shows the age distribution in this study.

**TABLE 1: AGE DISTRIBUTION**

| AGE | FREQUENCY | PERCENTAGE |
|---|---|---|
| 41-50 | 9 | 9.18 |
| 51-60 | 29 | 29.59 |
| 61-70 | 45 | 45.92 |
| >70 | 15 | 15.31 |
| TOTAL | 98 | 100 |

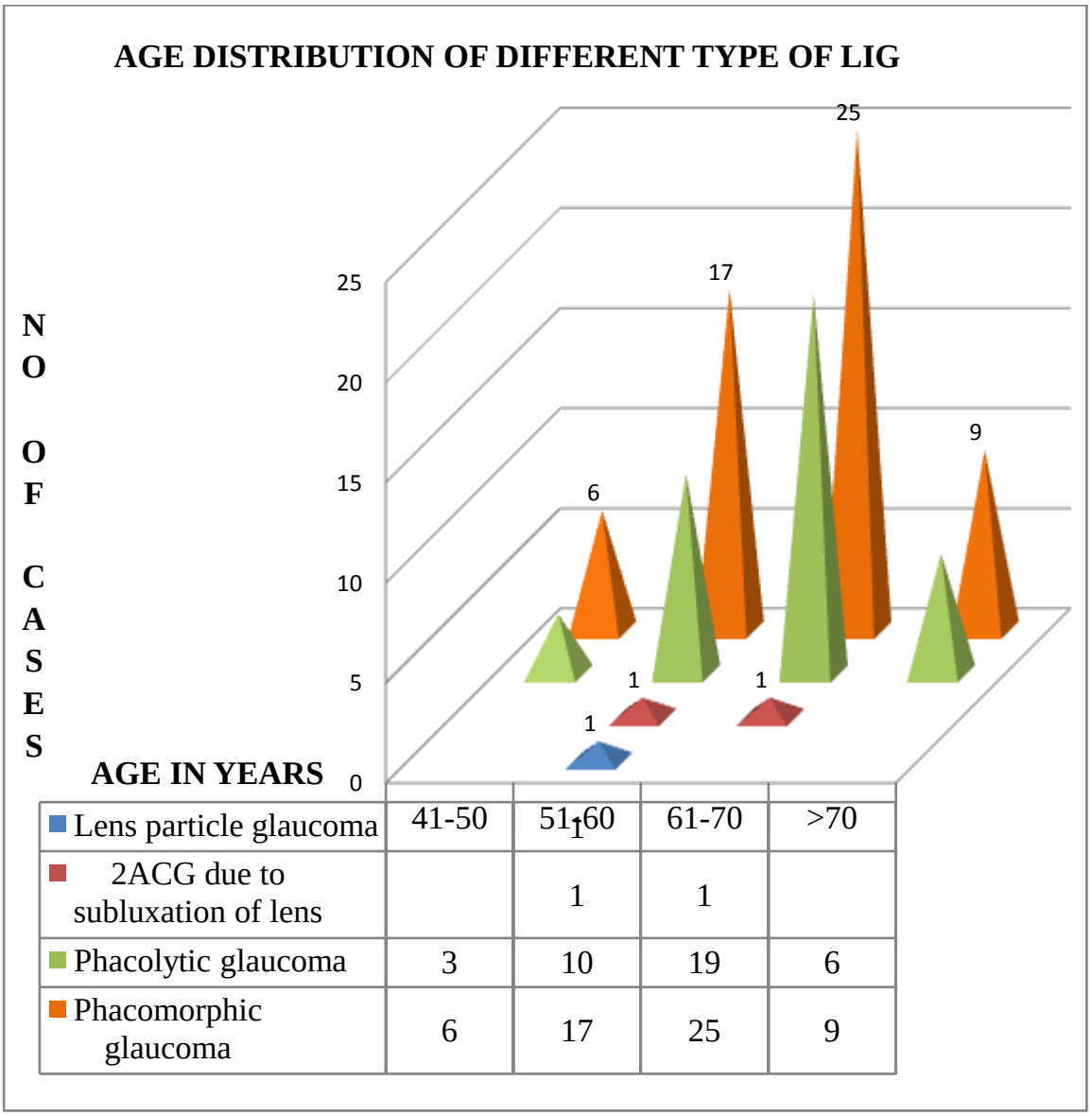



Majority of the patients were in the age group of 61– 70 years. We had 29 patients in the age group 51-60(29.59%), 15 patients between more than 70 years of age (15.30%) and 9 cases in the age group 41-50 years (9.18%). Youngest patient in the study was 45 years old and oldest patient was 93 years old.

**TABLE 2: AGE DISTRIBUTION AMONG DIFFERENT LIG**

| Age in years | Phacolytic glaucoma | Phacomorphic glaucoma | Lens particle glaucoma | 2ACG due to subluxation of lens | Total |
|---|---|---|---|---|---|
| 41-50 | 3 | 6 | | | 9 |
| 51-60 | 10 | 17 | 1 | 1 | 29 |
| 61-70 | 19 | 25 | | 1 | 45 |
| >70 | 6 | 9 | | | 15 |
| TOTAL | 38 | 57 | 1 | 2 | 98 |

In the age group of 61-70 years,19 cases (42.22%) of phacolytic glaucoma, 25 cases (55.56%) of phacomorphic glaucoma, 1 case (2.22%) of secondary angle closure glaucoma due to subluxation of lens anteriorly after trauma were found.




| | 41-50 | 51-60 | 61-70 | >70 |
|---|---|---|---|---|
| Lens particle glaucoma | | 1 | | |
| 2ACG due to subluxation of lens | | 1 | 1 | |
| Phacolytic glaucoma | 3 | 10 | 19 | 6 |
| Phacomorphic glaucoma | 6 | 17 | 25 | 9 |

Among the 38 cases of phacolytic glaucoma 18 cases (47.37%) belong to 61-70 years age group followed by 26.31% (10 cases) belonging to 51-60 years age.15.38% (6 cases) and 7.89% (3 cases) belong to more than 70 years and 41-50 years age group respectively.

Among the 57 cases of phacomorphic glaucoma 18 cases (43.85%) belong to 61-70 years age group followed by 29.82% (17 cases) belonging to 51-60 years age.15.79% (9 cases) and 10.52% (6 cases) belong to more than 70 years and 41-50 years age group respectively.

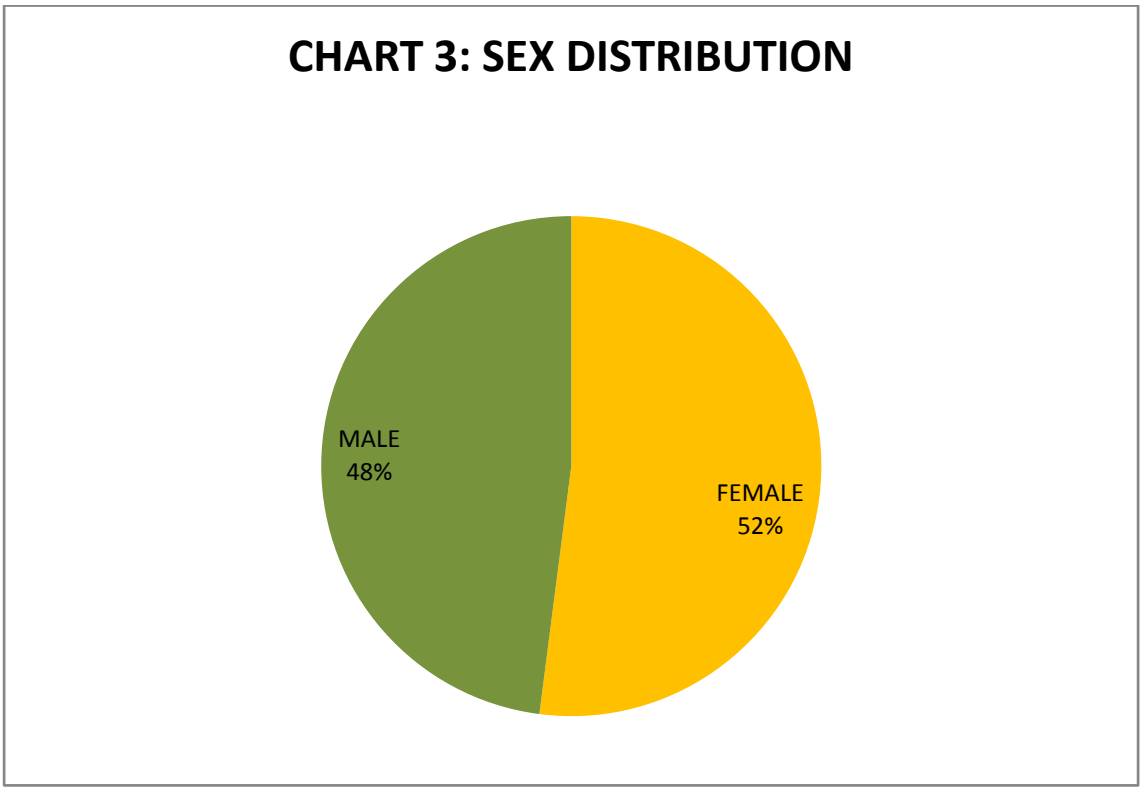
Lens particle glaucoma was found in 58 years patient and traumatic $2^0$ ACG was found 50% (1 case) in 51-60 years and 50%(1 case) 61-70 years.

## GENDER DISTRIBUTION

The following table shows the gender distribution in this study.

**TABLE 3: SEX DISTRIBUTION OF VARIOUS LIG**

| SEX | FREQUENCY | PERCENTAGE |
|---|---|---|
| MALE | 47 | 47.96 |
| FEMALE | 51 | 52.04 |
| | 98 | 100 |




Majority of the patients were females ( 51 patients, 52.04%).

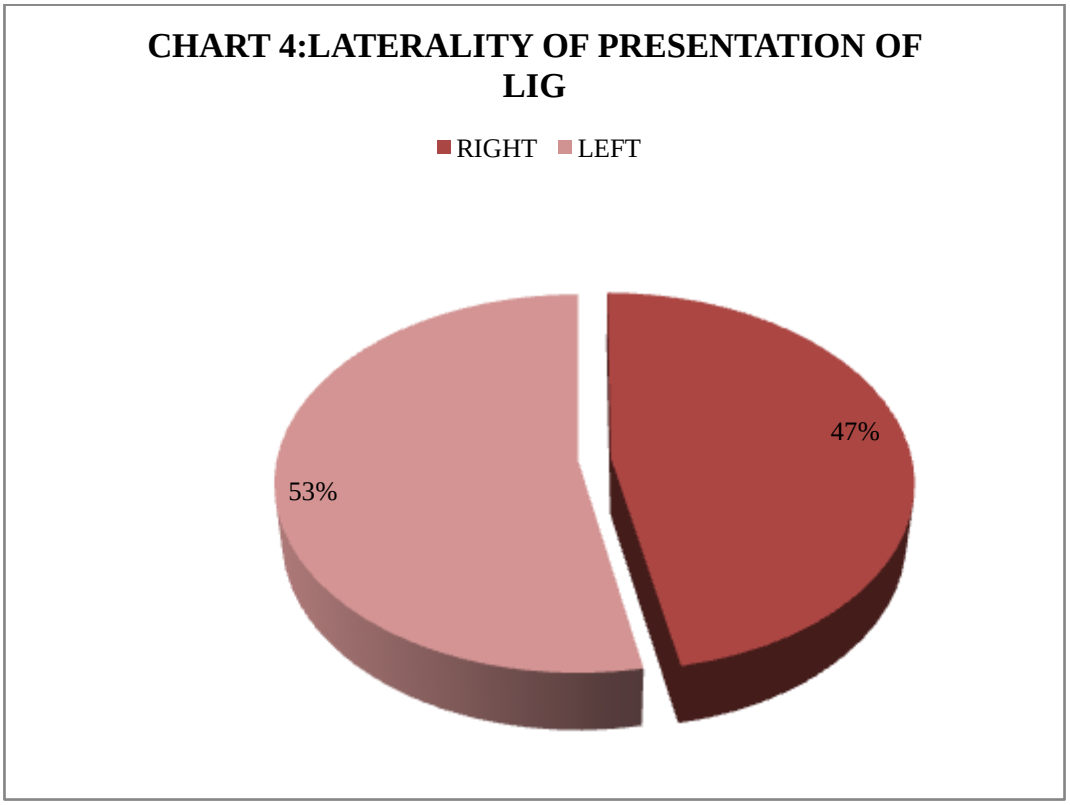
The incidence of LIG is slightly higher in females than in males(47 patients, 47.96%)

## EYE AFFECTED

## TABLE 4: EYE INVOLVED

| EYE INVOLVED | FREQUENCY | PERCENTAGE |
|---|---|---|
| RIGHT | 46 | 46.939 |
| LEFT | 52 | 53.061 |
| | 98 | 100 |

In 52 cases (53%), left eye was affected while in 46 cases (47%) left eye was affected.

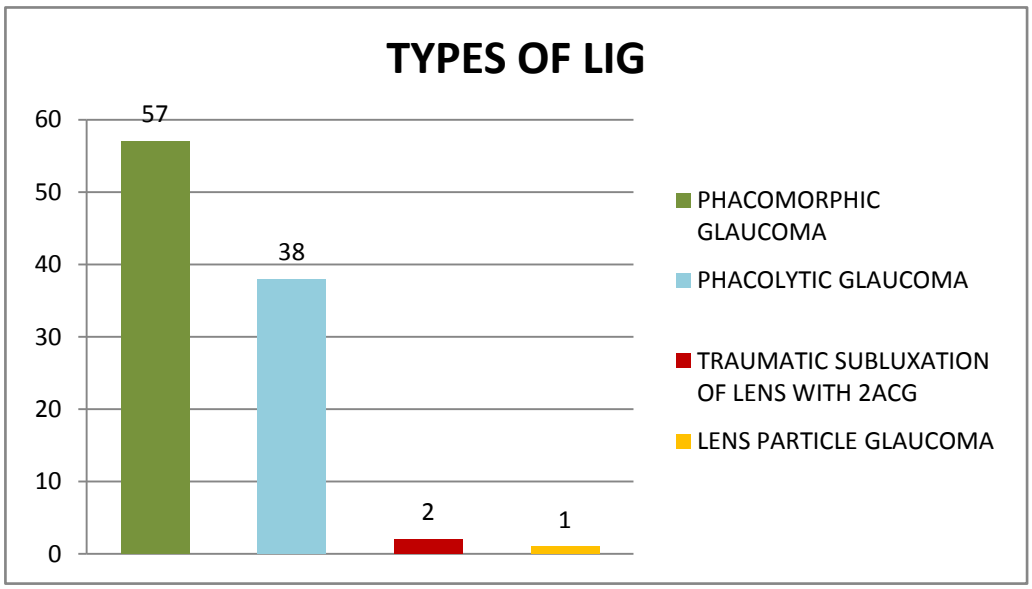


## AETIOLOGICAL DIAGNOSIS

## TABLE 5: AETIOLOGICAL DIAGNOSIS OF LIG

| TYPE OF GLAUCOMA | FREQUENCY | PERCENTAGE |
|---|---|---|
| PHACOMORPHIC GLAUCOMA | 57 | 58.16 |
| PHACOLYTIC GLAUCOMA | 38 | 38.77 |
| TRAUMATIC SUBLUXATION OF LENS WITH 2ACG | 2 | 2.04 |
| LENS PARTICLE GLAUCOMA | 1 | 1.02 |
| TOTAL | 98 | 100 |

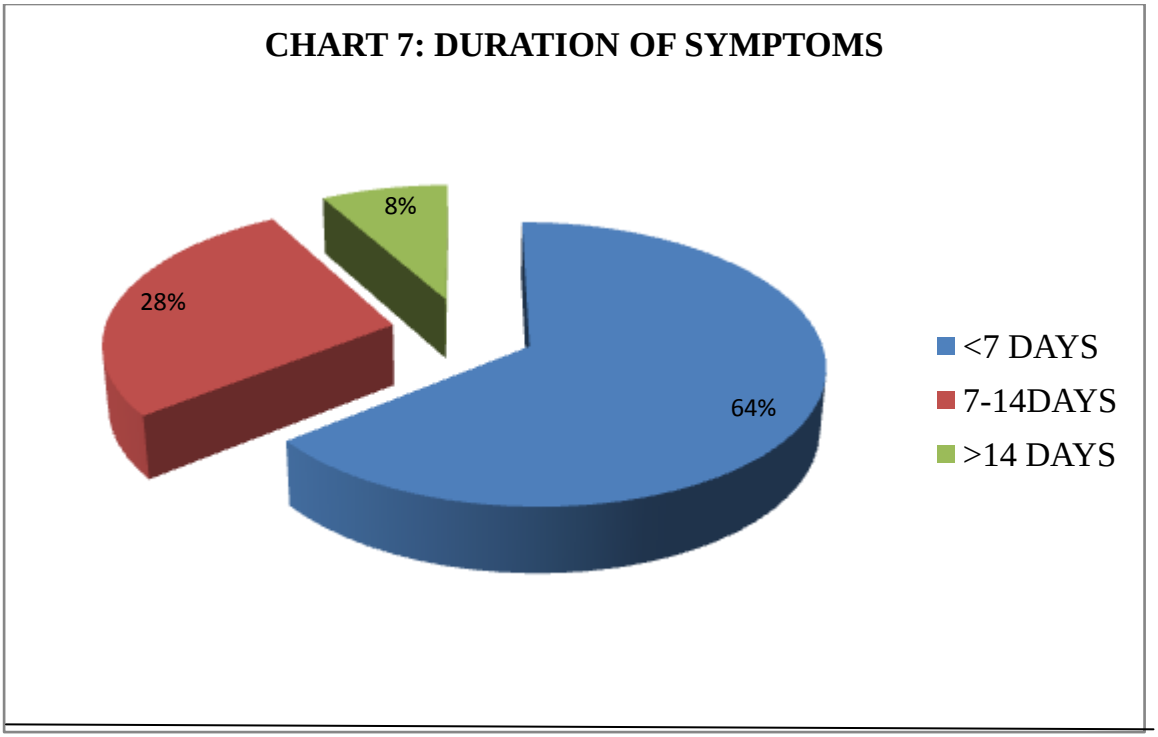
Out of 98 cases, 57 cases (58.16%) were diagnosed as phacomorphic glaucoma (PM), 38 cases (38.77%) were of phacolytic glaucoma (PL), 1 case (1.02%) was lens particle glaucoma (LP) and glaucoma secondary to subluxation/dislocation(S/D) of lens was found in 2 cases (2.04%).



## DURATION OF SYMPTOMS

## TABLE 7: DURATION OF SYMPTOMS

| DURATION OF SYMPTOMS | FREQUENCY | PERCENTAGE |
|---|---|---|
| <7 DAYS | 63 | 64.29 |
| 7-14DAYS | 27 | 27.55 |
| >14 DAYS | 8 | 8.16 |
| TOTAL | 98 | 100 |




Out of the 98 cases, 63 cases (64.29%) presented within the first week and 27 cases (27.55%) within 2nd week and rest presented more than 2 weeks after development of symptoms.

## STATUS OF OTHER EYE

## TABLE 8: STATUS OF OTHER EYE

| | FREQUENCY | PERCENTAGE |
|---|---|---|
| PSEUDOPHAKIA | 35 | 35.71 |
| IMMATURE CATARACT | 57 | 58.16 |
| MATURE CATARACT | 2 | 2.04 |
| CLEAR | 2 | 2.04 |
| APHAKIA | 1 | 1.02 |
| PHTHISICAL | 1 | 1.02 |
| | 98 | 100.00 |

In the 98 cases, 35 cases (35.71%) presented with pseudophakia, 57 cases (58.16%) presented with immature cataract and 2 cases (2.04%) each presented with mature cataract and clear lens, 1 case (1.02%), each of aphakia and phthisical eye was found.

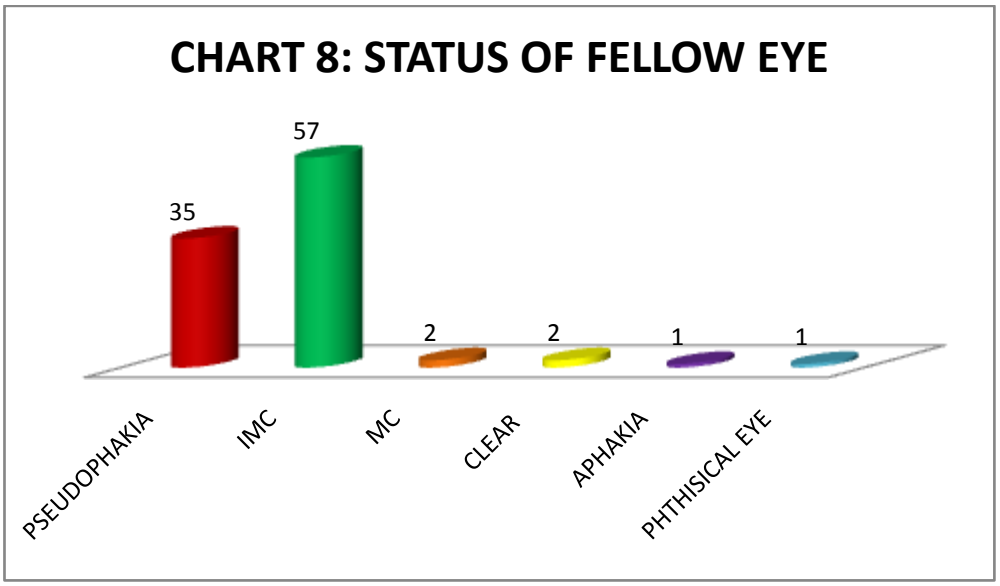
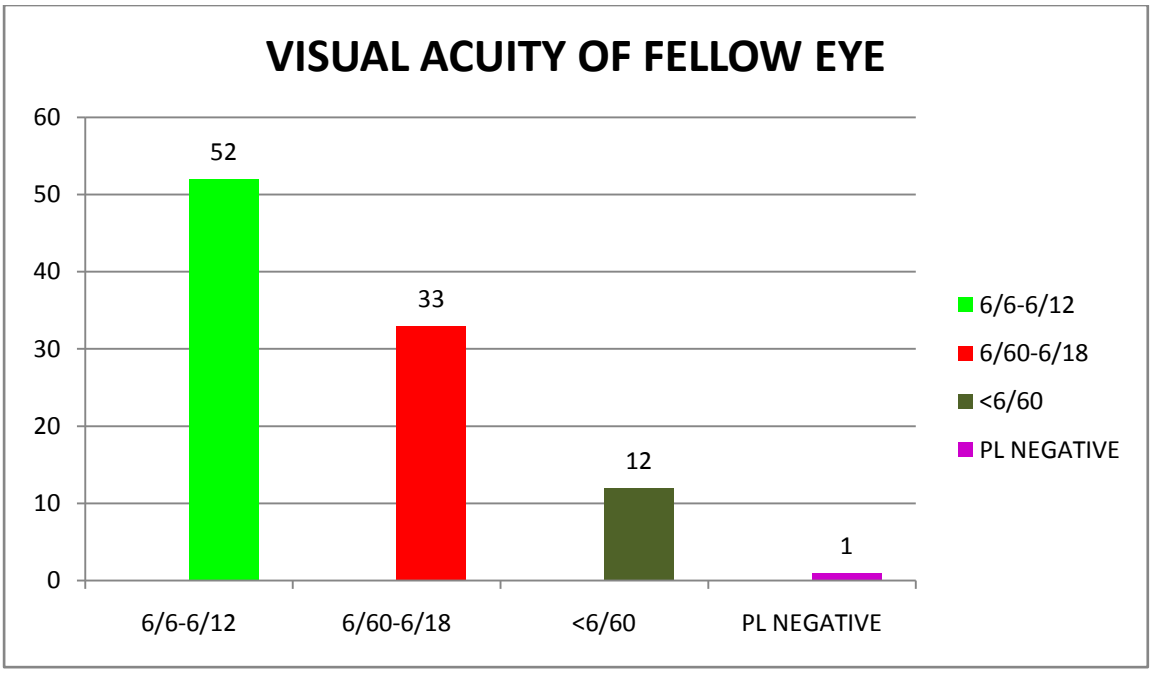


**TABLE 9 : VISUAL ACUITY OF FELLOW EYE**

| BCVA | FREQUENCY | PERCENTAGE |
|---|---|---|
| PL NEGATIVE | 1 | 1.02 |
| <6/60 | 12 | 12.24 |
| 6/60-6/18 | 33 | 33.67 |
| <6/18 | 52 | 53.06 |
| | 98 | 100.00 |




Overall, 52cases (53.06%) had good vision in the other eye i.e <6/18 while the remaining 33 cases (33.67%) had vision of 6/18-6/60 and 12 cases(12.24%) had vision of less than 6/60 in the other eye.one case who had phthisical eye had PL negative vision. This implies that with good vision in fellow eye, patient neglects the defective vision of the affected eye for a longer period of time.

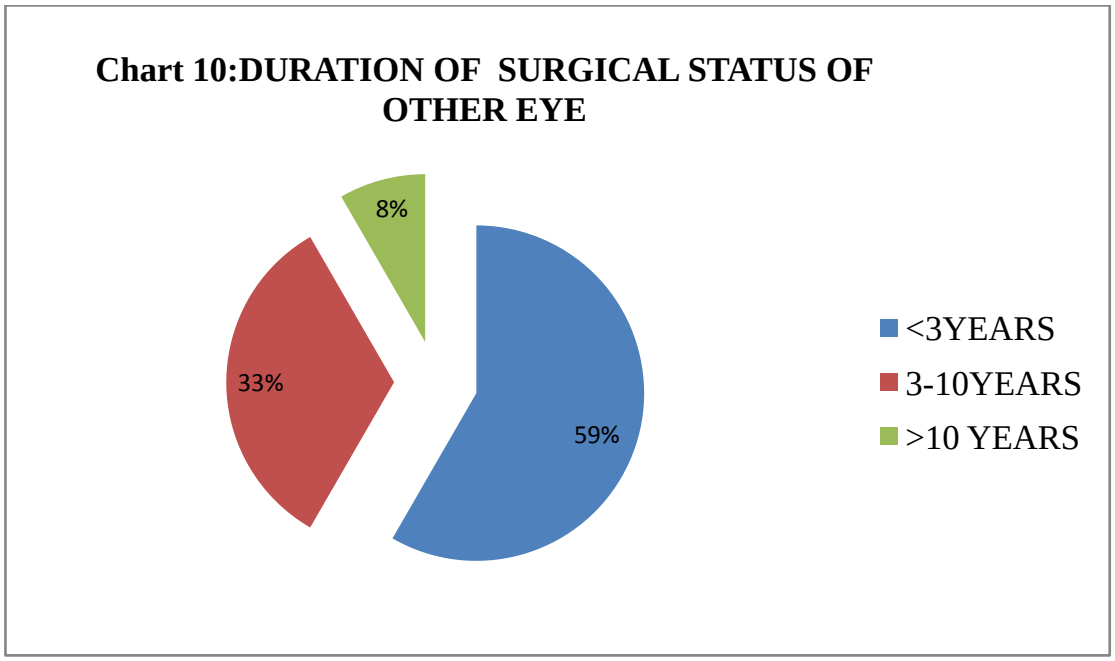



**TABLE 10: DURATION OF SURGICAL STATUS OF OTHER EYE**

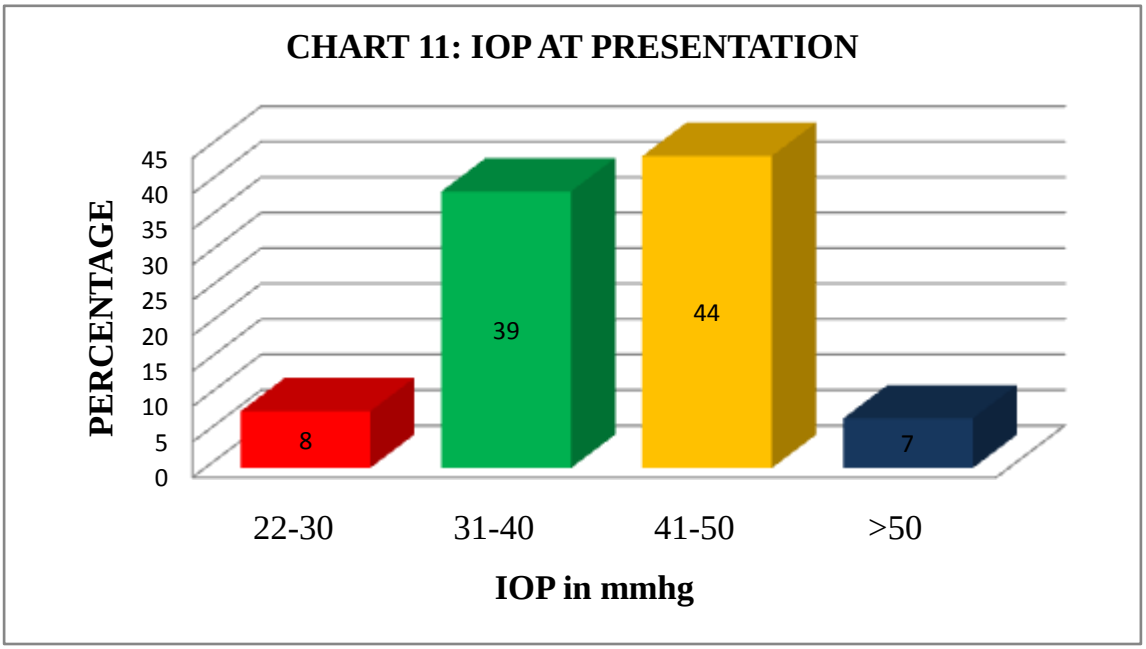
| DURATION | FREQUENCY | PERCENTAGE |
|---|---|---|
| <3YEARS | 21 | 58.33 |
| 3-10YEARS | 12 | 33.33 |
| >10 YEARS | 3 | 8.33 |
| | 36 | 100.00 |

Out of 36 cases, 21 cases (58.33%) got operated in less than 3 years, 12 cases(33.33%) got operated in between 3 years and 10 years and 3 cases(8.33 %) got operated 10 years ago. In our study there is a case of aphakia who got operated before 15 years.

**PREOPERATIVE INTRAOCULAR PRESSURE AT PRESENTATION:**

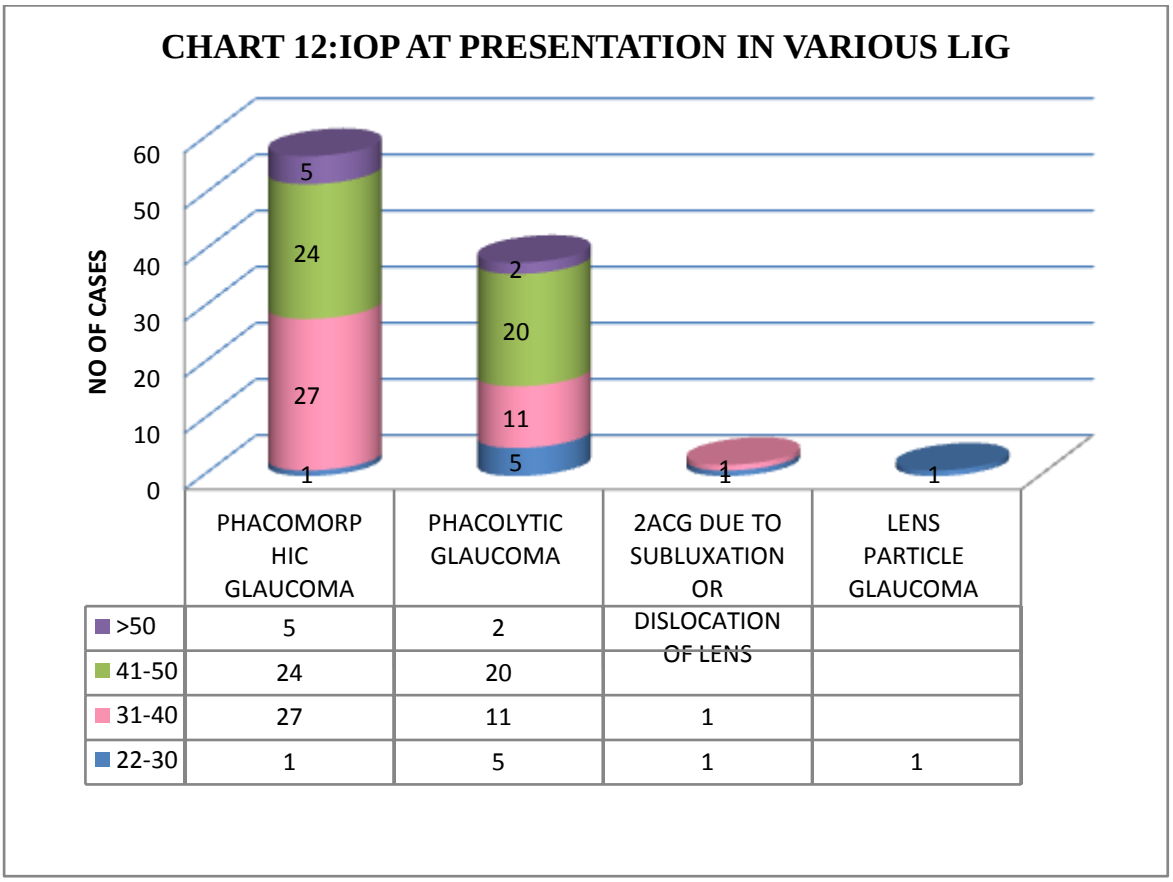
**TABLE 11: INTRAOCULAR PRESSURE AT PRESENTATION:**

| IOP AT PRESENTATION | FREQUENCY | PERCENTAGE |
|---|---|---|
| 22-30 | 8 | 8.16 |
| 31-40 | 39 | 39.80 |
| 41-50 | 44 | 44.90 |
| >50 | 7 | 7.14 |
| | 98 | 100 |




A total of 8 cases (8.16%) presented with IOP less than 30mm Hg, 39 cases (39.80%) with IOP between 30- 40mm Hg. 44 cases presented with IOP 40-50 mm Hg (44.90%)and 7 cases(7.14%) measured IOP of more than 50mmhg. The highest pressure recorded during the study was 58.00 mm Hg.

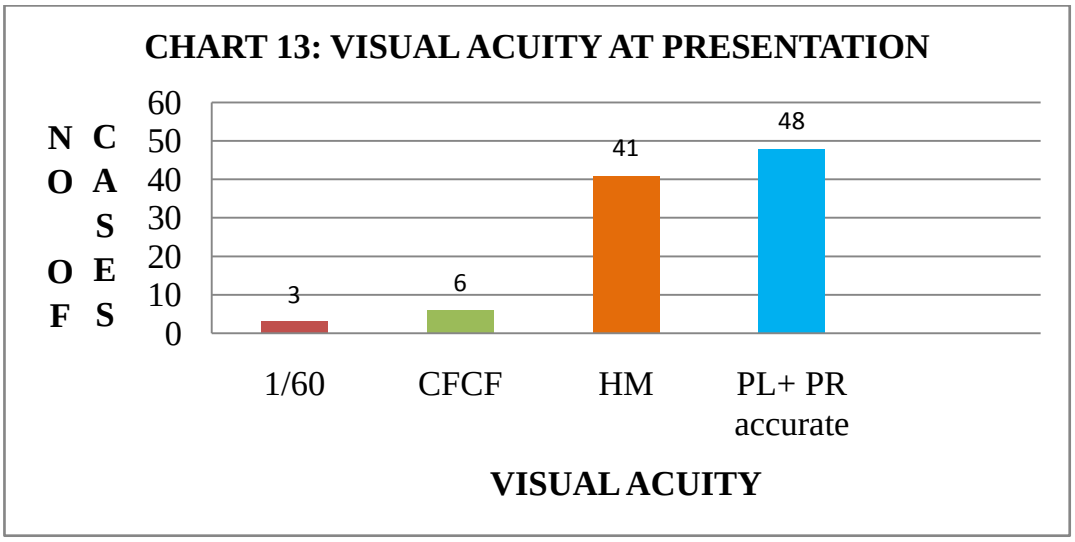
The range of intraocular pressure at the time of presentation of patients to OPD in various lens induced glaucomas in our study is depicted in chart 12.




## VISUAL ACUITY

## TABLE 12: VISUAL ACUITY AT PRESENTATION

| VISUAL ACUITY | FREQUENCY | PERCENTAGE |
|---|---|---|
| 1/60 | 3 | 3.06 |
| CFCF | 6 | 6.12 |
| HM | 41 | 41.84 |
| PL+ PR accurate | 48 | 48.98 |
| TOTAL | 98 | 100.00 |



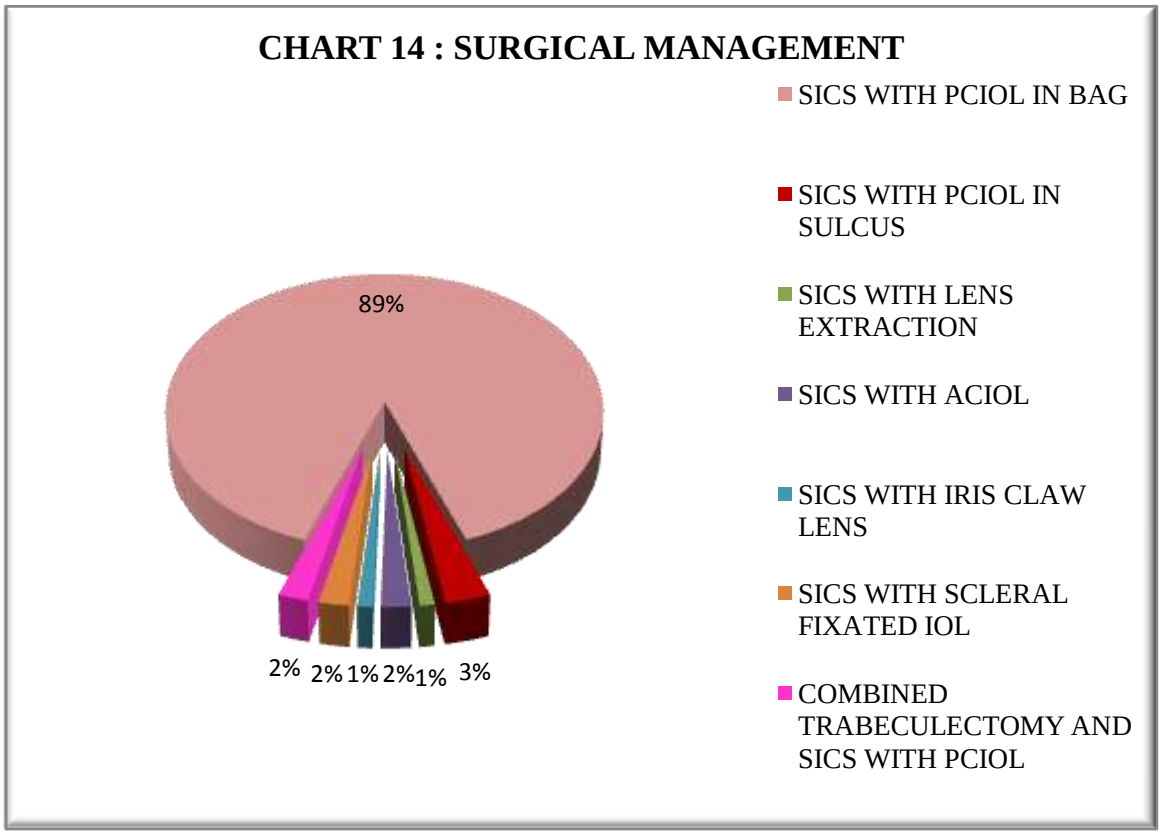

48 out of 98 cases (48.98%) had visual acuity of positive perception of light with accurate projection of light rays. Hand movements was present in 41 cases (41.84%), counting fingers close to face was present in 6 cases (6.12%) and counting fingers at the distance of one metre was found in 3 cases (3.06%).

**SURGICAL MANAGEMENT**

The following table shows the surgical management done in various lens induced glaucoma.

**TABLE 13: SURGICAL MANAGEMENT IN VARIOUS LIG**

| TYPE OF SURGERY | FREQUENCY | PERCENTAGE |
|---|---|---|
| SICS WITH PCIOL IN BAG | 87 | 88.77 |
| SICS WITH PCIOL IN SULCUS | 3 | 3.06 |
| SICS WITH LENS EXTRACTION | 1 | 1.02 |
| SICS WITH ACIOL | 2 | 2.04 |
| SICS WITH IRIS CLAW LENS | 1 | 1.02 |
| SICS WITH SCLERAL FIXATED IOL | 2 | 2.04 |
| COMBINED TRABECULECTOMY AND SICS WITH PCIOL | 2 | 2.04 |
| | 98 | 100 |



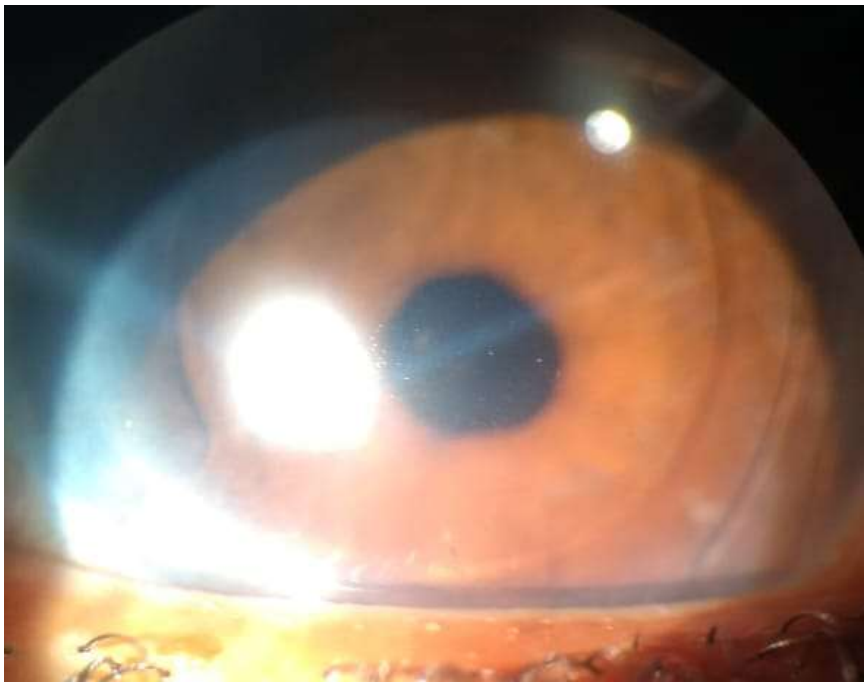



**FIGURE 20 : ANTERIOR CHAMBER IOL**

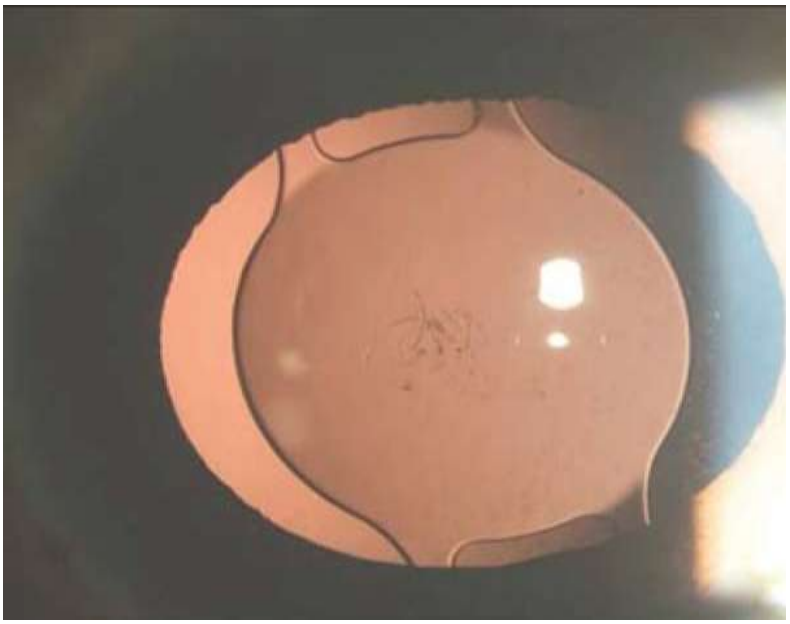


**FIGURE 21 : IRIS CLAW LENS**

Among 98 patients who underwent manual small incision cataract surgery, Posterior Chamber Intraocular Lens (PCIOL) was kept in capsular bag in 87 cases (88.78%). In 3.06% i.e in 3 cases PCIOL was placed in sulcus. Peripheral iridectomy was done in cases of phacomorphic glaucoma for more than 1 week. One patient (1.02%) was left aphakic after lens extraction as the patient's other eye status was aphakia. Anterior chamber Intraocular Lens was placed in 2 cases (2.04%) and scleral fixated lens was placed in 2 cases (2.04%). 1 patient underwent SICS with Iris Claw lens implantation accounting for 1.02%. 2 patients (2.04%) underwent combined trabeculectomy with SICS with placement of PCIOL in the capsular bag. They presented after 2 weeks of development of symptoms and had peripheral anterior synechiae in gonioscopy.

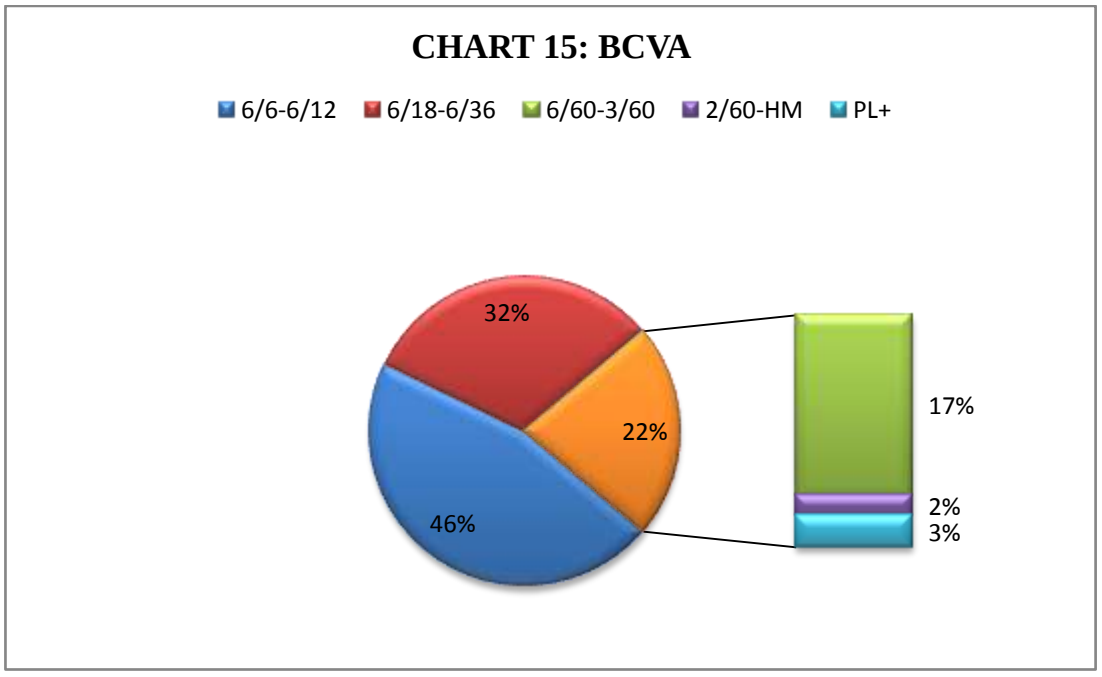
Except the patients who were done SICS with PCIOL implantation in the bag or sulcus, all others had visual acuity of ≤6/24.

## VISUAL OUTCOME AFTER SICS

The following table shows the Best corrected visual acuity (BCVA) attained by the patient after 1 month of cataract surgery.

## TABLE 14: BEST CORRECTED VISUAL ACUITY AFTER 1 MONTH OF SURGERY

| BCVA | FREQUENCY | PERCENTAGE |
|---|---|---|
| 6/6-6/12 | 45 | 45.92 |
| 6/18-6/36 | 31 | 31.63 |
| 6/60-3/60 | 17 | 17.35 |
| 2/60-HM | 2 | 2.04 |
| PL+ | 3 | 3.06 |
| TOTAL | 98 | 100 |



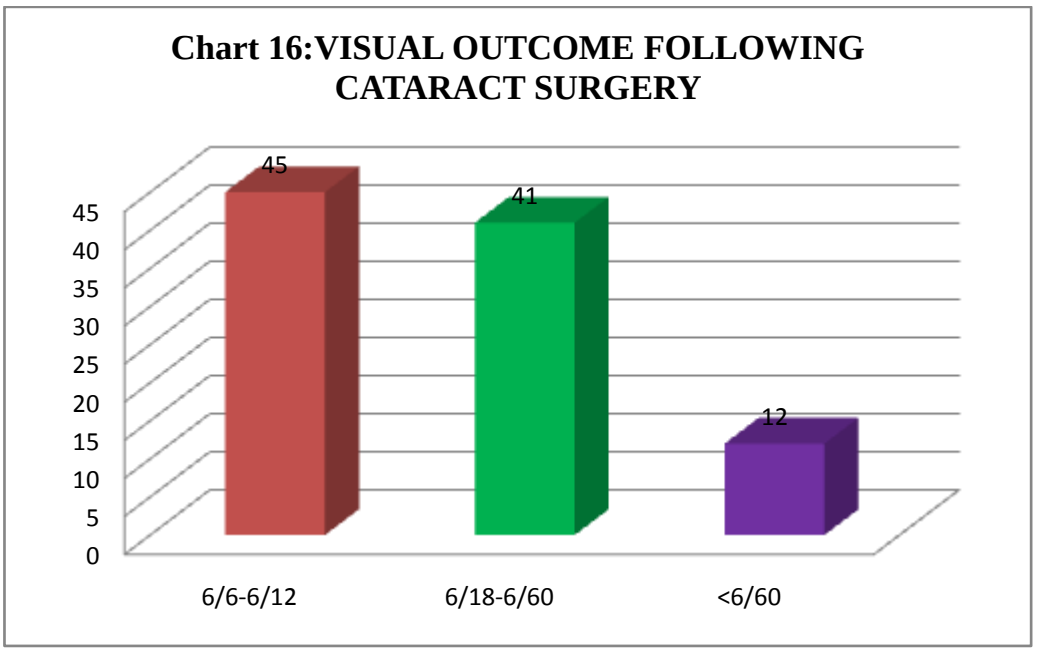

The chart 15 showed 3 patients had perception of light.

## DURATION OF SYMPTOMS AND BCVA

For analysing the visual outcome following cataract surgery, the grading of BCVA at the end of one month is done as per WHO guidelines as follows:

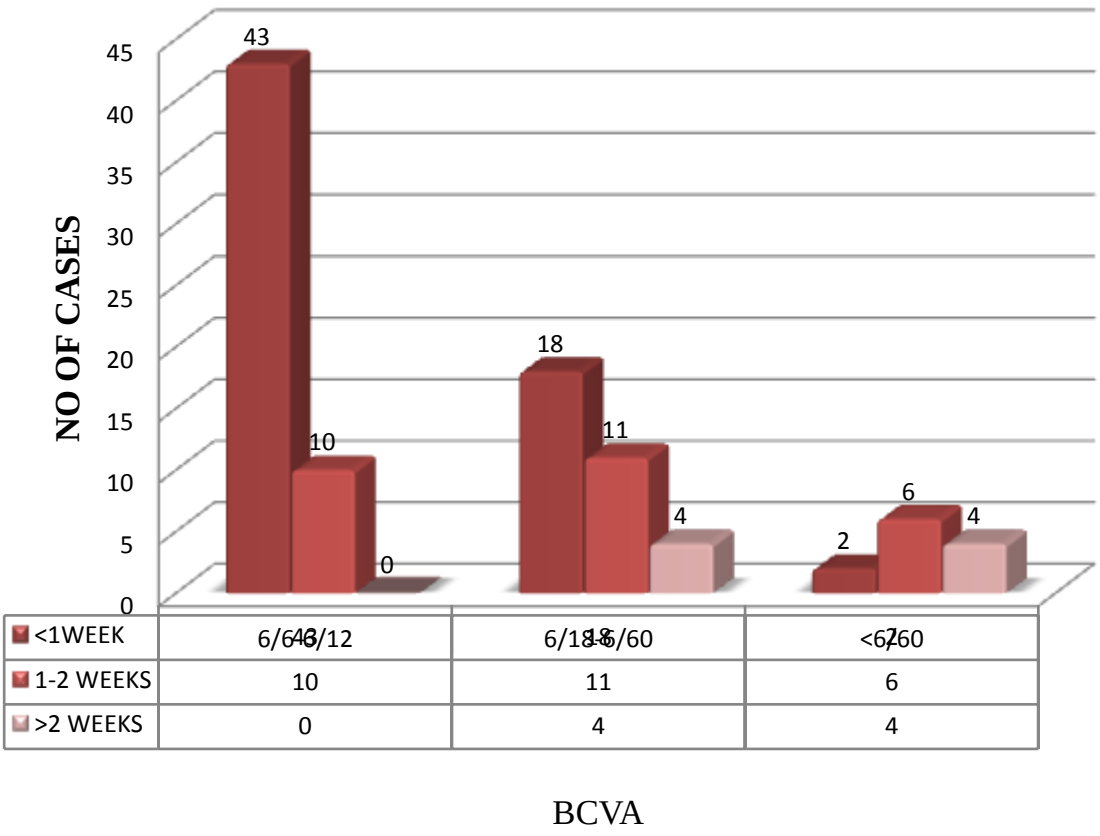
**TABLE 15: GRADING OF VISUAL OUTCOME:**

| GRADING | BCVA | FREQUENCY | PERCENTAGE |
|---|---|---|---|
| GOOD | 6/6-6/12 | 45 | 45.92 |
| BORDERLINE | 6/18-6/60 | 41 | 41.84 |
| POOR | <6/60 | 12 | 12.24 |
| | | 98 | 100 |




Poor visual outcome of less than 6/60 visual acuity was found in 12.24% of cases, borderline visual acuity(6/18-6/60) was found in 41.63% cases and good visual outcome (>6/18) was found in 45.92% cases.

**CHART 17: DURATION OF SYMPTOMS AND BCVA**




The above chart describes that the BCVA after cataract surgery is good (≤6/18)in 53 cases, of them 43 cases( 81.13%)were presenting earlier that is less than 1 week and in 10 cases (18.87%) presented between one to two weeks . The BCVA is borderline (6/24-6/60) in 33 cases, among them 18 cases (43.90%) presented in less than one week, 11cases (26.83%) presented between one to two weeks and 4 cases (9.76%) presented more than 2 weeks. The poor visual outcome (BCVA<6/60) was found in 4 cases. In 4 cases presenting late i.e after 2 weeks which accounts to 33.33%, 6 cases presenting between one to two weeks (50%), and 2 cases (16.67%) who presented early in less than one week also had poor visual outcome.

**TABLE 16: DURATION OF SYMPTOMS AND BCVA**

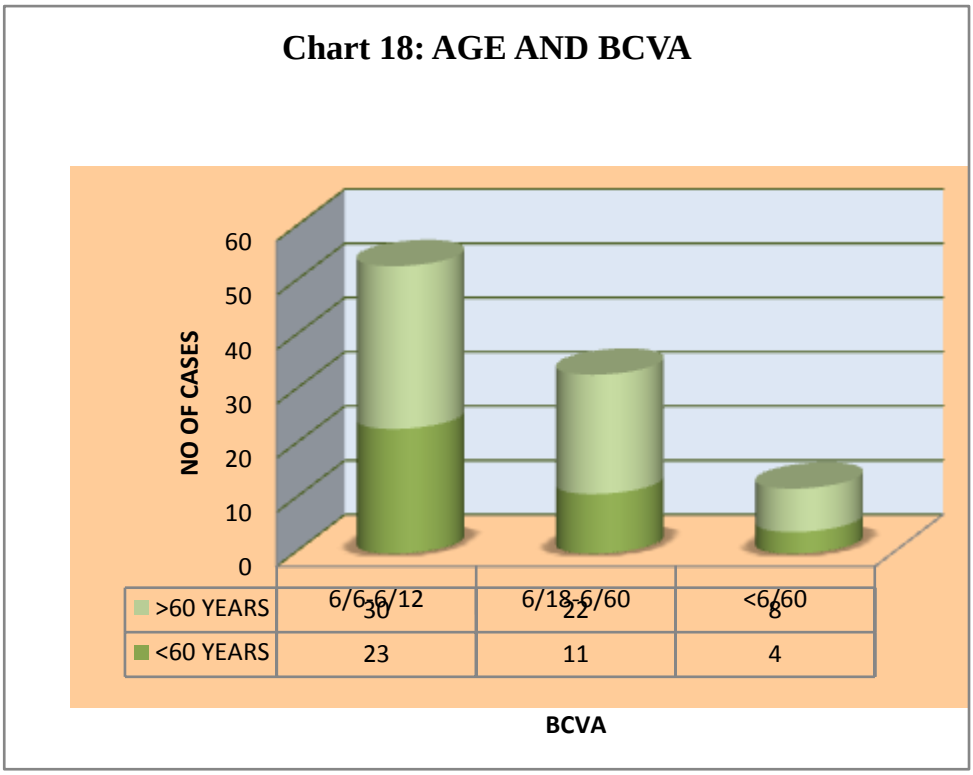
| PARAMETER | BCVA | N | MEAN(95% CONFIDENCE INTERVAL) |
|---|---|---|---|
| DURATION IN DAYS | 1 | 53 | 5.03(4.25-5.82) |
| | 2 | 33 | 7.09(5.54-8.63) |
| | 3 | 12 | 10.5(6.77-14.22 |
| | Total | 98 | 6.39(5.55-7.24) |

There is a statistical significance between duration of symptoms and the BCVA as the p value <0.01%.

**AGE OF PATIENT AND VISUAL OUTCOME**

The following chart describes the relation of age of patients in the study group and their final BCVA:

**Chart 18: AGE AND BCVA**



In the age group of less than sixty years, visual acuity was poor in 4 cases (10.52%), borderline in 11 cases (36.67%) and good in 23 cases (60.53%).

In our study, age group of more than sixty years were 60 cases, out of them 30 cases (50%) had good vision, 22 cases (36.67%) had borderline vision, 8 cases (13.33%) had poor vision.

The following table 17 gives the 95% confidence level between age and BCVA.

| PARAMETER | BCVA | n | MEAN(95% CONFIDENCE INTERVAL) |
|---|---|---|---|
| AGE | 6/6 to6/12 | 53 | 62.37(60.25-64.49) |
| | 6/18-6/60 | 33 | 64.33(61.34-67.32) |
| | <6/60 | 12 | 68.5(60.10-76.89) |
| | Total | 98 | 63.78(62.00-65.56) |

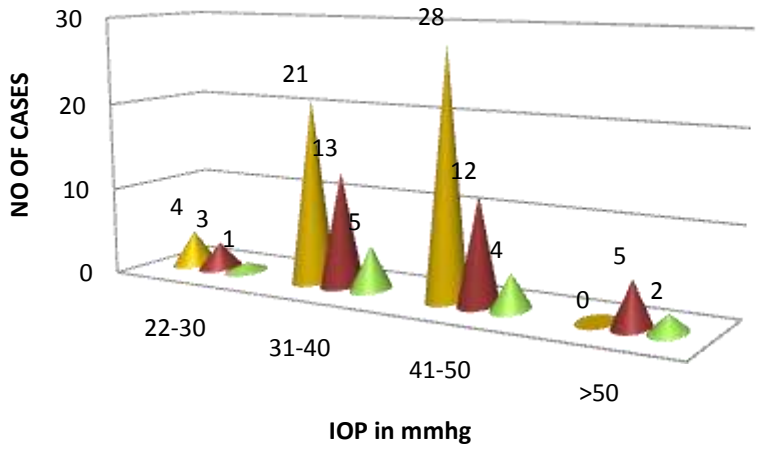
| Dependent Variable | (I) Group | (J) Group | Mean Difference (I-J) | Std. Error | Sig. | 95% Confidence Interval | |
|---|---|---|---|---|---|---|---|
| | | | | | | Lower Bound | Upper Bound |
| AGE | 1.00 | 2.00 | -1.95 | 1.94 | .316 | -5.8083 | 1.8963 |
| | | 3.00 | -6.12 | 2.79 | .031 | -11.6765 | -.5688 |
| | 2.00 | 1.00 | 1.95 | 1.94 | .316 | -1.8963 | 5.8083 |
| | | 3.00 | -4.16 | 2.94 | .161 | -10.0230 | 1.6896 |
| | 3.00 | 1.00 | 6.12 | 2.79 | .031 | .5688 | 11.6765 |

When comparing groups of good and poor visual outcome there is statistical significance of p value less than 0.05(p=0.031).

## BCVA AND IOP AT PRESENTATION

The IOP at presentation and best corrected visual acuity was evaluated and the following tabulation is made.

**Chart 19: BCVA AND IOP AT PRESENTATION**




| | 22-30 | 31-40 | 41-50 | >50 |
|---|---|---|---|---|
| 6/6-6/12 | 4 | 21 | 28 | 0 |
| 6/18-6/60 | 3 | 13 | 12 | 5 |
| >6/60 | 1 | 5 | 4 | 2 |

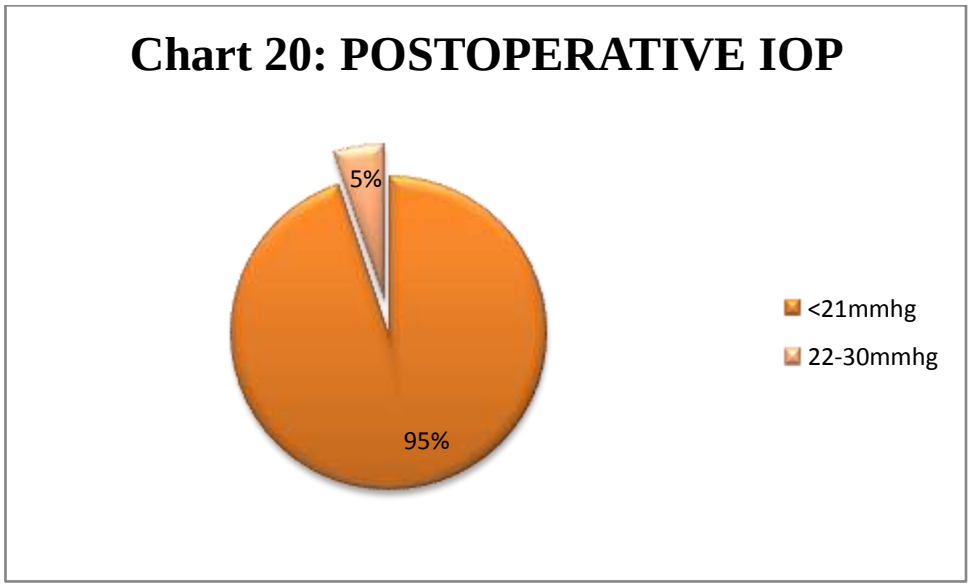
The mean IOP at presentation was 40.71±7.02. The patients with poor visual acuity of less than 6/60 had IOP at the time of admission as more than 50mmhg in 2 cases(16.67%), 41 to 50mmhg in 4 cases(33.33%), 31 to 40mmhg in 5 cases (41.67%) and 22to 30mmhg in (8.33%) 1 case.

When IOP at presentation and and BCVA at the end of one month are related, the p value is more than 0.05(p =0.54) which narrates that both are not statistically significant.

**TABLE 18: IOP AT PRESENTATION AND BCVA**

| PARAMETER | BCVA | n | MEAN(95% CONFIDENCE INTERVAL) |
|---|---|---|---|
| IOP | 6/6 to6/12 | 53 | 40.15(38.47-41.83) |
| | 6/18-6/60 | 33 | 41.81(39.10-44.54) |
| | <6/60 | 12 | 40.16(34.42-45.91) |
| | Total | 98 | 40.71(39.31-42.12) |

**POST OPERATIVE INTRAOCULAR PRESSURE**



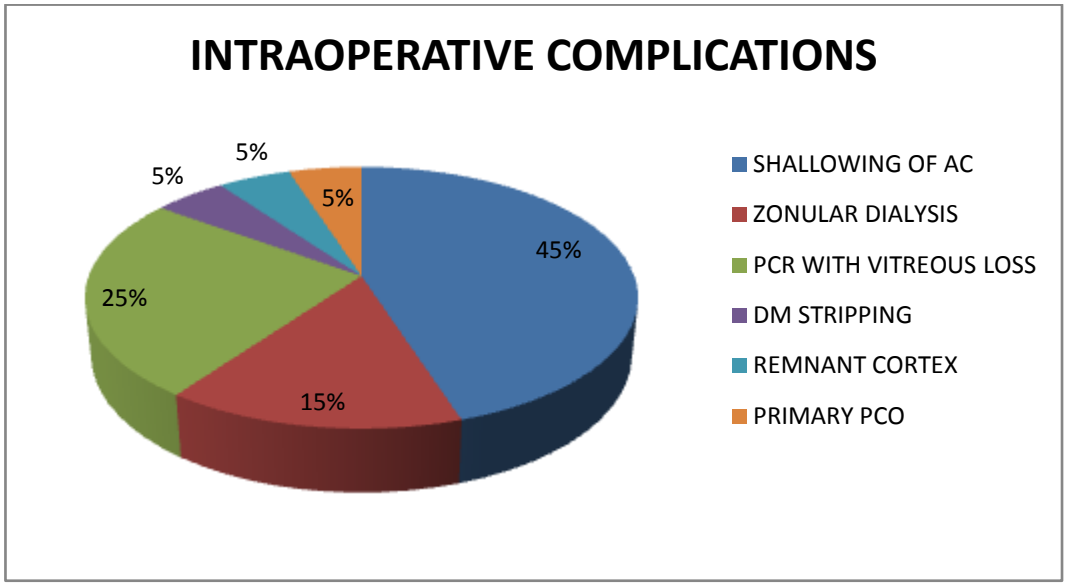

The IOP recorded in the post operative period during discharge was found within normal limits in 95% of cases and 5% had raise in IOP more than 22 mmhg. Of the 5 cases with raise in IOP, 3 cases were of phacolytic glaucoma (40%) and 2 cases were of phacomorphic glaucoma (60%). The IOP was controlled with topical antiglaucoma medications and vigorous treatment of inflammation with topical steroids.

## POSTOPERATIVE AND INTRAOPERATIVE COMPLICATIONS:

The following table shows the intraoperative complications (21.43%) and post operative complications (15.31%) encountered during and after surgery respectively.

**TABLE 19: INTRAOPERATIVE COMPLICATIONS**

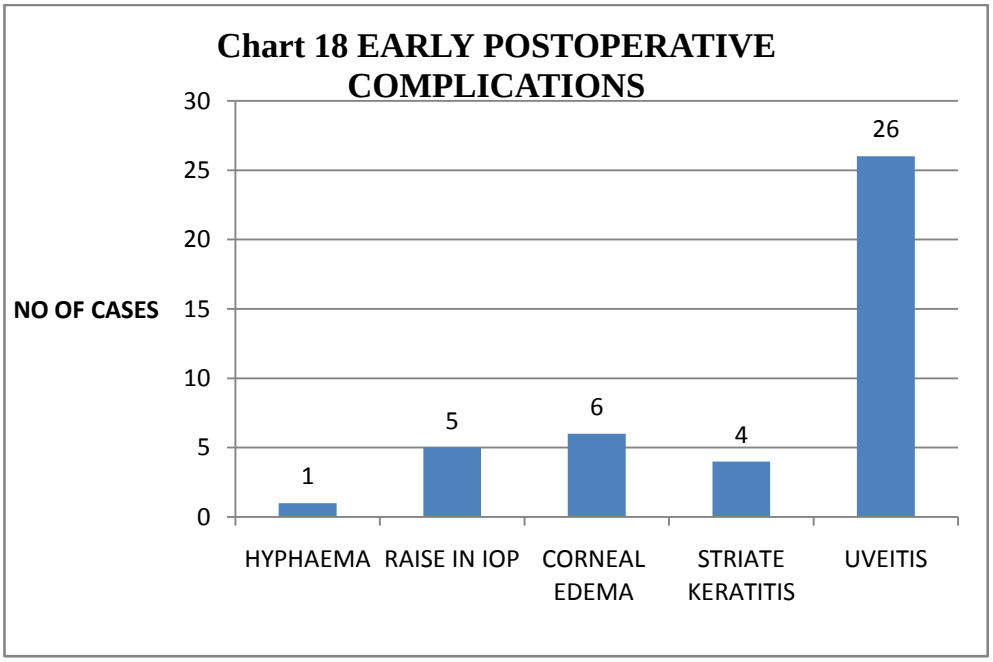
| COMPLICATION | N | % |
|---|---|---|
| SHALLOWING OF AC | 9 | 9.18 |
| ZONULAR DIALYSIS | 3 | 3.06 |
| PCR WITH VITREOUS LOSS | 5 | 5.10 |
| PRIMARY PCO | 1 | 1.02 |
| DM STRIPPING | 1 | 1.02 |
| REMNANT CORTEX | 1 | 1.02 |
| TOTAL | 20 | 20.41% |




The shallowing of anterior chamber is the most common intraoperative complication (9.18%)noticed in 9 cases in our study.Posterior capsular rent with vitreous loss was found in 26% of cases. Primary Posterior Capsular Opacification was noted in one case.

**TABLE 20: EARLY POST OPERATIVE COMPLICATIONS**

| COMPLICATION | N | % |
|---|---|---|
| RAISE IN IOP | 5 | 5.10 |
| CORNEAL EDEMA | 6 | 6.12 |
| STRIATE KERATITIS | 4 | 4.08 |
| UVEITIS | 27 | 27.55 |



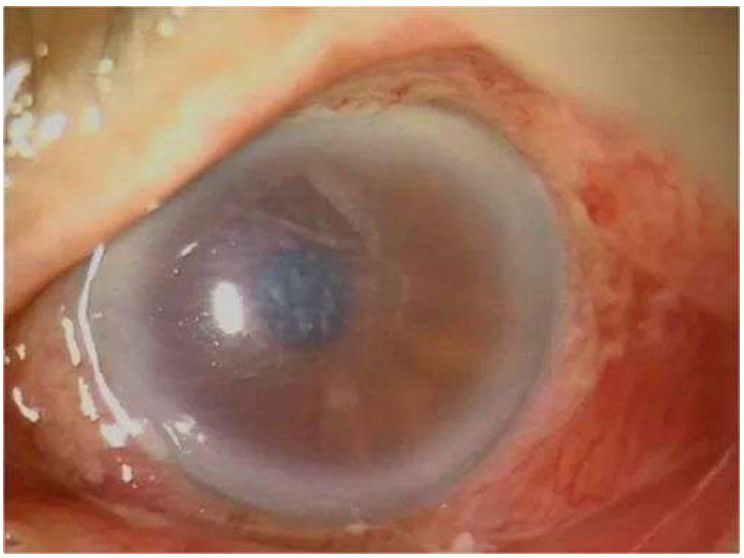



**FIGURE 22 : STRIATE KERATOPATHY**

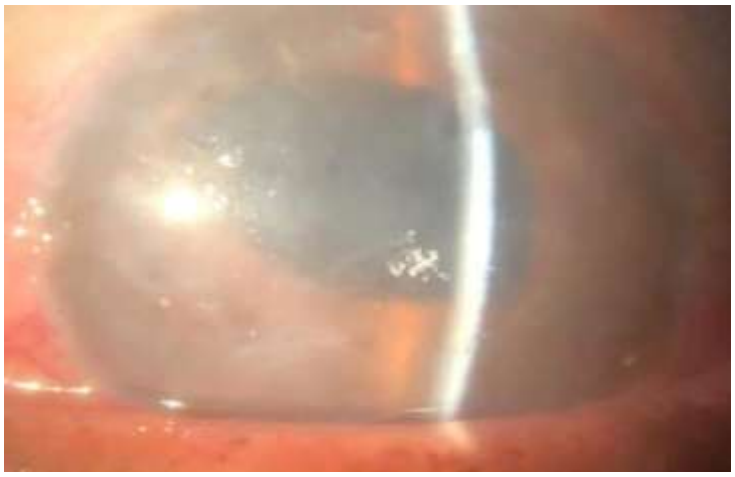
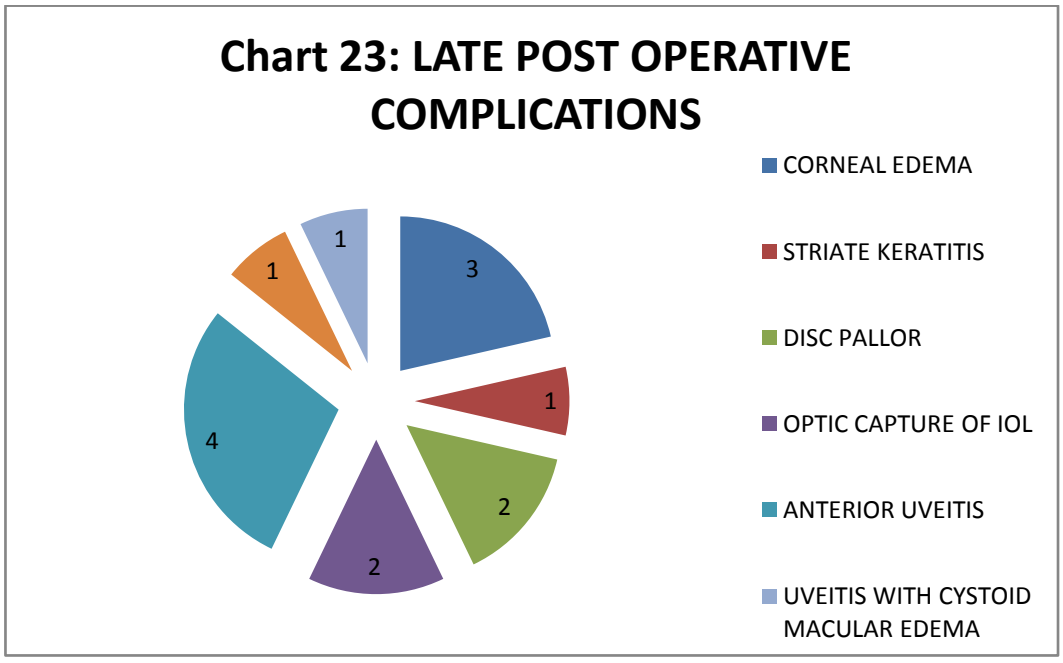


**FIGURE 24 : CORNEAL EDEMA**

Out of 98 cases, 26 cases (26.53%) had postoperative uveitis, of which 14 cases had phacolytic glaucoma(53.84%), 10 cases had phacomorphic glaucoma(38.46%) and 2 cases had traumatic 2ACG due to subluxation of lens one case of lens particle glaucoma. They were treated with topical steroid eye drops. 6 cases(6.12%) had corneal edema and 4 cases had striate keratitis and they were treated topical 5% hypertonic saline and at the end on one month 3 cases persisted to have corneal edema and 1 case of striate keratitis was present.



The following table shows late postoperative complications at the end of one month found in our study.

**TABLE 21: LATE POST OPERATIVE COMPLICATIONS**

| COMPLICATION | N | % |
|---|---|---|
| CORNEAL EDEMA | 3 | 3.06 |
| STRIATE KERATITIS | 1 | 1.02 |
| PRE EXISTING DISC PALLOR | 2 | 2.04 |
| OPTIC CAPTURE OF IOL | 2 | 2.04 |
| ANTERIOR UVEITIS | 4 | 4.08 |
| UVEITIS WITH CYSTOID MACULAR EDEMA | 1 | 1.02 |
| TOTAL | 14 | 14.29 |

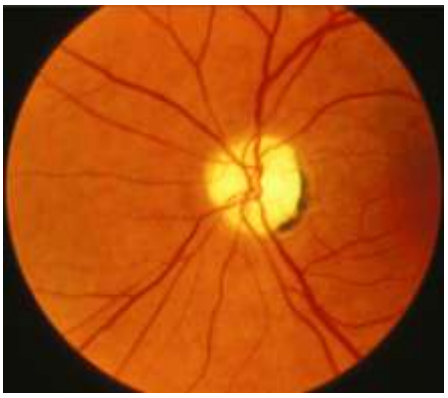


**FIGURE 25 : PALLOR OF OPTIC DISC**

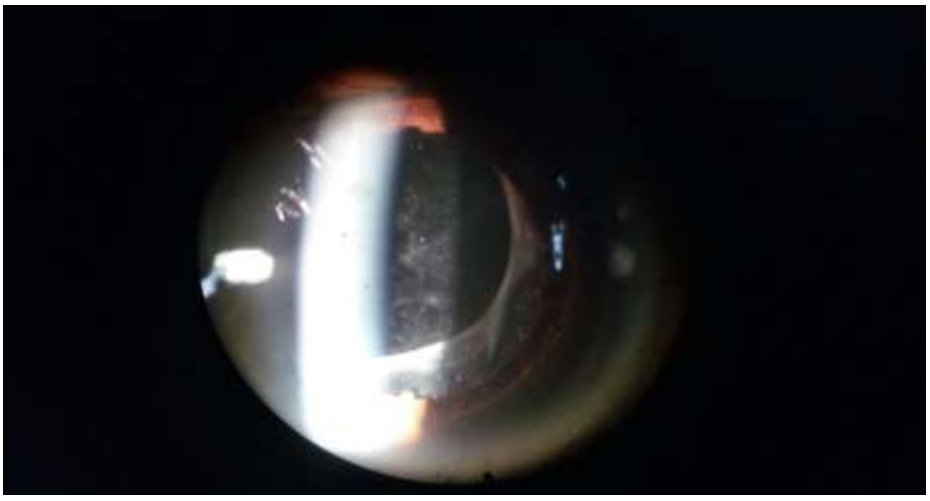


**FIGURE 26 : POSTERIOR CAPSULAR OPACIFICATION**

One case each of posterior capsular opacification, uveitis with cystoid macular edema and striate keratitis was found in our study. Disc pallor and optic capture of IOL was present in 2 cases each. Corneal edema was present in 3 cases and 4 cases had uveitis. All these complications resulted in the reduction of the visual outcome following cataract surgery for LIG.

# DISCUSSION

Lens induced glaucomas are a common occurrence in India, where the there is an increase in the cataract cases than the number of surgeries performed. The main cause is that these are lens induced which compromise the function of the optic nerve due to rise of intraocular pressure. Cataract surgery is definitive treatment for these cases. The final visual outcome is uniformly guarded(39).

This cross sectional observational study was undertaken to study the different types of LIG. This study included 98 patients with various types of lens induced glaucomas. The patients were followed up from the time of admission and surgery till 4 weeks post-operatively. The factors influencing the final visual outcome were analysed.

For analyzing statistically Chi-Square test ($\chi^2$) and Probability values (p) were used and p-value < 0.05 was considered statistically significant.

## AGE DISTRIBUTION:

In this study, age range was 45 to 93 years with a mean age of 63.78±8.88 years.

Highest number of cases occurred in the age group 61-70 years (45.92%), indicating that the lens- induced glaucoma is a condition of old age.

Pradhan et al(37) has showed that in the age range of 40 to 80 years with 35 % of cases occurring in less than 60 years and the rest more than 60 years.

## SEX DISTRIBUTION:

In this study, female preponderance is observed. The ratio of female to male is 1.08:1. A study done at Madurai by Prajna in 1996(4), has found marginally significant increased risk of having these glaucomas, in females (p=0.05).[5]

In Pradhan et al(37) has reported female to male ratio of 1.7:1.

## LATERALITY:

All cases of lens induced glaucomas studied were unilateral. In 52 cases (53.06%) left was affected while in 46 cases (46.94%) right eye was affected.

## TYPE OF LENS INDUCED GLAUCOMA

In our study phacomorphic glaucoma is the most common LIG 58.16% followed by phacolytic glaucoma contributing to 58.16%.

Distribution of various lens induced glaucoma in different studies are tabulated below.

| | **Phacomorphic glaucoma** | **Phacolytic glaucoma** |
|---|---|---|
| Prajna et. al. (1996)(4) | 52.7 % | 47.3 % |
| Pradhan et.al.(2001)(37) | 72 % | 28 % |
| Rijal AP (2006)(40) | 65 % | 35 % |
| Murty et. al. (2015)(41) | 62 % | 38 % |
| Bhuyan et al(2016) (42) | 58.86%) | 33.33% |
| Our study | 58.16% | 38.77% |

**DURATION OF SYMPTOMS AND BCVA:**

Out of the 98 cases, 63 cases (64.29%) presented within the first week and 27 cases (27.55%) within 2nd week and rest presented more than 2 weeks after development of symptoms.

In our study the BCVA after cataract surgery is good (>6/18)in 53 cases, of them 43 cases( 81.13%)were presenting earlier that is less than 1 week and in 10 cases (18.87%) presented between one to two weeks . The BCVA is borderline (6/18-6/60) in 33 cases, among them 18 cases (43.90%) presented in less than one week, 11cases (26.83%) presented between one to two weeks and 4 cases (9.76%) presented more than 2 weeks. The poor visual outcome (BCVA<6/60) was found in 4 cases. In 4 cases presenting late i.e after 2 weeks which accounts to 33.33%, 6 cases presenting between 1 to 2 weeks (50%), and 2 cases (16.67%) who presented early in less than one week also had poor visual outcome.

There is statistical significance between duration of symptoms and the BCVA as the p value <0.01% which implies more the delay in the presentation, poorer was the visual outcome.

The Lahan study by Pradhan et al(37), found that duration of pain and high IOP at presentation in phacomorphic group was associated with poor visual outcome at discharge.(43)[38]

Prajna et al(4) has found that the duration of symptoms exceeding 5 days is a significant risk factor for poor visual outcome in LIG. In study by Bhuyan et al(42),

52.64 % cases presented earlier than 7 days and 74.07% had good visual outcome. It showed that a significant risk of poor visual acuity was found when the duration between the onset of symptom and presentation to our OPD was more than 7days (p value= 0.0249).

**AGE AND BCVA**

When comparing between the groups of good and poor visual outcome there is a statistical association between age and final visual outcome, as the p value is less than 0.05 (p=0.031).

In the study by Prajna et al(4) there is significant risk of poor visual outcome when the age of the patient more than 60 years.

Bhuyan et al (42)in his also quoted that there is no statistical significance between the age of the patient and the final visual outcome (p=1.00) which is comparable with our study.s

**IOP AT PRESENTATION AND BCVA**

The mean IOP at presentation was 40.71±7.02. The patients with poor visual acuity of less than 6/60 had IOP at the time of admission as more than 50mmhg in 2 cases(16.67%), 41 to 50mmhg in 4 cases(33.33%), 31 to 40mmhg in 5 cases (41.67%) and 22to 30mmhg in (8.33%) 1 case.

When IOP at presentation and and BCVA at the end of one month are related, the p value is more than 0.05(p =0.54) which narrates that both are not statistically significant.

On comparing with the study done by Prajna et al (4)there is no statistical significant association between the intraocular pressure preoperatively and final visual outcome.p=0.07.

## POST OPERATIVE COMPLICATIONS

Out of 98 cases, 26 cases (26.53%) had postoperative uveitis, of which 14 cases had phacolytic glaucoma(53.84%), 10 cases had phacomorphic glaucoma(38.46%) and 2 cases had traumatic 2ACG due to subluxation of lens and 1 case of lens particle glaucoma(3.84%). They were treated with topical steroid eye drops. 6 cases(6.12%) had corneal edema and 4 cases had striate keratitis and they were treated topical 5% hypertonic saline. At the end on one month 3 cases persisted to have corneal edema and 1 case of striate keratitis was present.

Prajna et al has observed iritis in 25 cases of phacomorphic glaucoma accounting to 69%and 18 cases of phacolytic glaucoma (75%). Posterior capsular rent was in 9 cases and vitreous loss in 10 cases.

## SUMMARY

A clinical study was done with 98 cases of lens induced glaucoma presenting to the Department of Ophthalmology, Government Stanley Medical College, parameters such as clinical presentation, management and visual outcome following surgery were recorded and studied. The following observations were made.

1. In our study, age of the patients presenting with LIG ranged between 45-93 years with mean age being 63.78±8.88 years.
2. There is a female preponderance of 52.02%. The male: female ratio is 1:1.08
3. The most common type of LIG in our study is phacomorphic glaucoma which accounts to 58.16% followed by phacolytic glaucoma that accounts to 38.77%.
4. Majority of patients had immature cataract in other eye (58.16%) followed by pseudophakia (35.71%).
5. The fellow eye had good visual acuity of 6/6-6/12 in 53.06%.
6. In our study 64.29% of cases presented within seven days of onset of symptoms.
7. About 44.90% of the cases in our study had the intraocular pressure in the range of 41-50mmhg with the mean being 40.71±7.01 mmhg.
8. On comparing the IOP at presentation with final visual outcome, the p value was found to be 0.54 which is not found to be statistically significant.
9. Most patients underwent small incision cataract surgery with PCIOL implantation in the bag (88.77%).

10. The most common complication following surgery is uveitis 26.53% and is present more often in phacolytic glaucoma (53.84%) than is found in patients who had phacomorphic glaucoma(38.46%).
11. In our study good visual outcome of 6/6 to 6/12 was observed in 45.92% and poor visual outcome <6/60 was observed in 12.24% cases.
12. When the age of the patient is compared between the groups of good and poor visual outcome there is a statistical significant correlation with the p value of 0.031.
13. The best corrected visual acuity when compared with duration of symptoms the p value was found to be less than 0.01% which shows there is a strong statistical association between BCVA and duration of symptoms.

# CONCLUSION

Being a developing nation, the incidence of cataract is common in India and hence complications related to cataractous lens such as lens induced glaucoma are still commoner.

The majority of patients had good vision in the other eye so neglected the affected eye and this resulted in the development of lens induced glaucoma. The socioeconomic background and older age of people are important other factors which limited them in using the cataract surgical services early.

When the patients presented late after the development of the symptoms of lens induced glaucoma, the visual outcome is poor even after cataract surgery in some cases and hence patient education and screening for cataract (even a door to door approach by Village Health Nurse or other health worker) is a must. Health education regarding the development of cataract and the complications associated with it should be given to patient.

Surgery is the definitive treatment in cases of lens induced glaucoma and the visual outcome after surgery is good when the patient presents early.

The postoperative complications after surgery in lens induced glaucoma is more common when compared with the normal population and hence the visual acuity post surgery is affected much and hence visual prognosis is guarded.

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## PROFORMA OF CASE STUDY

Case no:

**I)** Particulars of the patient:

Name: I.P/ O.P No.
Age/Sex:
Occupation:
Address:

**II)** Chief complaints:

| | | **RE** | **LE** |
|---|---|---|---|
| 1) | Diminution of vision: | | |
| 2) | Pain, Redness, watering Photophobia | | |
| 3) | Other associated features: Headache/vomiting etc | | |
| 4) | H/O Injury: | | |
| 5) | H/O Previous surgery: | | |

**III)** History of presenting complaints:

**IV)** **Past History:**

**V)** **Family History:**

**VI)** **Personal History:**

**VII)** **General Examination**:

Pulse B.P Temperature

**VIII)** Ocular Examination:

1) Head posture

2) Facial symmetry

3) Orthophoria

4) Extra ocular movements

5) Adnexa:
Eye brows

Eye lids

Eyelashes

6) Conjunctiva:
   a) Congestion:
   b) Chemosis:
7) Cornea: Transparency
Keratic precipitates(KP's)
vascularisation
8) Anterior Chamber:
   i) Depth
   ii) Flare/Cells:
   iii) Abnormal Contents:
9) Iris:
   i) Color
   ii) Pattern
   iii) Synechiae
   iv) Neovascularisation
   v) Iridodonesis
   vi) Other details:

10) Pupil:

i) Size
ii) Shape
iii) Position
iv) Posterior synechiae
v) Reaction to light:

Direct Consensual

11) Crystalline Lens:

i) Position:
ii) Phacodonesis:
iii) Anterior lens capsule
iv) Grading of cataract (cortical/nuclear):

- Immature
- Mature
- Hypermature

12) Intraocular Pressure:

- Applanation tonometer

13) Lacrimal Apparatus:

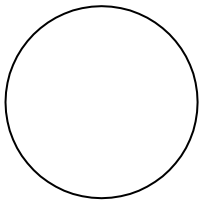
- Sac syringing test

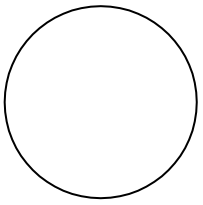
14) Visual Acuity:

- PH
- Near vision

15) Slit lamp biomicroscopy

16) Fundoscopy:





17) A-Scan:
18) B- Scan
19) Gonioscopy (If possible):

20) Diagnosis:

21) Investigations:

**I)** Urine: sugar, Albumin, Microscopy

**II)** FBS

**III)** PPBS

**IV)** CBC

**V)** RFT

**VI)** ECG

22) Management:

Pre-operative/Medical:ss

a. Ocular hypotensives:

I. Tab Diamox 250mg

II. Timolol eye drops 0.5%

III. IV Manitol 20%

b. Antibiotics:

I. Local

II. Systemic

III. NSAIDS

Surgical Management:

i. Type of anesthesia:

a. Local-

ii. Procedure:

iii. Intra-operative Complications:

iv. Condition at discharge:

v. Advice on discharge:

IX. FOLLOW-UP:

1st POD Day 3 4wks

Cornea

AC

Vision

IOP

Fundus

## சுய ஒப்புதல் படிவம்

ஆராய்ச்சி நிலையம் மருத்துவமனை. : அரசு ஸ்டான்லி மருத்துவக் கல்லூரி

பங்கு பெறுபவரின் பெயர்:

பங்கு பெறுபவரின் எண்:

பங்கு பெறுபவர் இதனை (. ) குறிக்கவும்

கண்புரையினால் ஏற்படும் கண் அழுத்த நோய் மற்றும் அதன் விளைவுகள் பற்றிய ஓர் ஆய்வு.

ஆய்வு பற்றிய விபரங்கள் எனக்கு விளக்கப்பட்டன. என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பு அளிக்கப்பட்டது.

இந்த ஆராய்ச்சியின் விபரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

மேற்கண்ட பரிசோதனையின் போது ஏற்படக்கூடிய பின்விளைவுகளையும் முழுவதும் உணர்ந்து இந்த பரிசோதனைக்கு மனமார சம்மதிக்கிறேன்.

பங்கு பெறுபவரின் கையொப்பம்

ஆய்வில் உங்கள் பங்கேற்பு மற்றும் உரிமைகள்

இந்த ஆய்வில் தங்களின் பங்கேற்பு தன்னிச்சையானது. மற்ற காரணங்கள் எதையும் கூறாமலேயே நீங்கள் இந்த ஆய்விலிருந்து எந்த ஒரு நேரத்திலும் உங்களுக்கு திருப்தி இல்லை என்று உணர்ந்தால் அல்லது வேறு ஏதேனும் உடல்நலக்குறைவு உண்டானாலோ உங்களை கவனித்து வரும் மருத்துவரிடம் உடனடியாக தெரிவிக்கவும்.
உங்களுக்கு சிகிச்சை பொருத்தமாக இருக்காது என தோன்றினால் உடனடியாக நிறுத்தப்படும்.

வேறு ஏதேனும் கேள்விகள் பிரச்சினைகள் பற்றி நீங்கள் கேட்க விரும்பினால் கீழ்கண்ட நம்பரை தொடர்பு கொள்ளவும்.

மருத்துவர் பனிமலர்.இரா
முதுநிலை மருத்துவ மாணவி
கண் இயல் துறை,
அரசு ஸ்டான்லி மருத்துவக் கல்லூரி மருத்துவமனை,
சென்னை.
தொலைபேசி எண்: 8939135405

KEY TO MASTER CHART

CORNEA

| | |
|---|---|
| CLEAR | 0 |
| EDEMA | 1 |
| STRIATE KERATITIS | 2 |

ANTERIOR CHAMBER

| | | |
|---|---|---|
| CELLS/ FLARE GRADE | 1 | 1+ |
| | 2 | 2+ |
| | 3 | 3+ |
| | 4 | 4+ |

SHALLOW-5
DEEP - 6

MEDICAL MANAGEMENT

| | |
|---|---|
| GIVEN | 1 |
| NOT GIVEN | 0 |

| S.NO | NAME | AGE | SEX | IP NO | COMPLAINTS | DURATION IN DAYS | RE/LE | VA | SIGNS | | IOP | BSCAN | DIAGNOSIS | OTHER EYE | VA IN OTHER EYE | OPERATED BEFORE | MEDICAL TREATMENT | | | | SURGERY | INTRA OP COMPLICATION | AT THE TIME OF DISCHARGE | | | | | Day 30 | | | | Complications | GROUP |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | CORNEAL EDEMA | AC | | | | | | | A-TOPICAL BRIMONIDINE | B-TOPICAL TIMOLOL | C-T-ACETAZOLAMIDE 1-0-1 | D- INJ MANNITOL | | | VA | IOP | CORNEA | AC REACTION | complications | VA | IOP | CORNEA | AC | | |
| 1 | SUGANYA | 48 | F | 173164 | REDNESS PAIN | | RE | PL+PR ACCURATE | 1 | 5 | 44 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/9 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 18 | 0 | | | 6/9 | 18 | 0 | | | 1 |
| 2 | ZAITHUN BEE | 65 | F | 1733295 | REDNESS PAIN | | LE | PL+PR ACCURATE | 1 | 2,6 | 36 | NAD | PHACOLYTIC GLAUCOMA | NS2 PSC | 6/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 16 | 1 | 3 | ANTERIOR UVEITIS | 6/24 | 16 | 0 | | | 2 |
| 3 | RANI | 60 | F | 1735046 | PAIN | 15 | RE | HM | 1 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/36 | 0 | 1 | 1 | 0 | 1 | COMBINED TRAB WITH SICS AND PCIOL | | 6/36 | 16 | 1 | | CORNEAL EDEMA | 6/24 | 16 | 0 | | | 2 |
| 4 | RAJENDRAN | 68 | M | 1735116 | PAIN | | RE | PL+PR ACCURATE | 1 | 5 | 40 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/9 | 3 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | SHALLOWING OF AC | 6/9 | 18 | 0 | | | 6/9 | 18 | 0 | | | 1 |
| 5 | SIVAJI | 60 | M | 1735635 | DEFECTIVE VISION | 7 | LE | HM | 1 | 3,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 14 | 2 | | MOSAIC SK | 6/24 | 14 | 2 | | MOSAIC SK | 2 |
| 6 | VARATHAN | 65 | M | 1738611 | PAIN | | RE | CFCF | 1 | 5 | 42 | NAD | PHACOMORPHIC GLAUCOMA | NS3 | 5/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/6 | 16 | 0 | | | 6/6 | 16 | 0 | | | 1 |
| 7 | NAGAMMAL | 60 | F | 1740463 | PAIN REDNESS | | LE | PL+PR ACCURATE | 1 | 5 | 44 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/12 | 5 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 18 | 1 | 2 | | 6/12 | 18 | 0 | | | 1 |
| 8 | DILLI | 64 | M | 1741349 | PAIN REDNESS | 10 | LE | HM | 1 | 3,6 | 52 | NAD | PHACOLYTIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 18 | 0 | | | 6/36 | 16 | 0 | | | 2 |
| 9 | SUMATHY | 62 | F | 1701912 | PAIN | | RE | PL+PR ACCURATE | 1 | 5 | 58 | NAD | PHACOMORPHIC GLAUCOMA | NS4 | 3/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | SHALLOWING OF AC | 6/60 | 14 | 0 | | | 6/60 | 14 | 0 | | | 2 |
| 10 | TAMILARASI | 50 | F | 1741967 | DEFECTIVE VISION | 15 | LE | PL+PR ACCURATE | 1 | 3,6 | 42 | NAD | PHACOLYTIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | HYPHAEMA <1/3 | 4/60 | 14 | 0 | 3 | ANTERIOR UVEITIS | 4/60 | 14 | 0 | 2 | UVEITIS | 3 |
| 11 | DEVARAJAN | 63 | M | 1741992 | PAIN REDNESS | 6 | RE | PL+PR ACCURATE | 1 | 5 | 46 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/9 | 2 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | REMNANT CORTEX | 6/60 | 20 | 0 | 2 | ANTERIOR UVEITIS | 6/60 | 16 | 0 | | | 2 |
| 12 | MUNA | 65 | F | 1751775 | PAIN REDNESS | | LE | HM | 1 | 3,6 | 38 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/36 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 12 | 0 | | | 6/12 | 12 | 0 | | | 1 |
| 13 | RANI | 60 | F | 1750188 | PAIN REDNESS | | RE | HM | 1 | 2,6 | 38 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/12 | 3 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/12 | 14 | 0 | | | 6/12 | 14 | 0 | | | 1 |
| 14 | MUMTAJ | 45 | F | 1750170 | PAIN REDNESS | 7 | LE | PL+PR ACCURATE | 1 | 5 | 36 | NAD | PHACOMORPHIC GLAUCOMA | PSC | 6/9 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/36 | 14 | 2 | | SK | 6/36 | 16 | 0 | | | 2 |
| 15 | BASKAR | 47 | M | 1752040 | PAIN REDNESS | | LE | HM | 1 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/12 | 14 | 0 | | | 6/12 | 14 | 0 | | | 1 |
| 16 | THOMAS | 83 | M | 1753353 | PAIN REDNESS | 7 | RE | PL+PR ACCURATE | 1 | 5 | 32 | NAD | PHACOMORPHIC GLAUCOMA | NS4 | 2/60 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL IN SULCUS | PCR WITH VITREOUS LOSS | 4/60 | 14 | 1 | 2 | ANTERIOR UVEITIS | 4/60 | 16 | 0 | 3 | UVEITIS WITH CYST | 3 |
| 17 | VIJAYA | 50 | F | 1753894 | PAIN REDNESS | | LE | PL+PR ACCURATE | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | SHALLOWING OF AC | 6/60 | 14 | 0 | | | 6/60 | 14 | 0 | | | 2 |
| 18 | SUSILA | 70 | F | 1755108 | PAIN | 9 | RE | PL+PR ACCURATE | 1 | 5 | 52 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/12 | 6 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/60 | 16 | 0 | 2 | ANTERIOR UVEITIS | 4/60 | 16 | 0 | | PCO | 3 |
| 19 | SIVAGAMI | 60 | F | 1756227 | PAIN | 4 | LE | HM | 1 | 5 | 46 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/18 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 18 | 0 | | | 6/36 | 18 | 0 | | | 2 |
| 20 | PRAKASH | 53 | M | 1757979 | PAIN REDNESS | | LE | HM | 1 | 2,6 | 36 | NAD | PHACOLYTIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/18 | 16 | 0 | 1 | ANTERIOR UVEITIS | 6/9 | 16 | 0 | | | 1 |
| 21 | EGAVALLI | 70 | F | 1759310 | PAIN REDNESS | 14 | LE | HM | 1 | 3,6 | 28 | NAD | PHACOLYTIC GLAUCOMA | MC | HM | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 2/60 | 14 | 0 | | | PL+ | 14 | 0 | | CORNEAL EDEMA | 3 |
| 22 | SEETHA | 93 | F | 1767456 | PAIN | 7 | RE | PL+PR ACCURATE | 1 | 5 | 32 | NAD | PHACOMORPHIC GLAUCOMA | APHAKIA | 6/60 | 15 | 1 | 1 | 0 | 1 | SICS WITH LENS EXTRACTION | SHALLOWING OF AC | 4/60 | 16 | 0 | 2 | ANTERIOR UVEITIS | 4/60 | 16 | 0 | 3 | UVEITIS | 3 |
| 23 | LEKSHMI | 54 | F | 1768253 | PAIN REDNESS | | LE | HM | 0 | 1,6 | 28 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/6 | 1 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/12 | 14 | 0 | | | 6/12 | 14 | 0 | | | 1 |
| 24 | SYED | 50 | M | 1768317 | PAIN REDNESS | | RE | PL+PR ACCURATE | 1 | 5 | 46 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/6 | 0 | 1 | 0 | 1 | 1 | SICS WITH PCIOL | | 6/9 | 16 | 0 | | | 6/9 | 16 | 0 | | | 1 |
| 25 | MOHAMMED ALI | 59 | M | 1771726 | PAIN,REDNESS | 8 | LE | PL+PR ACCURATE | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 3/60 | 26 | 1 | 3 | UVEITIS CORNEAL EDEN | 3/60 | 18 | 1 | | CORNEAL EDEMA | 3 |
| 26 | MUNIAMMAL | 60 | F | 1771951 | PAIN REDNESS | 10 | RE | HM | 1 | 5 | 52 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/6 | 1 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 16 | 0 | | | 6/36 | 16 | 0 | | | 2 |
| 27 | GOWSE BEE | 70 | F | 1802036 | PAIN REDNESS | 12 | LE | HM | 1 | 3,6 | 50 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/18 | 5 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/12 | 16 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 28 | SELVAM | 58 | M | 1805446 | PAIN | | LE | HM | 0 | 3,6 | 28 | NAD | LENS PARTICLE GLAUCOMA | PCIOL | 6/12 | 2 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/18 | 14 | 0 | 2 | ANTERIOR UVEITIS | 6/18 | 16 | 0 | | | 1 |
| 29 | YENGAMMAL | 70 | F | 1804855 | PAIN | 12 | LE | PL+PR ACCURATE | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/36 | 12 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/12 | 12 | 0 | | | 6/12 | 14 | 0 | | | 1 |
| 30 | MANORANJITHAM | 65 | F | 1805724 | PAIN REDNESS | | RE | HM | 1 | 3,6 | 46 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/18 | 3 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/24 | 16 | 2 | | SK | 6/24 | 16 | 0 | | | 2 |
| 31 | VELU | 55 | M | 1808810 | REDNESS | | RE | PL+PR ACCURATE | 1 | 5 | 42 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/60 | 0 | 0 | 1 | 1 | 0 | SICS WITH PCIOL | SHALLOWING OF AC | 6/12 | 18 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 32 | NOZAN BEE | 70 | F | 1808801 | PAIN | 13 | RE | PL+PR ACCURATE | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/12 | 5 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/9 | 14 | 0 | | | 6/9 | 18 | 0 | | | 1 |
| 33 | YESBU | 70 | M | 1807962 | REDNESS | 10 | RE | HM | 1 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | NS4 | 2/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 28 | 0 | | | 6/12 | 20 | 0 | | | 1 |
| 34 | MUNIYAMMAL | 60 | F | 1801874 | PAIN | 10 | LE | HM | 1 | 3,6 | 40 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/12 | 1 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/24 | 14 | 0 | 3 | ANTERIOR UVEITIS | 6/24 | 16 | 0 | | | 2 |
| 35 | GLEN THOMAS | 64 | M | 1812730 | PAIN | | LE | PL+PR ACCURATE | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | NS3 | 5/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 16 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 36 | THARMARAJ | 57 | M | 1811372 | PAIN,REDNESS | | LE | 1/60 | 1 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/36 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/6 | 14 | 0 | | | 6/6 | 16 | 0 | | | 1 |
| 37 | ALIMA BEE | 65 | F | 1818920 | PAIN | 12 | LE | HM | 1 | 5 | 42 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/36 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/9 | 16 | 0 | | | 6/9 | 14 | 0 | | | 1 |
| 38 | ANBALAGAN | 67 | M | 1824031 | PAIN | | LE | PL+PR ACCURATE | 1 | 3,6 | 50 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/12 | 2 | 1 | 1 | 1 | 1 | SICS WITH IRIS CLAW LENS | ZONULAR DIALYSIS | 6/60 | 18 | 0 | 2 | ANTERIOR UVEITIS | 6/60 | 16 | 0 | | | 2 |
| 39 | JEYAPAUL | 70 | M | 1829822 | PAIN REDNESS | | RE | HM | 1 | 3,6 | 46 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/24 | 5 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/60 | 16 | 1 | | CORNEAL EDEMA | 6/36 | 18 | 0 | | | 2 |
| 40 | ARELMAN | 74 | M | 1835838 | PAIN REDNESS | 13 | LE | PL+PR ACCURATE | 1 | 3,6 | 48 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/60 | 5 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | PL+ | 14 | 0 | | | PL+ | 16 | 0 | | DISC PALLOR | 3 |
| 41 | DURAI | 65 | M | 1838529 | PAIN REDNESS | | RE | HM | 1 | 2,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/36 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/24 | 18 | 0 | 2 | ANTERIOR UVEITIS | 6/24 | 18 | 0 | | | 2 |
| 42 | MANI | 70 | M | 1916157 | PAIN | 8 | LE | HM | 1 | 5 | 44 | NAD | PHACOMORPHIC GLAUCOMA | NS3 | 6/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/24 | 14 | 2 | | SK | 6/12 | 14 | 0 | | | 1 |
| 43 | MANORAMA | 58 | F | 1866193 | PAIN REDNESS | | LE | CFCF | 1 | 3,6 | 48 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/12 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/24 | 26 | 1 | 3 | ANTERIOR UVEITIS | 6/12 | 16 | 0 | | | 1 |
| 0 | ABINESH | 56 | M | 1882561 | PAIN REDNESS | | LE | HM | 1 | 5 | 56 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/6 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 18 | 0 | | | 6/36 | 18 | 0 | | | 2 |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 45 | VASANTHA | 55 | F | 1844361 | PAIN | | LE | PL+PR ACCURATE | 0 | 5 | 36 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/6 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/12 | 16 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 46 | PUSHPA | 65 | F | 1842856 | PAIN,REDNESS | 14 | LE | HM | 0 | 5 | 32 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/9 | 6 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/60 | 14 | 0 | 3 | ANTERIOR UVEITIS | 6/60 | 14 | 0 | | | 2 |
| 47 | KUPPU | 68 | F | 1884764 | PAIN REDNESS | | RE | PL+PR ACCURATE | 1 | 3,6 | 42 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/12 | 2 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/18 | 12 | 0 | 3 | ANTERIOR UVEITIS | 6/18 | 14 | 0 | | | 1 |
| 48 | CHELLAMA | 60 | F | 1888382 | PAIN | | LE | 1/60 | 0 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/60 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/60 | 18 | 0 | | | 6/60 | 18 | 0 | | | 2 |
| 49 | JANAKIRAMAN | 60 | M | 1900157 | PAIN,REDNESS | | LE | CFCF | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/12 | 1 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/18 | 16 | 0 | | | 6/18 | 16 | 0 | | | 1 |
| 50 | CHELLAPAN | 60 | M | 1904265 | PAIN REDNESS | 7 | RE | PL+PR ACCURATE | 1 | 3 | 36 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/9 | 2 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/9 | 18 | 0 | | | 6/9 | 18 | 0 | | | 1 |
| 51 | LALITHA | 55 | F | 1911667 | PAIN | | LE | HM | 1 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/9 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | SHALLOWING OF AC | 6/18 | 16 | 0 | | | 6/18 | 16 | 0 | | | 1 |
| 52 | FATHIMA | 52 | F | 1969561 | PAIN REDNESS | | RE | HM | 1 | 3,6 | 52 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/18 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 5/60 | 14 | 0 | | | 5/60 | 16 | 0 | 3 | UVEITIS | 3 |
| 53 | SHANKARI | 50 | F | 1983236 | PAIN REDNESS | | LE | PL+PR ACCURATE | 1 | 2,6 | 36 | NAD | PHACOLYTIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/36 | 24 | 0 | 3 | ANTERIOR UVEITIS | 6/36 | 14 | 0 | | | 2 |
| 54 | RAJENDRAN | 55 | M | 1950911 | PAIN | | RE | 1/60 | 0 | 2,6 | 32 | NAD | TRAUMATIC SUBLUXATION OF LENS AND ZACG | CLEAR | 6/6 | 0 | 1 | 1 | 1 | 0 | SICS WITH LENS EXTRACTION ,AV WITH SFIOL | ZONULAR DIALYSIS VITREOUS IN AC | 4/60 | 20 | 1 | 3 | UVEITIS CORNEAL EDEN | 4/60 | 16 | 1 | | CORNEAL EDEMA | 3 |
| 55 | SIVAJI | 65 | M | 1950264 | PAIN | | LE | CFCF | 1 | 5 | 44 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/6 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 14 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 56 | SENDAMARAI | 68 | F | 1951765 | PAIN | 10 | RE | HM | 0 | 4 | 42 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/9 | 3 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/18 | 16 | 0 | | | 6/18 | 14 | 0 | | | 1 |
| 57 | MUNUSAMY | 58 | M | 1953981 | PAIN | | LE | PL+PR ACCURATE | 0 | 5 | 32 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/9 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/9 | 18 | 0 | | | 6/9 | 16 | 0 | | | 1 |
| 58 | XAVIER | 52 | M | 1965234 | PAIN | | LE | HM | 0 | 5 | 36 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/9 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/18 | 20 | 0 | | | 6/9 | 14 | 0 | | | 1 |
| 59 | SARVESH | 69 | M | 1965987 | PAIN | | RE | PL+PR ACCURATE | 1 | 5 | 44 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/18 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 18 | 0 | | | 6/36 | 16 | 0 | | | 2 |
| 60 | RAASATHI | 75 | F | 1923768 | PAIN | | RE | HM | 1 | 2,6 | 42 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/60 | 7 | 1 | 1 | 1 | 1 | SICS WITH PCIOL IN SULCUS | PCR WITH VITREOUS LOSS | 6/60 | 16 | 0 | 3 | ANTERIOR UVEITIS | 6/24 | 14 | 0 | | | 2 |
| 61 | MARIMUTHU | 62 | M | 1943265 | PAIN | | RE | HM | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/6 | 2 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 18 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 62 | CHEZIAN | 58 | M | 1987634 | PAIN,REDNESS | | RE | PL+PR ACCURATE | 0 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/9 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/18 | 12 | 0 | | | 6/12 | 14 | 0 | | | 1 |
| 63 | ELLAMMAL | 69 | F | 1898787 | PAIN | | LE | HM | 0 | 1,6 | 28 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/18 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/12 | 14 | 0 | | | 6/12 | 12 | 0 | | | 1 |
| 64 | RAMACHANDRAN | 64 | M | 1897653 | PAIN REDNESS | 8 | LE | PL+PR ACCURATE | 0 | 1,6 | 30 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/18 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/36 | 18 | 0 | | | 6/24 | 16 | 0 | | | 2 |
| 65 | RAMANINATHAN | 72 | M | 1812354 | PAIN REDNESS | | RE | PL+PR ACCURATE | 1 | 5 | 50 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/36 | 5 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | SHALLOWING OF AC | 6/9 | 16 | 0 | | | 6/9 | 16 | 0 | | | 1 |
| 66 | SIVA | 66 | M | 1821342 | PAIN REDNESS | | RE | PL+PR ACCURATE | 1 | 3,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/6 | 3 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 18 | 1 | 2 | UVEITIS CORNEAL EDEN | 6/12 | 18 | 0 | | | 1 |
| 67 | RASI | 67 | F | 1821657 | PAIN | 7 | RE | HM | 0 | 5 | 42 | NAD | PHACOMORPHIC GLAUCOMA | NS3 PSC | 4/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/18 | 16 | 0 | | | 6/18 | 16 | 0 | | | 1 |
| 68 | PARIMALARANI | 66 | F | 1832674 | PAIN | 7 | LE | HM | 0 | 2,6 | 38 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/9 | 2 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/24 | 16 | 0 | | | 6/24 | 16 | 0 | | | 2 |
| 69 | RAJATHILAGAM | 68 | F | 1856421 | PAIN | | LE | PL+PR ACCURATE | 0 | 5 | 36 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/60 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | DM STRIPPING | 6/36 | 18 | 0 | | | 6/36 | 14 | 0 | | | 2 |
| 70 | SOORIYANATHAN | 62 | M | 1897656 | PAIN | | LE | PL+PR ACCURATE | 1 | 3,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/6 | 3 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/36 | 18 | 0 | 3 | ANTERIOR UVEITIS | 6/12 | 16 | 0 | | OPTIC CAPTURE OF | 1 |
| 71 | KUMARI | 57 | F | 1909123 | PAIN REDNESS | 8 | RE | HM | 1 | 2,6 | 46 | NAD | PHACOLYTIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/60 | 16 | 0 | 3 | ANTERIOR UVEITIS | 6/60 | 16 | 0 | | | 2 |
| 72 | KUPPAIAH | 64 | M | 1902341 | PAIN REDNESS | | RE | HM | 1 | 5 | 40 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/24 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | SHALLOWING OF AC | 6/9 | 14 | 0 | | | 6/9 | 14 | 0 | | | 1 |
| 73 | KOLANJI | 48 | F | 1908767 | PAIN REDNESS | | LE | PL+PR ACCURATE | 1 | 3,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/18 | 16 | 0 | | | 6/18 | 16 | 0 | | | 1 |
| 74 | LOSAYA | 76 | F | 1876384 | PAIN | | LE | HM | 1 | 5 | 42 | NAD | PHACOMORPHIC GLAUCOMA | NS3 | 3/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 14 | 0 | | | 6/12 | 14 | 0 | | | 1 |
| 75 | MICHAEL | 56 | M | 1790822 | PAIN | | LE | PL+PR ACCURATE | 0 | 5 | 32 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/9 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/9 | 16 | 0 | | | 6/9 | 16 | 0 | | | 1 |
| 76 | POTHUM PONNU | 69 | F | 1782190 | PAIN | | RE | PL+PR ACCURATE | 0 | 5 | 36 | NAD | PHACOMORPHIC GLAUCOMA | NS3 PSC | 6/60 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/9 | 20 | 0 | | | 6/6 | 14 | 0 | | | 1 |
| 77 | NATHAN | 65 | M | 1897234 | PAIN REDNESS | | RE | HM | 0 | 3,6 | 26 | NAD | PHACOLYTIC GLAUCOMA | NS2 | 6/24 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/24 | 18 | 1 | | CORNEAL EDEMA | 6/24 | 16 | 1 | | | 2 |
| 78 | PAVUNNU | 58 | F | 1937345 | PAIN | | RE | HM | 0 | 5 | 28 | NAD | PHACOMORPHIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/6 | 16 | 0 | | | 6/6 | 16 | 0 | | | 1 |
| 79 | KARUPPU | 48 | M | 1890283 | PAIN REDNESS | | RE | CFCF | 0 | 5 | 32 | NAD | PHACOMORPHIC GLAUCOMA | CLEAR | 6/6 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 16 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 80 | SOMASUNDARI | 76 | F | 1892789 | DEFECTIVE VISION | | RE | PL+PR ACCURATE | 0 | 2,6 | 32 | NAD | PHACOLYTIC GLAUCOMA | NS3 | 5/60 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/18 | 14 | 0 | 3 | ANTERIOR UVEITIS | 6/18 | 14 | 0 | | | 1 |
| 81 | VANITHA | 67 | F | 1812908 | PAIN | | LE | PL+PR ACCURATE | 1 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/6 | 2 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/36 | 16 | 0 | | | 6/36 | 16 | 0 | | | 2 |
| 82 | REVATHY | 62 | F | 1834234 | PAIN | | RE | HM | 1 | 5 | 36 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/12 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/60 | 12 | 0 | 2 | ANTERIOR UVEITIS | 6/12 | 12 | 0 | | | 1 |
| 83 | RISHWANA | 64 | F | 1876198 | PAIN REDNESS | | LE | PL+PR ACCURATE | 0 | 2,6 | 32 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/12 | 3 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/12 | 14 | 0 | | | 6/12 | 14 | 0 | | | 1 |
| 84 | BOBY | 58 | F | 1890345 | PAIN REDNESS | 10 | RE | PL+PR ACCURATE | 1 | 2,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/6 | 2 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/18 | 16 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 85 | SOOSAIYA | 69 | M | 1908982 | PAIN REDNESS | | LE | HM | 1 | 5 | 46 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/24 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/18 | 18 | 0 | | | 6/9 | 18 | 0 | | | 1 |
| 86 | JAMMAKA | 78 | F | 1876309 | DEFECTIVE VISION | 20 | LE | PL+PR ACCURATE | 0 | 3,6 | 34 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/12 | 5 | 1 | 1 | 1 | 0 | COMBINED TRAB WITH SICS AND PCIOL | | 6/36 | 20 | 0 | | | 6/36 | 14 | 0 | | | 2 |
| 87 | FAKRUDIN | 63 | M | 1809212 | PAIN AFTER TRAUMA | | LE | CFCF | 0 | 2,6 | 30 | NAD | TRAUMATIC SUBLUXATION OF LENS AND ZACG | NS1 | 6/12 | 0 | 1 | 1 | 1 | 1 | SICS WITH LENS EXTRACTION ,AV WITH SFIOL | ZONULAR DIALYSIS VITREOUS IN AC | 6/60 | 18 | 0 | 2 | ANTERIOR UVEITIS | 6/60 | 14 | 0 | | OPTIC CAPTURE OF | 2 |
| 88 | ALEN THOMAS | 67 | M | 1813563 | PAIN | 7 | LE | PL+PR ACCURATE | 0 | 5 | 32 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/24 | 0 | 1 | 1 | 1 | 1 | SICS WITH ACIOL | PCR WITH VITREOUS LOSS | 2/60 | 18 | 0 | 3 | ANTERIOR UVEITIS | 2/60 | 18 | 0 | | | 3 |
| 89 | ANNAPOTTU | 82 | F | 1789234 | PAIN | 15 | RE | PL+PR ACCURATE | 1 | 5 | 40 | NAD | PHACOMORPHIC GLAUCOMA | MC | HM | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 4/60 | 20 | 1 | | CORNEAL EDEMA | 6/60 | 16 | 0 | | | 2 |
| 90 | RAHIM | 75 | M | 1983453 | PAIN | 10 | RE | HM | 0 | 5 | 38 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/12 | 3 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/60 | 18 | 0 | | | 6/36 | 18 | 0 | | | 2 |
| 91 | MUTHUSELVAN | 62 | M | 1976342 | PAIN | | RE | HM | 0 | 5 | 42 | NAD | PHACOMORPHIC GLAUCOMA | NS2 | 6/18 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | SHALLOWING OF AC | 6/36 | 12 | 0 | 2 | | 6/36 | 12 | 0 | | | 2 |
| 92 | RAAMAYI | 72 | F | 1718762 | PAIN REDNESS | | RE | PL+PR ACCURATE | 1 | 2,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/12 | 5 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 6/24 | 14 | 0 | 3 | ANTERIOR UVEITIS | 6/12 | 14 | 0 | | | 1 |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 93 | MUTHAZHAGI | 62 | F | 1733289 | PAIN REDNESS | 4 | LE | HM | 1 | 4,6 | 44 | NAD | PHACOLYTIC GLAUCOMA | NS1 | 6/12 | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | 6/12 | 16 | 0 | | | 6/9 | 16 | 0 | | | 1 |
| 94 | BACKIYALAKSHMI | 85 | F | 1789256 | PAIN REDNESS | 12 | LE | PL+PR ACCURATE | 1 | 5 | 52 | NAD | PHACOMORPHIC GLAUCOMA | PCIOL | 6/24 | 10 | 1 | 1 | 1 | 1 | SICS WITH ACIOL | PCR WITH VITREOUS LOSS | 5/60 | 26 | 0 | 3 | ANTERIOR UVEITIS | 6/60 | 18 | 0 | | | 2 |
| 95 | SIVANESAN | 72 | M | 1809823 | PAIN REDNESS | 8 | RE | PL+PR ACCURATE | 1 | 2,6 | 46 | NAD | PHACOLYTIC GLAUCOMA | PCIOL | 6/18 | 5 | 1 | 1 | 1 | 1 | SICS WITH PCIOL IN SULCUS | PCR WITH VITREOUS LOSS | 6/12 | 12 | 0 | | | 6/12 | 16 | 0 | | | 1 |
| 96 | LENIN BRITO | 82 | M | 1908732 | PAIN REDNESS | 20 | LE | PL+PR ACCURATE | 0 | 5 | 36 | NAD | PHACOMORPHIC GLAUCOMA | NS3 | 3/60 | 0 | 1 | 1 | 0 | 1 | SICS WITH PCIOL | | 3/60 | 14 | 0 | | | 2/60 | 14 | 0 | 2 | UVEITIS | 3 |
| 97 | JAYACHITRA | 77 | M | 1950234 | PAIN | 8 | RE | PL+PR ACCURATE | 0 | 5 | 34 | NAD | PHACOMORPHIC GLAUCOMA | NS3 PSC | 5/60 | 0 | 1 | 1 | 1 | 0 | SICS WITH PCIOL | | 6/12 | 16 | 0 | | | 6/6 | 18 | 0 | | | 1 |
| 98 | PARTHIBAN | 67 | M | 1767872 | PAIN | 20 | LE | PL NEG | 1 | 5 | 48 | NAD | PHACOMORPHIC GLAUCOMA | PTHISICAL | PL NEG | 0 | 1 | 1 | 1 | 1 | SICS WITH PCIOL | | PL + | 18 | 0 | | | PL + | 16 | 0 | | DISC PALLOR | 3 |