A Prospective Observational Study to Assess the Post-Surgical Outcomes following Cataract Surgery in Patients with Lens Induced Glaucoma

Dissertation submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the requirements for the degree of MS Ophthalmology

BRANCH – III

OPHTHALMOLOGY

REG NO: 221713456

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CERTIFICATE

This is to certify that this dissertation entitled **"A Prospective Observational study to assess the post- surgical outcomes following Cataract Surgery in patients with Lens Induced Glaucoma"** submitted to the Tamil Nadu Dr MGR Medical University, is a bonafide work done by Dr Praveen Kumar P, M.B.B.S., under our guidance and supervision in the Glaucoma department of Aravind Eye Hospital (AEH) and Post – Graduate Institute of Ophthalmology, Madurai during his residency programme from May 2017 – May 2020.

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DECLARATION

I, Dr. Praveen Kumar solemnly declare that the dissertation titled "A prospective observational study to assess the post-surgical outcomes following cataract surgery in patients with Lens Induced Glaucoma" has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other individual for any reward, degree, diploma to any other University Board either in India or abroad. This dissertation is submitted to the **Tamil Nadu Dr. M.G.R. Medical University,** Chennai in partial fulfilment of the rules and regulation for the award of **M.S Ophthalmology (BRANCH III)** to be held in May 2020.

Place: Madurai

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CONTENTS

PART A

BACKGROUND

Glaucoma is the leading cause of irreversible blindness worldwide with an estimated 60.5 million people affected in 2010 and 12 million people are estimated to be blind due to this disease [1]. India is home to 12 million people with glaucoma and 1.5 million are blind due to it, as per the major prevalence studies in India in the recent past [2-7.]

Glaucoma by definition is "a group of disorders of multifactorial aetiology characterised by optic neuropathy with potentially progressive clinically visible changes at the optic nerve head, comprising focal or generalized thinning of the neuro retinal rim with excavation and enlargement of the optic cup, representing neurodegeneration of retinal ganglion cell axons and deformation of the lamina cribrosa, with corresponding diffuse and localized nerve fibre bundle pattern visual field loss" [8.]

Lens Induced Glaucoma (LIG) is said to be a distinct pathological entity, which is clinically recognizable, they are easily preventable and curable by lens extraction [9]. Cataract has been documented to be the most significant cause of bilateral blindness in India and as well as on a global scale [10-13]. It is found that in India, the most common cause of preventable blindness is caused by senile cataract which accounts to 63.7% [14].

In developing countries like India, lens induced glaucoma is a common entity due to lack of awareness of cataract and delayed surgical intervention. Though

the surgical services are reasonably available to some extent in our country, despite which there are various social, economic barriers which prevents people from accessing eye health care services. Despite easy and improved availability of eye care and surgical facilities, the prognosis of visual recovery postoperatively remains guarded, until and unless they are diagnosed early and managed efficiently [15].

Worldwide, Glaucoma is considered to be the second leading cause of blindness [43]. LIG is a the common cause of ocular morbidities in countries like India where the uptake of eye care services by the people in rural community has been sub optimal [16]. Lens extraction seems to be definite management for these types of glaucomas. The preferred technique of surgery is Small incision cataract surgery with PC IOL[17]. Phaco emulsification in selected cases can be considered.

Despite the presence of several previous publications on LIG, this study has been undertaken due to the following factors:

- Though rare in developed countries; LIG is still prevalent in developing countries due to large backlog of cataract, poor health condition, poor socio-economic status, fear of operation.
- To understand the prevalent factors still existing today in developing countries leading to LIG despite the increased availability of surgical facilities, recent advances in cataract surgery with short recovery time

along with the concerted efforts of various national blindness control programmes, increased number of outreach camps and improved transportation.

- Our study aim is to analyse the common presentation of LIG's, factors affecting post-operative outcomes like Visual Acuity (VA), Intra Ocular Pressure (IOP), and thereby increase awareness about this preventable and curable condition and in reducing ocular morbidity due to this condition.

LENS INDUCED GLAUCOMA

In 1900 **Gifford** and **Von Ruess** independently described Lens induced glaucoma [18, 19]. Gifford described it as Hypermature cataract associated with Glaucoma, while Von Ruess described it as glaucoma which is associated with the spontaneous absorption of lens substance through the intact lens capsule [18].

Based on the mechanism of lens and its particles causing glaucoma, LIG is categorised into two major categories.

- Secondary angle closure glaucoma
- Secondary open angle glaucoma

SECONDARY ANGLE CLOSURE GLAUCOMA

Increased Intraocular Pressure (IOP) is caused due to blockage of anterior flow of aqueous humor by the crystalline lens.

PHACOMORPHIC GLAUCOMA

Condition(s) related to the size of the lens.

- Intumescent cataract
- Traumatic cataract

Mechanism

- Pupillary block
- Direct angle closure

- Combination

PHACOTOPIC GLAUCOMA

Condition related to the site of the lens

- Subluxated
- Dislocated

Mechanism

- Direct angle closure
- Pupillary block

SECONDARY OPEN ANGLE GLAUCOMA

The secondary open angle glaucoma is characterized by [44]

- Blockage of the trabecular meshwork from high molecular weight lens proteins (Phacolytic Glaucoma)
- Lens material or debris.
- Rarely by phacoanaphylactic response to lens material.

PHACOLYTIC GLAUCOMA (LENS PROTEIN GLAUCOMA)

Condition(s) related to soluble lens proteins.

Mechanism

- Heavy Molecular Weight protein [HMW]
- Macrophagic response to the lens Protein.

LENS PARTICLE GLAUCOMA

Condition related to lens particles

Mechanism

- Blockage of Trabecular Meshwork by lens particles e.g. - retained lens matter following cataract surgery
  - Post. YAG capsulotomy

EMBRYOLOGY OF LENS

Lens develops from surface ectoderm at 4-4.5mm stage, in front of optic vesicle as thickening to form the lens plate. Later lens plate invaginates to form lens vesicle and detaches from surface ectoderm [21]. At the stage of 9-10 mm size, initially the most central cells of the posterior wall of lens vesicle followed by the peripheral cells of the posterior wall of the lens vesicle form primary lens fibers and later the equatorial cells rapidly multiply to form the secondary lens fibers. These secondary lens fibers are laid down layer by layer encircling the centre nucleus of primary lens fibres. Lens capsule is formed by basement membrane of surface ectoderm at about 8mm size.

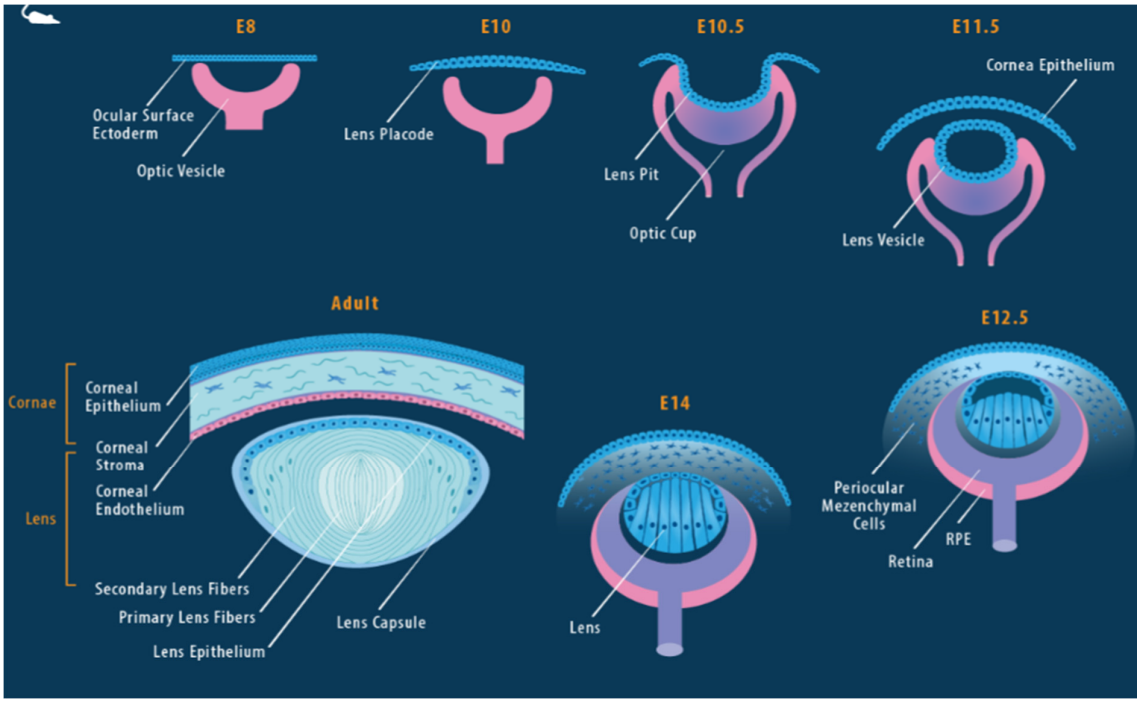

Figure -1 Developement of lens

ANATOMY OF LENS

The lens is a transparent, biconvex, avascular structure placed in patellar fossa behind iris and in front of vitreous. The lens is suspended from ciliary body by zonules. Lens continuously increases in size, thickness and colour, depending on cataract changes and nuclear sclerosis. The antero-posterior diameter or thickness increases from 3.5mm at birth to 5mm at about 80- 90 years [21, 22]. The equatorial diameter also changes 6mm to 9mm at old age. The shape of lens is spherical at birth and starts flattening during childhood and acquires biconvex shape with radius of curvature for the anterior surface of about 10-11mm and for the posterior surface of about 6mm.

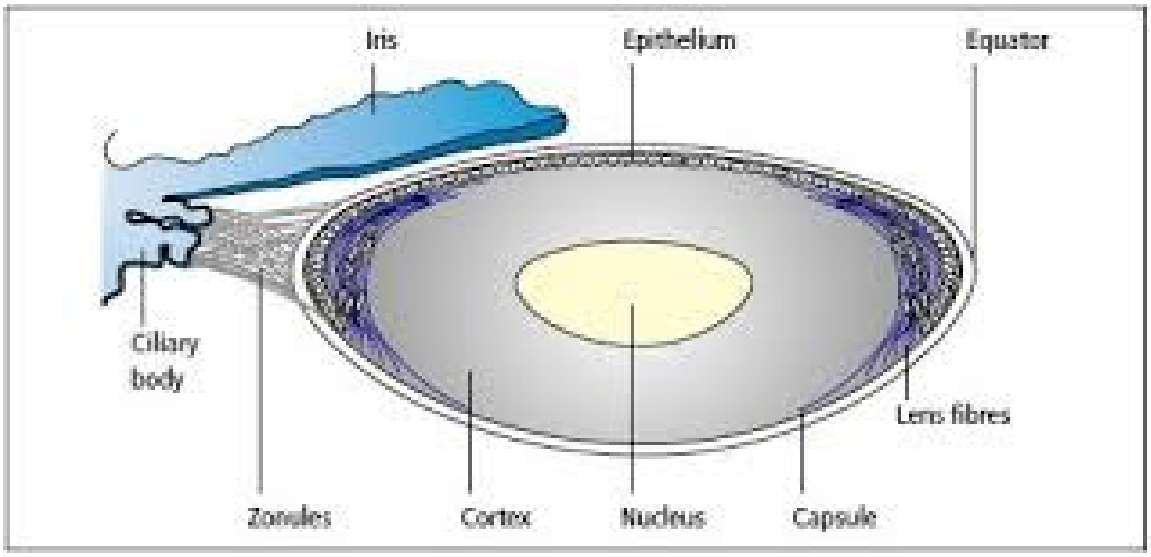

Figure -2 Anatomy of lens

Aqueous secreted by ciliary body first enters the posterior chamber, and then enters the anterior chamber through pupil. Aqueous drains from anterior chamber to episcleral veins through trabecular meshwork at the angle of anterior chamber. Any block in the drainage pathway results in accumulation of aqueous producing raised IOP. Rate of formation of aqueous is 2µl/min

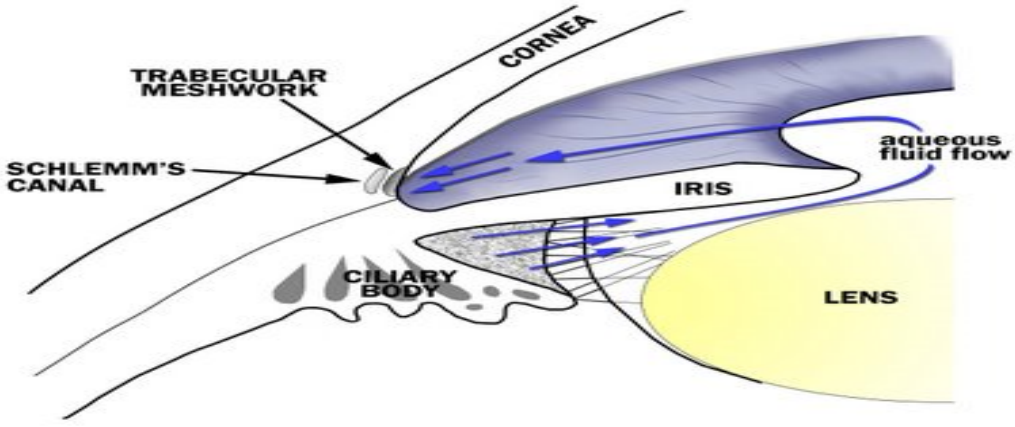

Figure-3 Aqueous drainage

PHYSIOLOGY OF LENS

Throughout the life, the lens epithelial cells at the equator divide which further develops into lens fibers, resulting in continual growth of the lens [23].The lens cells that are present with the highest metabolic rate are found to be in the epithelium and the outer cortex. These cells are superficial; it utilizes the oxygen and glucose for the active transport of carbohydrates, electrolytes and amino acids into the lens. The older cells, which are found at the center of the lens, must be able to communicate with the superficial cells and also with the environment outside the lens. This communication is accomplished with the help of low-resistance gap junctions that facilitate in the exchange of small molecules from cell to cell. Lens fiber cells have abundant amount water channels in their membranes which are made from Major Intrinsic Protein (MIP). It is not yet certain whether or if MIP serves primarily as a water channel, or as an adhesion molecule that helps in minimizing the extracellular space between fiber cells, or as both in the lens. Minimizing the extracellular space between the fiber cells is very important to reduce the scattering of light because it passes through the lens [23].

Maintenance of Lens Water and Cation Balance

The normal human lens contains approximately 66% water and 33% protein, and this proportion changes very little with aging. Lens nucleus is less hydrated than the lens cortex. The water found between the lens fibers in the

extracellular spaces accounts to 5% of the lens volume [45]. Within the lens, sodium and potassium concentrations are maintained at 20 μL and 120 μL, respectively. The most important aspect of lens physiology is the mechanism that controls water and electrolyte balance, which is critical to lens transparency. Because transparency is highly dependent on the structural and macromolecular components of the lens, perturbation of cellular hydration can readily lead to opacification. It is noteworthy that disruption of water and electrolyte balance is not a feature of nuclear cataracts. In cortical cataracts, however, the water content rises significantly.

Lens epithelium: Site of active transport

The lens is less hydrated and has higher levels of potassium ions (K+) and amino acids than the surrounding aqueous and vitreous. Conversely, the lens contains lower levels of sodium ions (Na+), chloride ions (Cl–), and water than the surrounding environment. The cation balance between the inside and outside of the lens is the result both of the permeability properties of the lens cell membranes and of the activity of the sodium-potassium pumps, which reside within the cell membranes of the lens epithelium and each lens fiber. The sodium-potassium pumps function by pumping sodium ions out while taking potassium ions in. This mechanism depends on the breakdown of ATP and is regulated by the enzyme Na+, K+-ATPase. Inhibition of Na+, K+-ATPase leads to loss of cation balance and elevated water content in the lens.

Pump–leak theory

The combination of active transport and the membrane permeability is referred to the pump–leak system of the lens. According to this theory, potassium and other molecules like amino acids are actively transported into the lens through the epithelium anteriorly [46]. When there are no active transport mechanisms, they diffuse out with the concentration gradient via the back of the lens. Conversely, the sodium flows inside through the back of the lens with the concentration gradient. Then sodium is actively exchanged for potassium by the epithelium. In support to this theory, an anteroposterior gradient was found for both ions.

- Potassium was concentrated in the anterior lens [46]
- Sodium was concentrated in the posterior lens [46].

Most of the Na+,K+-ATPase activity is found in the lens epithelium and the superficial cortical fiber cells. The active-transport mechanisms are lost if the attached epithelium and the capsule are removed from the lens. The mechanism is not lost if the capsule alone is removed by enzymatic degradation with the help of collagenase [47]. Thus we can say that epithelium is the most primary site for active transport in the lens with the help of these findings [47].

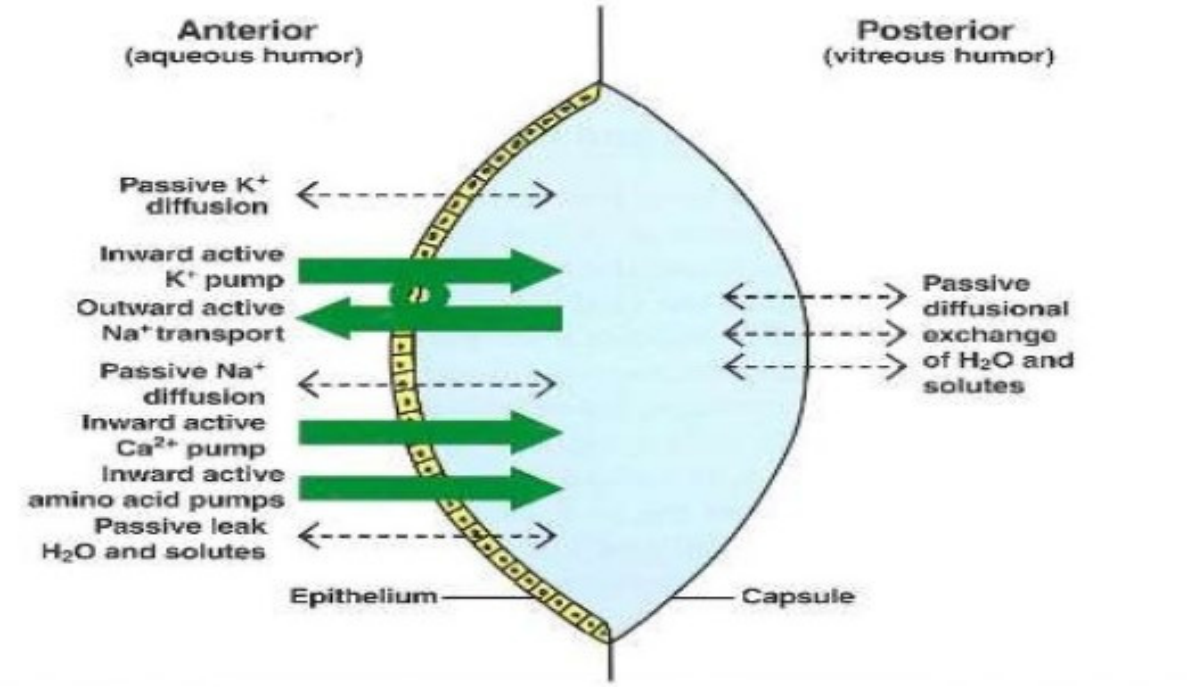

Figure 4: Pump leak hypothesis mechanism

Ageing changes in the lens

As the age progresses, the lens increases in weight and thickness and the nucleus becomes hard (nuclear sclerosis). The soluble proteins of lens fibers aggregate into higher molecular weight proteins. The chemical modification of nuclear lens proteins produces yellow or brown pigmentation. In ageing lens, the chemical concentrations of glutathione and potassium will be decreased whereas the concentrations of sodium and calcium will be increased.

SENILE CORTICAL CATARACT

Age related cataract is the most common variety, occurring bilaterally. Males and females are equally affected.

Pathogenesis

Physiochemical process mainly hydration and replacement of soluble by insoluble proteins will get altered. Histologically cortical cataracts present with hydropic swelling of lens fibers and accumulation of eosinophilic materials between them.

The clinical course of development of senile cataract can be divided into five stages:

I. Stage of lamellar separation
II. Incipient stage
III. Intumescent stage
IV. Mature cataract
V. Hypermature cataract
    a. Morgagnian type
    b. Sclerotic type.

I. **Stage of lamellar separation**

a. Collection of fluid between the lens fibers resulting in lamellar separation. Generally there are no symptoms except the patient becomes slightly hyperopic.

II. **Incipient stage**

a. Presents with white radial or wedge shaped spokes of opacities in the periphery of lens; such opacities are known as cuneiform cataract. The patient has a defective vision especially in the night due to dilatation of pupil.

III. **Intumescent stage**

a. Diffuse and irregular lenticular opacities due to hydration of deep cortical layers. The progressive hydration causes swelling and opacification of lens which is known as Intumescent cataract. Such lens pushes the iris forward in already existing shallow anterior chamber cause angle closure glaucoma.

IV. **Mature cataract**

a. Opacification of entire cortex and the lens becomes totally opaque. Visual acuity is grossly reduced to hand movements or light perception.

V. **Hypermature cataract:**

It is usually of 2 types

a) **Morgagnian cataract:**

The soft cortex liquifies and the hard nucleus sinks to the bottom. The cortex appears milky and the nucleus appears as a brown mass. The nucleus changes its position with the head movement.

b) Sclerotic type:

Sometimes the loss of fluid from the mature cataractous lens continues and the lens becomes very inspissated and shrunken. The lens appears yellow due to the deposition of cholesterol crystals while dense capsular cataract is formed at the anterior pole. The shrinkage of the lens leads to the deepening of anterior chamber and tremulousness of iris. The associated degeneration of the zonules may lead to dislocation of the lens.

TYPES OF LENS INDUCED GLAUCOMA

Phacomorphic Glaucoma

Von Graefe (1869) was the first person to recognize early secondary rise in tension that is frequently associated with a rapid swelling of the lens (intumescence) occurring in two conditions.

- With rapidly developing intumescent cataract of senile type (priestly Smith 1879, Salus 1910, Marox 1922 and Sugar 1941)
- In traumatic cataract caused by a perforating injury of the lens.

Progressive hydration of the cortical layers may cause swelling of the lens thus making the anterior chamber shallow (intumescent cataract). In the early stages of cataract, particularly the rapidly developing forms, hydration is a prominent feature, so that frequently droplets of fluid collect under the capsule, forming lacunae between the fibers. The entire tissue swells and becomes opaque. To some extent this process is reversible. The process is seen dramatically in traumatic cataract. When the capsule is ruptured, the lens fibers swell and bulge out into the anterior chamber. Hypermetropic eyes are more prone for attack of Phacomorphic glaucoma[18, 19].

The mechanism of Glaucoma

In Phacomorphic Glaucoma, senile cataractous lens can become intumescent and it increases in thickness which leads to pupillary block [48]. This iridolenticular apposition disrupts the flow of aqueous humor from the posterior chamber to the anterior chamber [24]. This results in the accumulation of aqueous in the posterior chamber, pushing the iris root forward, which may ultimately contact the trabecular meshwork and lead to angle closure. Risk factors predisposing to Phacomorphic glaucoma include hyperopia, which is most commonly associated with a shallow anterior chamber [48].

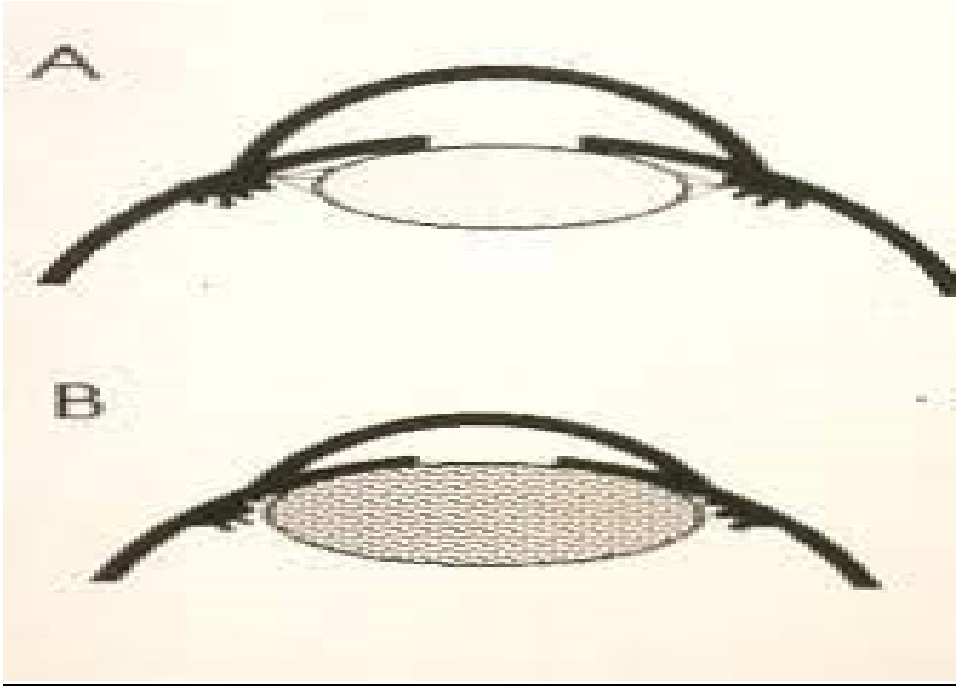

Figure 5: Swollen lens - pupillary block - iris bombae - angle closure

Clinical Features

1. Acute onset of pain and redness
2. Gradual reduction of visual acuity over preceding months.
3. Higher intraocular pressure >21mm Hg
4. Corneal edema
5. Anterior chamber is shallow
6. Pupil is mid dilated and fixed not reacting to light.
7. Formation of peripheral anterior synechiae in long duration.
8. Intumescent cataract.

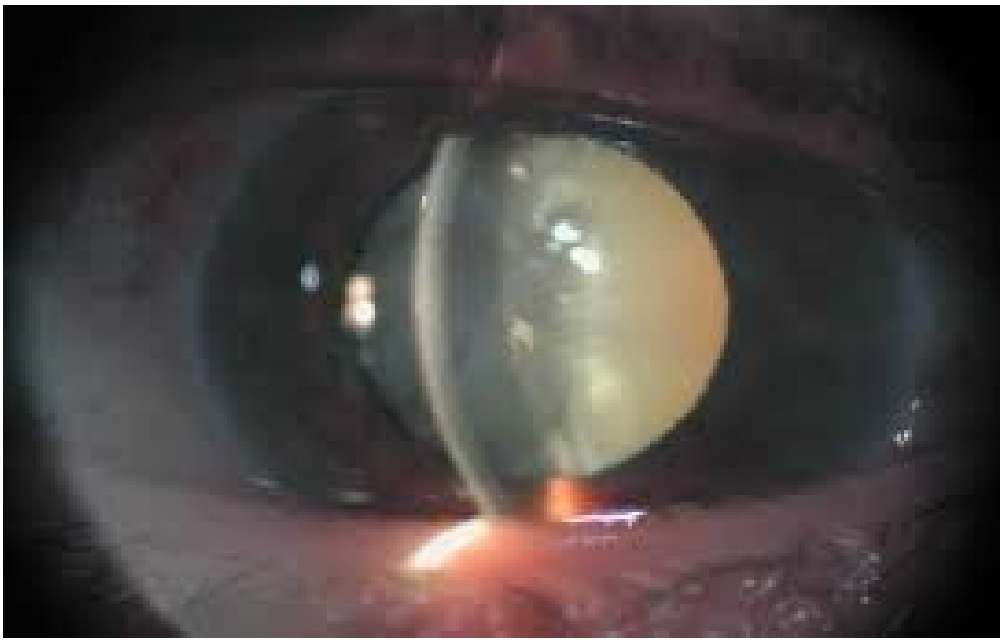

Figure 6: Phacomorphic glaucoma

Management

The first line of management for Phacomorphic Glaucoma includes IOP-lowering medications[24]. They include

- Hyperosmotic agents (mannitol IV), Oral glycerol.
- Carbonic anhydrase inhibitors (Systemic and Topical)
- Aqueous suppressants - Topical betablockers
- Topical steroids

In resistant cases Yag iridotomy can be performed. Prophylactic laser iridotomy should be performed in the fellow eye if the fellow eye is anatomically predisposed to the angle closure glaucoma [44].

After IOP is brought under the control, lens extraction remains the definite treatment in these patients. Manual Small Incision Cataract Surgery (MSICS) and Phacoemulsification achieves excellent surgical outcomes with very less complication rate [33]. Good visual recovery with control of IOP has been reported by most of the studies following this procedure [15, 25, 26].

Phacotopic glaucoma

Phacotopic glaucoma is due to crystalline lens displacement from its natural position. It may be due to acquired or congenital or hereditary causes. The displacement is partial (subluxation) or complete (luxation) [27]. The Zonules of Zinn that holds the crystalline lens to ciliary body are lost either partially or completely because of

1. Trauma, the most common cause.
2. Congenital - Partial or total absence of zonules (microspherophakia)
3. Hereditary causes (Weil-Marchesani Syndrome, Marfans syndrome)
4. Metabolic causes (Homocyctinuria)
5. Degenerations of zonules (uveitis, old age, Hypermature cataract)
6. Sulfite oxidase deficiency- defective zonular apparatus.

The displaced lens may occupy any part of posterior chamber or pupil or anterior chamber. If all the zonules are absent the crystalline lens changes its shape from biconvex to spherical shape increasing its anteroposterior diameter (micro-spherophakia). In some cases angle of the anterior chamber anomalies are associated with above conditions. Trauma can produce any type of lens induced glaucoma. The altered shape of lens in microspherophakia occludes the pupil and is aggravated and relieved by miotics but by mydriatics, and called as 'Inverse glaucoma'.

Clinical features

Dislocated lens cause either pupillary block which leads to angle closure glaucoma or directly encroach upon the angle [24].

- Painful and red eye
- Decreased visual acuity
- Phacodonesis
- Iridodonesis
- Shallowing of anterior chamber either symmetrically or asymmetrically.

Management

Approach for the management of Phacotopic Glaucoma depends on the symptoms and degrees of dislocation. A conservative non-intervention strategy can be followed, in case of partial subluxation of the lens within the pupillary space which will not cause significant vision impairment or pupillary block [24]. Laser peripheral iridotomy is the appropriate solution, if the subluxation is associated with pupillary block. Removal of lens is required in total anterior dislocation.

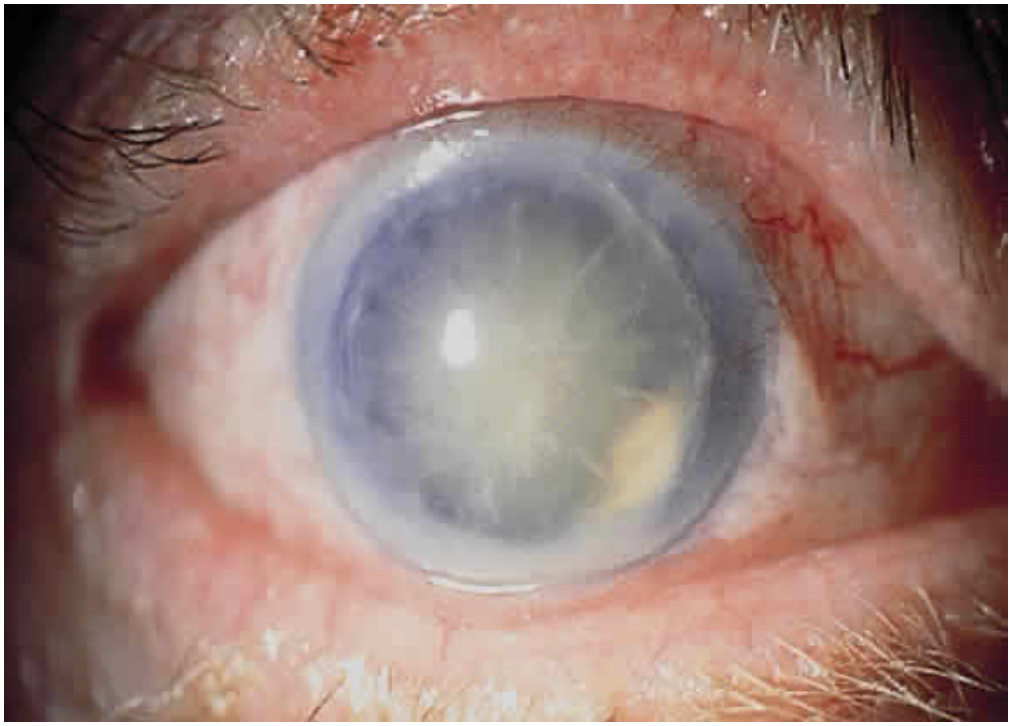

Figure 7: Phacotopic glaucoma

Phacolytic Glaucoma

Ulric (1882) first noticed glaucoma in a case of spontaneous capsular rupture and called this condition as "phacogenic glaucoma" and "lens protein glaucoma". In 1955, Flocks et al [21], named this condition as “Phacolytic glaucoma". Phacolytic glaucoma is a secondary open angle glaucoma due to heavy molecular weight lens proteins, which are lysed and engulfed by the large macrophages and infiltrate the angles of the trabecular space, obstructing the aqueous outflow.

Mechanism

The pathogenesis of this condition states that macrophages were responsible for the increase in IOP by blocking the trabecular meshwork [28, 29]. Lens material may cause aqueous outflow obstruction at the drainage angle. This may occur after injury or cataract extraction or when the lens material leaks through the lens capsule of the immature or hyper mature lens. Macrophages in an attempt to remove this material, engulf the lens protein which itself may cause blockage at the angle of anterior chamber.

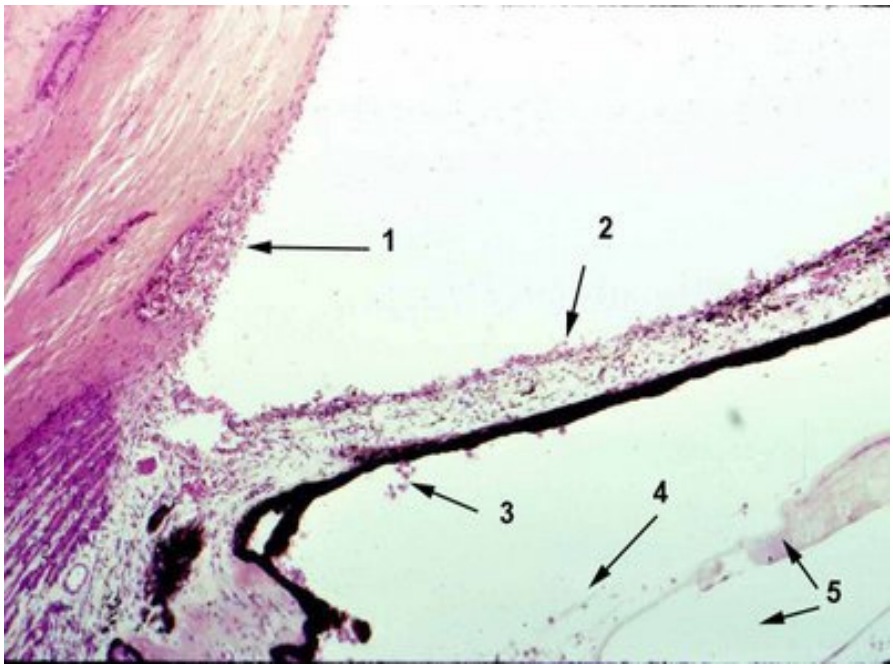

Figure 8: Histopathology of Phacolytic glaucoma

Histopathology

- Accumulation of proteinaceous material and macrophages in angle structures and trabecular meshwork (arrow 1) [49].
- Macrophages can be found on the anterior surface of iris (arrow 2)
- Posterior surface of iris (arrow 3)
- Lens capsule and lens zonules (arrow 4)
- Lens may appear shrunken with loss of nuclear and cortical material and the lens capsule may appear collapsed (arrow5).

Research done by Epstein et al., emphasized on the role of heavy molecular proteins (HMW) that are leaking from the lens, which further leads to aqueous outflow obstruction and it de-emphasized the role of the macrophages [28,29,30,44]. In a series of 13 patient's aqueous humour were obtained by paracentesis at the time of cataract surgery from six patients with Phacolytic glaucoma and from six control patients with immature cataracts. Quantities of HMW protein which is sufficient to obstruct aqueous outflow were identified in all the six Phacolytic aqueous humor specimens but it was not noted in any of the controls [28]. Three of the hypermature cataractous lenses from the above cases of Phacolytic glaucoma were found to have 14-fold greater quantities of HMW protein in their liquefying cortex than were present in the cortex of immature cataractous lenses.

Clinical Features

1. Acute or subacute attacks of pain and redness of the eye, nausea and vomiting in persons with a history of mature/ Hypermature Morgagnian cataract of several years duration.
2. Higher intraocular pressure
3. Diffuse corneal edema
4. Pupils sluggishly reacting.
5. Normal or deep anterior chamber containing floating lens particles and /or with pseudo hypopyon in severe cases.
6. Slit lamp examination reveals aqueous flare from minimal to dense opalescence, indicating a high concentration of protein in the aqueous, and free-floating particulate matter.
7. Diffuse keratic precipitates and intense flare were seen.

Management

The initial treatment of Phacolytic Glaucoma focused upon lowering of IOP using the combination of topical and systemic IOP lowering agents. Topical steroids helps in reducing inflammation and it also facilitate lowering of IOP and decrease the pain. Medical therapy is only a temporary measure until cataract surgery can be scheduled [24]. Cataract extraction is the definitive treatment, which is accompanied by thorough anterior chamber irrigation to remove all the lenticular matter. Manual SICS has been reported by some

surgeons to be beneficial with early visual recovery and good IOP control. In a series of thirty three cases Venkatesh and co-workers [31] reported this technique to be a better and safer technique in advanced cases but a challenging situation for the surgeons.

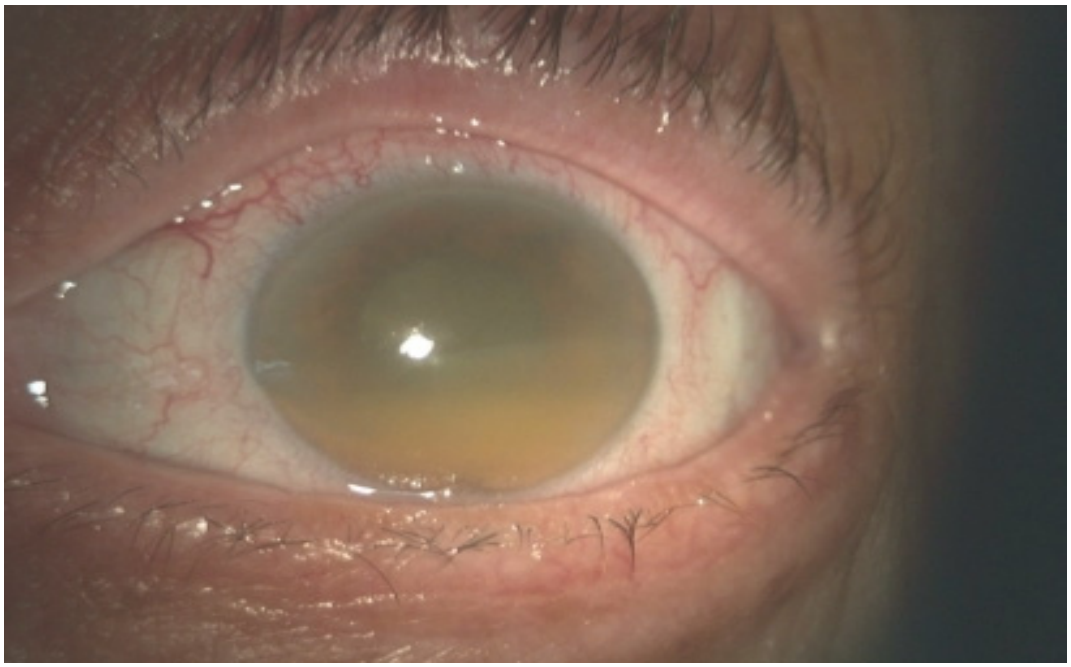

Figure 9: Phacolytic glaucoma

LENS PARTICLE GLAUCOMA

It is a form of LIG with open angle. It is characteristically associated with the disrupted capsule and presence of lens material in the anterior chamber. It may be seen in condition like post cataract surgery, post-trauma, post Yag-cap [24].

Mechanism

The mechanism of the lens particle glaucoma is characterised by the obstruction of the aqueous outflow at the trabecular meshwork by the floating lens particles or engulfed macrophages.

Clinical features

It is asymptomatic or it depends on the severity of intraocular inflammation. Elevated IOP may be present with redness, monocular eye pain and blurring of vision, previous history of trauma or intraocular surgery [26, 32]. The onset of lens-particle glaucoma has been reported to occur many years after cataract surgery [33, 34]. There is a variable degree of inflammation ranging from free-floating fragments of cortex to a layered pseudo-hypopyon. Particles of cortex or nucleus may be seen dislocated into the vitreous cavity.

Treatment

Initially, trial of antiglaucoma medications may be attempted. Miotics should be avoided due to its pro-inflammatory nature [24]. Mild to moderate doses of steroids should be given depending upon the severity of inflammation. A large amount of lens material in anterior chamber or vitreous cavity results in uncontrollable glaucoma, warranting surgical removal.

PHACOANAPHYLACTIC GLAUCOMA

It is a rare clinical entity due to inflammatory response directed against one's own lenticular antigen, which results in IOP elevation due to the

- Involvement of trabecular meshwork by inflammation
- Obstructions of trabecular meshwork by inflammatory cells.

This is called as Phacoanaphylactic uveitis or Phacoanaphylactic glaucoma.

Verhoeff and Lemoine (1922) first termed this condition as "endophthalimitis-phaco-anaphylactica". Phacoanaphylactic glaucoma is synonymous with lens induced uveitis and phacoantigenic endophthalmitis and it is a rare clinical entity. Straub described the occurrence of inflammation in the un-operated, non-traumatised eye after extracapsular extraction of the opposite eye. Circulating antibodies against lens proteins have been demonstrated in the vitreous, aqueous of the patients by Cooper (1957).

Pathogenesis

Usually this condition is preceded by lens capsule disruption, however in contrast to lens particle glaucoma; latent period of 24 hours to 14 days is usually present between trauma and onset of inflammation [24]. When patient gets sensitized to their own lens antigen, the proteins are kept in an immunologically privileged state. They are kept within the lens capsule. Post-surgery or trauma to the lens capsule, the lens antigens are exposed to circulation. This will further be recognised as "FOREIGN" by the immune system. Thus this further incites an inflammatory response. It seems to be an Arthus- type of immune complex reaction which is mediated by IgG and the complement system [35, 36].

The inflammatory activity is against the lens material with varying stages of degeneration with infiltration of the polymorph nuclear leucocytes, lymphocytes, plasma cells and eosinophils. This is surrounded by a zone of chronic granulomatous inflammation including phagocytic epithelial cells and giant cells.

Clinical features

Patients usually present hours to months after surgery or trauma. All inflammatory signs like conjunctival congestion, Keratic precipitates, aqueous flare and cells, initially low IOP and sterile hypopyon are present. Posterior synechiae formation followed by obstruction to aqueous flow produce iris bombe and peripheral anterior synechiae leading to rise of IOP. The

predominant presentation in such a condition is chronic granulomatous type of inflammation with associated glaucoma which may be a rare feature.

Management

Initial measure to control inflammation is by use of steroids. IOP control is by anti-glaucoma medications [24]. When medical measures are inadequate, the retained lens matter has to be removed surgically.

MANAGEMENT OF LENS INDUCED GLAUCOMA

Initially, Lens induced glaucoma is managed medically. But the definitive management for lens induced glaucoma is surgical removal of the lens. Before surgical removal of the lens, the elevated IOP and uveitis are controlled to prevent complications.

- Medical
- Surgical

Medical Management

The medical management is to reduce IOP, patients discomfort and to reduce inflammation so as to facilitate surgery, which is as follows:

I. To reduce raised IOP

1) Carbonic anhydrase inhibitors

Systemic - Acetazolamide (250mg tid/qid)

Topical - Dorzolamide, Brinzolamide

Mechanism of action

It decrease IOP by decreasing aqueous humor production by the inhibition of carbonic anhydrase II ISO enzyme in the ciliary epithelium

Side effects

- Metabolic acidosis
- Agranulocytosis
- Neutropenia
- Abdominal discomfort
- Exfoliative dermatitis
- Steven-johnson syndrome

Ocular side effects

- Hypersensitivity reactions like periorbital dermatitis
- Superficial punctate keratitis
- Burning sensation

Contraindication

- Renal failure
- H/o sulfa drug allergy
- Chronic liver disease

2) Hyper osmotic agents

- Intravenous 20% Mannitol (1-2 g/kg)
- Oral 50% Glycerine (1-1.5 g/kg)

Mechanism of action

Increase the osmolarity of plasma and thereby water from the eye (mainly from vitreous) moves to the hyperosmotic plasma. This movement of water reduces the vitreous volume and causes lowering of IOP

CNS action decreases the aqueous production.

Side effects

- Rebound of IOP
- Intra ocular haemorrhage
- Diuresis
- Electrolyte imbalance
- Hypovolemia
- Hyperglycaemia
- Hypersensitivity

Contraindications

- Anuria
- Severe dehydration
- Severe cardiac decompression
- Pulmonary edema

3) Beta-adrenergic blocking agents

- Timolol 0.5%
- Betaxolol 0.5%
- Levobunolol 0.5%

Mechanism of action

Topical beta blocker inhibits cyclic adenosine monophosphate (cAMP) production in ciliary epithelium, thereby decreasing aqueous production (20-50%) and hence reducing the IOP (20-30%).

Side effects

- OCULAR: Allergy, Punctate epithelial erosions, Decreased corneal sensation
- CVS: Bradycardia, Hypotension, Heart failure
- RS: Bronchospasm, Asthma, Emphysema
- CNS: Sleep disorder, Depression

Contraindications

- Congestive cardiac failure
- Second / third degree heart block
- Bradycardia
- Asthma
- COPD

4) Alpha- adrenergic agonists

- Brimonidine.
- Apraclonidine

Brimonidine

Mechanism of action

- Decrease aqueous production
- Increases uveo scleral outflow
- Neuroprotection

Apraclonidine

Mechanism of action

- Reduce aqueous production
- Improved trabecular outflow
- Reduced episcleral venous pressure

Side effects

- Ocular: Allergic blepharo-conjuctivitis, Anterior uveitis
- CVS: Bradycardia, Vasovagal attack, Postural hypotension
- CNS: Somnolence

Cycloplegic mydriatics

- Atropine Sulphate 1%
- Homotropine

Contraindication for LIG

- Pilocarpine nitrate 2%
- PG analogues

To Control Uveitis

- Steroids (Topical, systemic)

III. Symptomatic

- Analgesics / Anti-inflammator

SURGICAL MANAGEMENT

The definitive treatment of lens induced glaucoma is lens extraction with or without filtering surgery. The raised IOP should be controlled by medical treatment i.e. by beta blockers, carbonic anhydrase inhibitors and hyperosmotic agents prior to the surgery.

Recently a prospective, randomized control trial conducted in Nepal concluded that both Manual Small Incision Cataract Surgery (MSICS) and Phacoemulsification achieves excellent surgical outcomes with very less complication rate [33]. MSICS is significantly faster, less expensive, less technology dependent when compared to Phacoemulsification. Inspite of faster recovery with the phaco emulsification the most appropriate surgical procedure for the treatment of cataract is MSICS.

Anaesthesia

MSICS can be performed under peribulbar, retrobulbar, sub-Tenon's anaesthesia.

Creating a sclerocorneal tunnel

After attaining adequate mydriasis, a superior rectus bridle suture is placed. Hemostasis is achieved with the help of bipolar diathermy cautery when a fornix – based conjunctival flab is been created.

A curvilinear partial-thickness scleral incision is made with size of 3 mm posterior to the limbus. Several kinds of incisions have been described which includes straight, frown, smile and chevron. The incision is made smaller for a soft cataract and is made larger for a dense and hard cataract. Usually, the incision is of size 6 to 7 mm long for a cortical cataract, and size of 7 to 8 mm

long for a hard cataract (such as 4+ nuclear sclerosis). The depth of it should be approximately 0.3 mm.

This incision is made by advancing the crescent knife into the sclera and slowly cutting on either side, making room for the crescent knife. The adequate depth is judged by the visibility of the knife (i.e., the crescent should be just visible through the sclera). For ensuring uniform depth of the tunnel during the dissection process the heel of the crescent should be flat on the globe.

Once the limbus is reached, the tunnel is converted to a flap by forward and backward motion, cutting the tissue while coming out. On either side of the tunnel, scleral pockets are created. This helps to accommodate the nucleus during delivery. The width of the tunnel (i.e., the distance between the scleral incision and the inner corneal incision at the level of Descemet membrane) should be 4 mm .The tunnel isin the shape of trapezoidal this is because the inner corneal incision is 25 percent larger when compared to the scleral incision.

Creating a paracentesis

A paracentesis is made in the 9 o'clock position using a 15- degree lancet tip blade and 24-gauge. The lens capsule is stained with trypan blue injected through the side port under an air bubble to protect the corneal endothelium. It is desirable to wait for 30 seconds for adequate staining of the capsule. The

trypan blue is washed out of the eye by using balanced salt solution. The anterior chamber is deepened by injecting a viscoelastic.

Making the internal corneal incision

The Keratome is advanced through the tunnel carefully. It is then tilted downward and it is slowly advanced to extend the tunnel into the anterior chamber. Then it is moved forward and laterally, for creating an internal incision which will be parallel to the limbus. Further the internal incision is extended on both sides.

Capsulorhexis and nucleus delivery

A continuous curvilinear capsulorhexis is made using a cystotome or a rhexis forceps. For a easier prolapse of the nucleus a large rhexis is preferred.

The minimization of the aspiration of residual cortex happens when the multiple small hydro dissections facilitate prolapse of the nucleus into the anterior chamber.The nucleus can largely be prolapsed with hydrostatic force created by injecting BSS in the bag during hydro dissection. Any un-prolapsed portion of the nucleus can be captured using a Sinskey hook as one would use a tire iron.

Nucleus is delivered with an irrigating lens loop. The globe is then stabilized and then the irrigating lens loop is introduced through the tunnel. Further it is positioned between the iris and the nucleus. The nucleus is then engaged in the lens loop and is slowly withdrawn from the anterior chamber while the posterior

lip of the tunnel is depressed. It should be noted that depressing the posterior lip opens the tunnel, and lifting the anterior lip closes the tunnel. After the nucleus gets engaged in the tunnel the BSS is injected steadily. The hydrostatic pressure within the anterior chamber facilitates the delivery of the nucleus.

Residual cortex aspiration and implantation of a posterior chamber IOL

The residual cortex is aspirated using a Simcoe cannula, and the subincisional cortex is aspirated through the paracentesis port. The anterior chamber is washed with the help of Balanced Salt Solution (BSS), and then viscoelastic is injected into the anterior chamber [50]. A posterior chamber IOL is implanted in the bag through the tunnel and dialed in. Viscoelastic is aspirated and washed out with a Simcoe cannula.

Sealing the paracentesis port

The paracentesis port is sealed by hydrating the stroma [50]. This is done by injecting BSS steadily into the corners of the incision.Intra cameral Moxifloxacin should be given before hydrating the port.

Once the port site is sealed, the anterior chamber deepens, and the globe becomes firm [50]. The tunnel is then checked for integrity. Finally, the conjunctiva is apposed gently by bipolar diathermy cautery.

Special precautions to be taken during surgery for LIG :-

- Create appropriate sclerocorneal tunnel according to nucleus density
- Slow decompression of globe during paracentesis to avoid supra choroidal haemorrhage.
- Enhance visualisation of Anterior Lens Capsule (ALC) by using tryphan blue.
- Avoid hydrodissection
- Bimanual technique for nucleus prolapse
- Careful cortex aspiration and IOL implantation.
- Meticulous vitrectomy in case of intraoperative complications and vitreous loss.
- Thoroughly wash AC after IOL placement, remove residual lens matter and visco elastic to prevent post-operative IOP spike.
- Continue anti – glaucoma medications and intense antiflammatory therapy in post-operative period till IOP becomes normal.

PART B

MATERIALS AND METHODS

Objective

Primary Objective

- To access the Visual Acuity and IOP following cataract surgery in patients with lens included glaucoma.

Secondary Objective

- To access the extent of the disc damage in patients with lens included glaucoma following cataract surgery.
- To access the need for further medical and surgical management following cataract surgery in patients with lens included glaucoma.

Study Area

- This study was done at the Glaucoma department of AEH, Madurai, Post graduate institute of Ophthalmology, Madurai.

Study Design

- A prospective observational study.

Study Population

- Patient who were diagnosed with lens induced glaucoma in glaucoma department of AEH, Madurai were taken for the study.

Study Duration

This study was conducted from January 2018 – June 2018 at Aravind Eye Hospital (AEH), Madurai.

Inclusion Criteria

- All patients diagnosed with lens induced glaucoma.

Exclusion Criteria

- Patients with primary glaucoma.
- Patients with secondary glaucoma other than lens induced glaucoma.

Sample size

Patients who presented with lens induced glaucoma at the glaucoma department of AEH, Madurai from January 2018 – June 2018 were recruited for the study.

Statistical analysis

STATA 11.1 (Texas, USA) software was used for the statistical analysis. With and Confidence Interval (CI) OF 95% and $P<0.05$ for the statistical significance, Chi square test was used to analyse the association between two variables. Mann- Whitney U test was used to analyse the difference between continuous variable. Paired T test was used to analyse the differences.

METHODOLOGY

- A prospective observational study was conducted at the Aravind Eye Hospital and Post graduate Institute of Ophthalmology, Madurai, India.
- All the patients who attended the Glaucoma department during January 2018 to June 2018 with a diagnosis of LIG were included in this study.
- Patients with a history of primary open angle or narrow angle glaucoma, trauma and patients with LIG with prior ocular hypotensive management elsewhere were excluded in this study.
- A complete history related to the illness was taken with duration of decrease in vision, mode of onset, redness, watering, pain or any other associated symptoms.
- Phacomorphic glaucoma was recognized by the subjective complaints like pain, redness of the eye with the presence of corneal edema, shallow anterior chamber, fixed dilated pupil and an intumescent cataractous lens.
- Phacolytic glaucoma was diagnosed by the presence of pain, corneal edema, normal or deep anterior chamber with floating lens particles and/or with pseudo hypopyon in severe cases, flare, cells, with minimal KPs, the presence of mature or hypermature cataract with or without white spots on the anterior capsule

DATA COLLECTION METHOD

All patients underwent comprehensive preoperative evaluation which included were taken for the study.

- Consent was obtained from all patients.
- Demographic details of the study participants (Name, Age, sex, address, Occupation, mobile no, e mail ID)
- Visual acuity using Snellen's chart.
- IOP recording by Goldmann applanation Tonometry (GAT)
- Depth of anterior chamber by slit-lamp (VAN HERICK'S GRADING).
- Gonioscopy for the other eye was done to rule out the angle closure glaucoma.
- Patient's fellow eye with narrow angles and raised IOP were excluded from the study.
- Based on the slit lamp examination, the type of Lens induced glaucoma was determined.
- Fundus examination of the fellow eye to rule out any primary glaucoma.
- B scan ultrasonography: To evaluate the posterior segment and to rule out other pathology.
- Lacrimal syringing to rule out chronic dacryocystitis.

- Systemic evaluation for diabetes and hypertension and renal function test.
- Biometric measurements including axial length and IOL power calculation with IOL MASTER 700.
- Surgery done by senior medical officer.

The initial, control of IOP was done with

- Tab. acetazolamide 250 mg oral four times a day,
- Topical Timolol maleate 0.5% (BD)
- Intravenous Mannitol.
- Topical dexamethasone 0.1% eye drops 4 times a day was given to reduce inflammation.
- Post-operatively patients were given with topical antibiotics and steroids for 6-8 weeks.

On follow up at 1st day,1st month and 3rd month patients underwent independent ophthalmic examination, which includes

- Visual acuity,
- Anterior segment examination,
- Tonometry and fundus examination was done.

REVIEW OF LITERATURE

Flocks et al. [37] 1955 suggested that glaucoma was induced due to the obstruction of inter-trabecular spaces by macrophages distended with engulfed lens material and also by Morganian fluid that is escaped from the lens, and hence termed as Phacolytic glaucoma .

Jain Is, Gupta A et al [25] (January, 1978 to June, 1981) conducted a study on phacomorphic glaucoma and visual prognosis. Incidence of phacomorphic glaucoma among 2719 senile cataracts undergoing surgery was 3.91 %. Of the 86 cases available for study, 77 eyes underwent simple cataract extraction with peripheral or sector iridectomy. More than 90 % of the cases were normotensive at the end of the follow up. Preoperative IOP, accuracy of light perception and final visual recovery were significantly related to duration of glaucoma. A good functional recovery was obtained if the attack lasted less than 20 days, beyond which only a hand movement or perception of light could be recovered. More than 75% of the optic discs were normal if the attack lasted less than 10 days.

Prajna R V et al [38] conducted a study in **India (1994)** on LIG's visual outcomes and also the risk factors for final visual acuity. Their study showed incidence of Phacomorphic glaucomas (52.7%) is slightly higher than Phacolytic glaucoma (47.3%). The study concluded that there was no statistically significant difference between the two groups on the final postoperative visual recovery. 57% of Phacomorphic glaucomas and 61% of

Phacolytic glaucomas attained postoperative corrected visual acuity of 6/12 and better. A significant risk of poor visual acuity was found when the duration between the onset of pain and surgery exceeded five days (OR = 3.1; 95% CI: 1.21-8.13). Marginally significant risk of poor visual outcome was observed in cases of age higher than 60 years when compared with younger patients.

Rijal AP, Karki DB [26] conducted a study on visual outcome and IOP control after cataract surgery in lens induced glaucoma included 40 patients from January 2002 to December 2004 were analysed to find out the demographic features, visual outcome, IOP control, and duration between appearance of symptoms and surgical intervention. The total number of females (55.45%) has outnumbered males. The incidence of Phacomorphic glaucoma was more compared to the Phacolytic type (65:35). Pre-operative IOP ranged between 24.0- 59.0 mmHg and the post-operative IOP ranged between 14- 22 mmHg. The duration between the onset of symptoms and surgery was 1 week to 4 months [11].

Ramakrishanan R et al. [39] conducted a study at **Tirunelvelli, India (March 2006-April 2007)** on IOP control, visual prognosis and complications in Phacomorphic glaucoma followed by a manual small incisional cataract surgery. Mostly all the patients who were diagnosed with Phacomorphic glaucoma between March 2006 and April 2007 were included the study. The conclusion from this study said that MSICS achieved excellent visual outcome

with low complication rates and thus MSICS is the said to be the appropriate surgical procedure for the treatment of advanced cataract in the developing world.

Tomey KF et al.[40] 1992 this study stated that YAG laser iridotomy is the initial management of Phacomorhic glaucoma. The conclusion of this study showed that the angle closure attack was relieved in all cases when they were treated with laser iridectomy. This played an important role in bringing the pressure under control before proceeding further with cataract surgery.

Pradhan D et al. [15] 1998 done a prospective case series in Nepal, with 413 patients diagnosed as lens induced glaucoma to determine the frequency, types and reasons for late presentation and final outcome of management. In this series Phacomorphic glaucoma was more common (298 eyes; 72%) than Phacolytic glaucoma (115 eyes; 28%). LIG was more common in females, with a ratio of 1.7:1. It is possible that socio-economic and cultural constraints play a role leading to neglect and late presentation of cataract in this region. One in three patients reported that they had no escort to the hospital and a similar proportion stated that they could not afford surgery. Some patients in this part of Nepal and North India with cataract will wait for free eye camp surgery in the vicinity of their homes rather than visit a distant hospital for treatment.

Pant Sitoula et al [41] 2014. This was a retrospective analysis of prospectively collected data of 42 LIG patients in eastern Nepal. In this study Phacomorphic glaucoma was more common 57.5% than Phacolytic glaucoma 42.5%. Female to male ratio was 2.07:1. High female ratio is due to the fact that older females are given less attention and they are dependent on other family members physically and financially. This study shows the importance of educating the community about early cataract surgery and complications due to LIG. As a result people in the community would come out from the misbelief that cataract should not be operated unless it has matured. Cataract removal is the definite and best management of LIG.

Yaakub A et al [42] conducted a retrospective analysis of LIG patients between January 2003 and December 2008 in Malaysia. 38 patients were included in this study. This study shows that mean age was 70.2 years and women are more predisposed to LIG due to higher prevalence of cataract in them. In this study it was said that phacomorphic glaucoma was the main cause of LIG which is then followed by Phacolytic. This study revealed that the probable cause for late presentation to the eye care services even with the presence of pain was found to be due to the inaccessibility to eye care and lack of awareness. Early detection of lens-related problems can prevent the elevation of IOP, which may further lead to optic neuropathy if untreated. There must be public awareness on

the benefits of early detection. Early treatment of cataract is important in the prevention of LIG [42].

RESULTS

A total of 101 patients were recruited for this study. The Mean ± (SD) age is 64.36 ± (9.20) years and it ranges from 41 to 82 years. The highest numbers of patients with LIG were found in the age group of 61 – 70 years (35.6%), and the lowest were seen in the age group of 41 – 50 years (7.9%).

Table:1 Age distribution

Age	**n**	**%**
41 to 50	8	7.9
51 to 60	31	30.7
61 to 70	36	35.6
More than 70	26	25.7
Overall	**101**	**100**

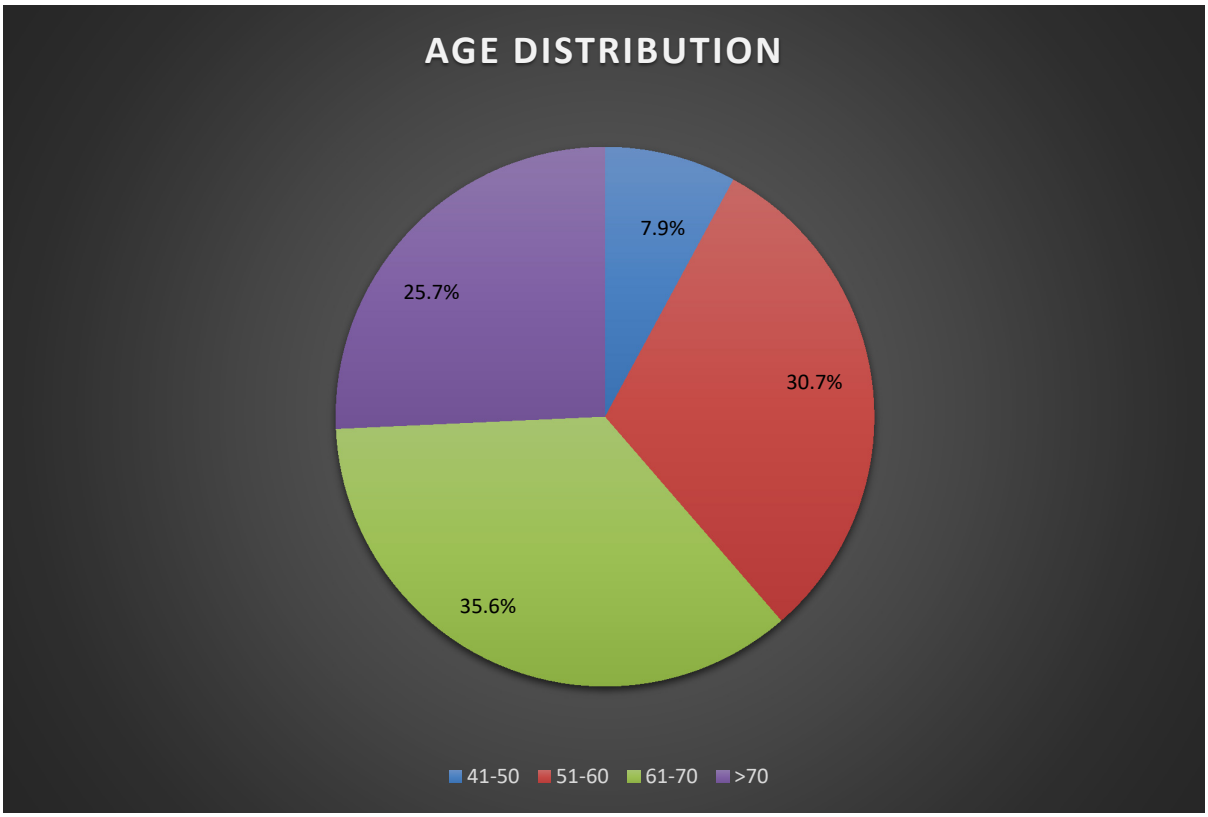

Fig 10: Age Distribution

Table:2 Gender: Incidence of LIG

Gender	n	%
Male	51	50.5
Female	50	49.5
Total	101	100

The incidence of LIG for both male and female is shown in Table 2. 50.5% of males are affected by LIG. This also shows that there is only a minor difference in the gender wise distribution of data and both males and females have equal chance of getting affected by the disease.

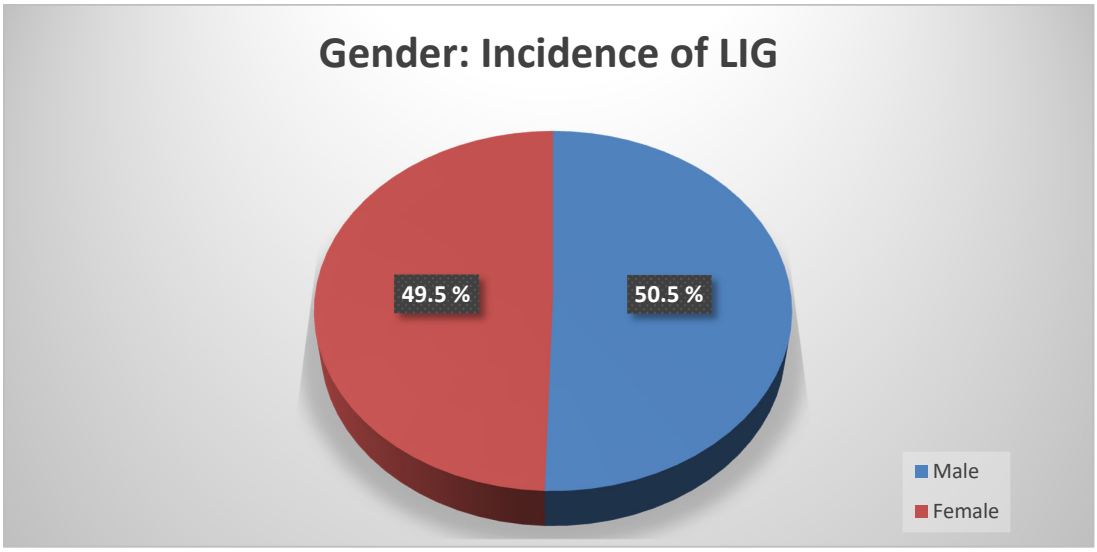

Fig 11: Incidence of LIG among male and female

Table:3 Duration of acute symptoms

Duration of acute symptoms	n	%
<1 week	53	52.5
1 to 2 weeks	27	26.7
>2 weeks	21	20.8
Total	101	100

Among 101 patients, 52.5% of cases presented within a week (<1 week) of acute onset of symptoms. 26.7% patients presented within 1 – 2 weeks and 20.8% of patients presented 2 weeks after the occurrence of acute symptoms. 47.5% of patients presented with the complaints of acute symptoms after 1 week .The demographics are shown in table 3.

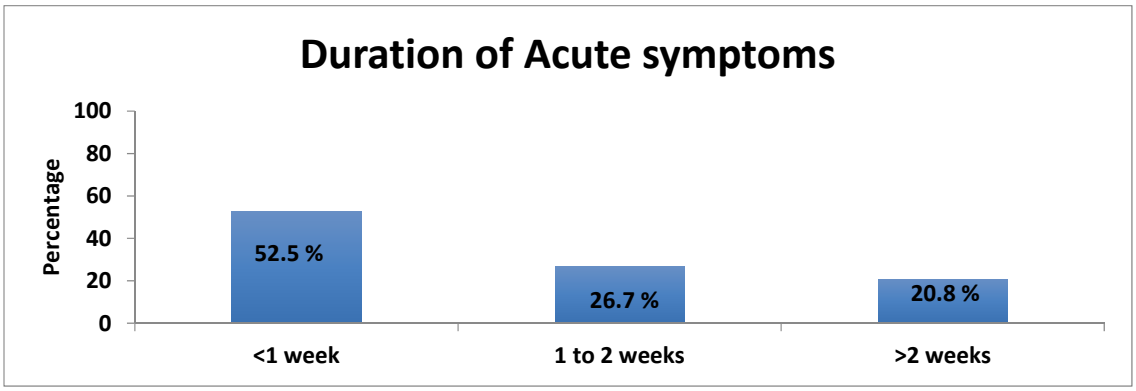

Fig 12: Duration of acute symptoms

Table: 4 Laterality

Affected eye	n	%
RE	57	56.4
LE	44	43.6
Total	101	100

Comparing the laterality between both the eyes LIG was found to be more common in right eye (56.4%).

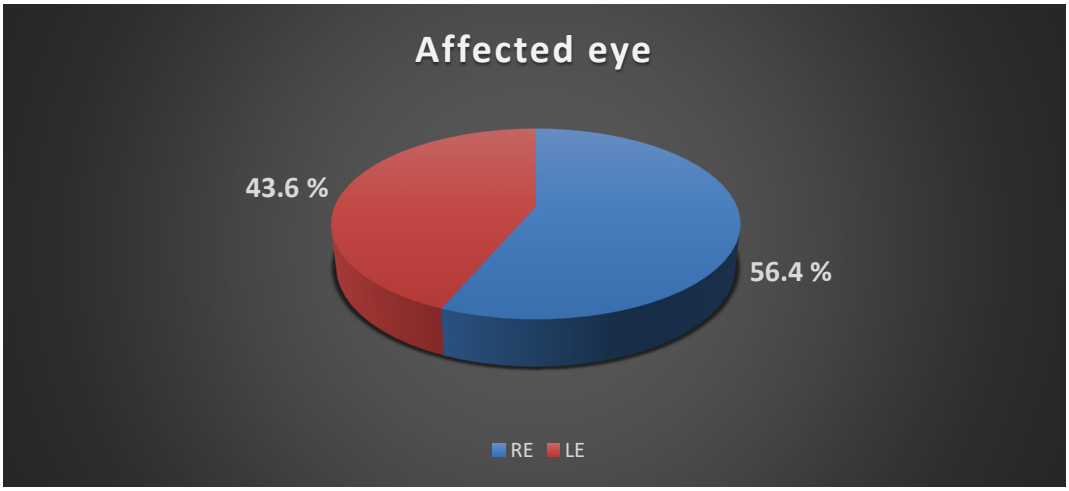

Fig 13:- Laterality of the affected eye

Table: 6 Incidence of LIG

Incidence of LIG	n	%
Phacomorphic	44	43.5
Phacolytic	55	54.5
Others	2	2.0
Total	101	100

In our study, Phacolytic glaucoma (54.5%), was found to be more common than Phacomorphic glaucoma (43.5 %).

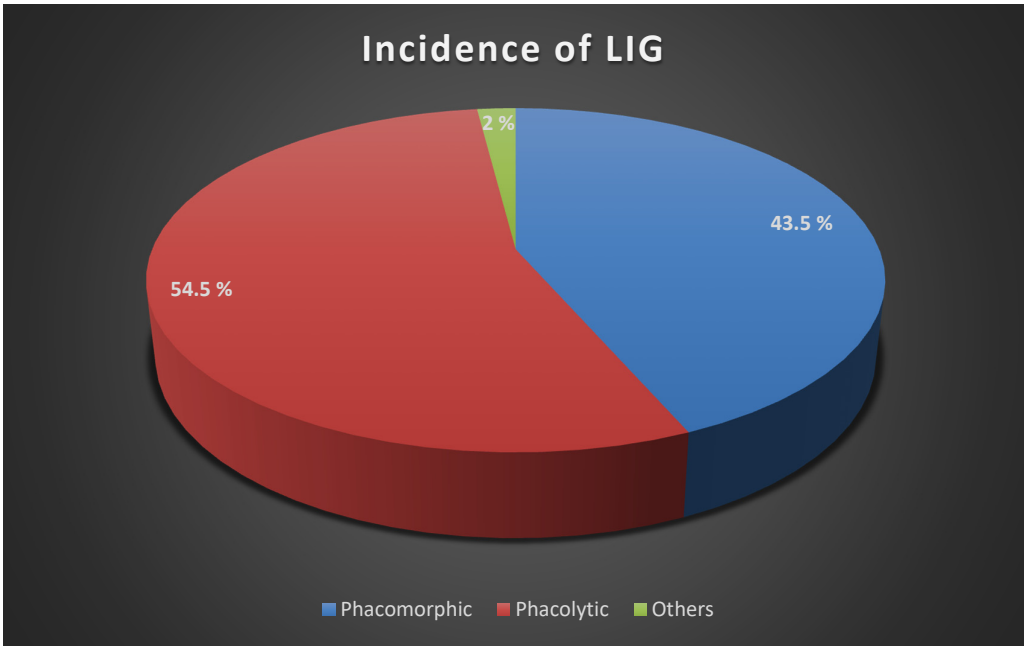

Fig 14: Incidence of LIG

Table: 7 Incidence of LIG vs. gender

LIG	Gender		P-value
	Male	Female	
Phacomorphic	16(36.4)	28(63.6)	**0.02**
Phacolytic	33(60.0)	22(40.0)	
Others	2(100.0)	-	
Overall	51	50	-

When comparing the incidence of LIG with gender it was found that 63.6% of females were more commonly affected by Phacomorphic Glaucoma whereas 60.0% of male were found to be more commonly affected by Phacolytic Glaucoma. 2% of males were affected by Phacotopic Glaucoma. High level of statistical significance was found *(P <0.02)*. This concludes that there is an association between gender and LIG.

Table: 8 Nature of Cataract

Nature of cataract	n	%
Hyper mature cataract	55	54.5
Immature cataract	2	2.0
Mature cataract	44	43.5
Total	101	100

Among the101 patients who were diagnosed with LIG, hyper-mature cataract (54.5%) was found to be the most common type of cataract and following it was the mature cataract (43.5%) and the rest 2% was caused by the immature cataract.

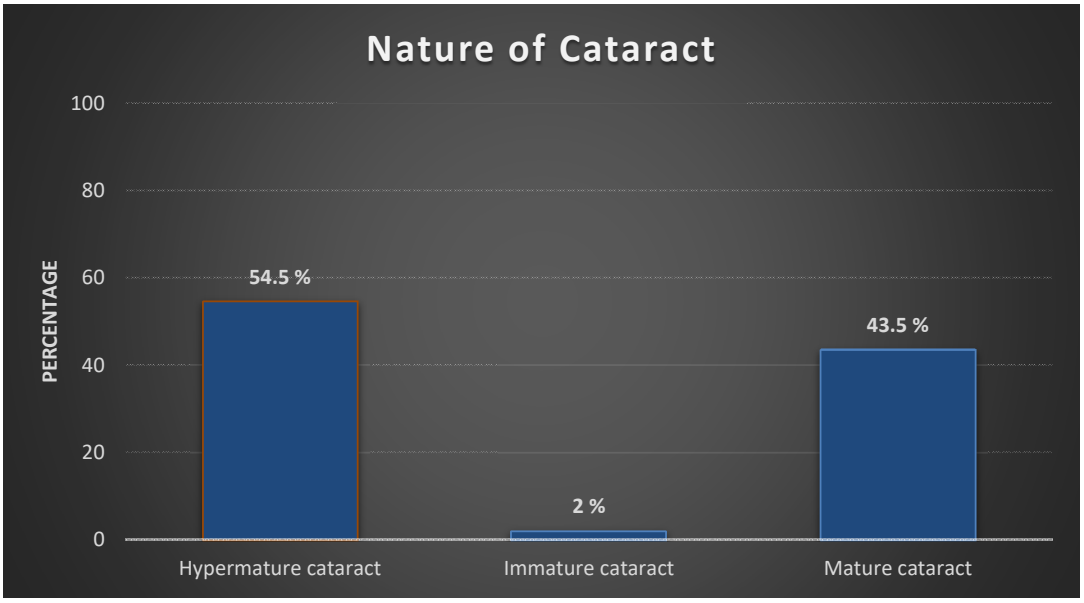

Fig 15: Nature of cataract

Table: 9 Pre-operative baseline visual acuity (BCVA)

BCVA baseline	n	%
6/18	1	1.0
1/2/60	1	1.0
2/60	1	1.0
FCF	4	4.0
HM	55	54.5
LP+	39	38.6
Total	**101**	**100**

Table 9 shows the Pre- Operative Best Corrected Visual Acuity (BCVA) of the patients diagnosed with LIG. Among 101 cases majority of the patients presented with BCVA of ≤HM (93.1%).

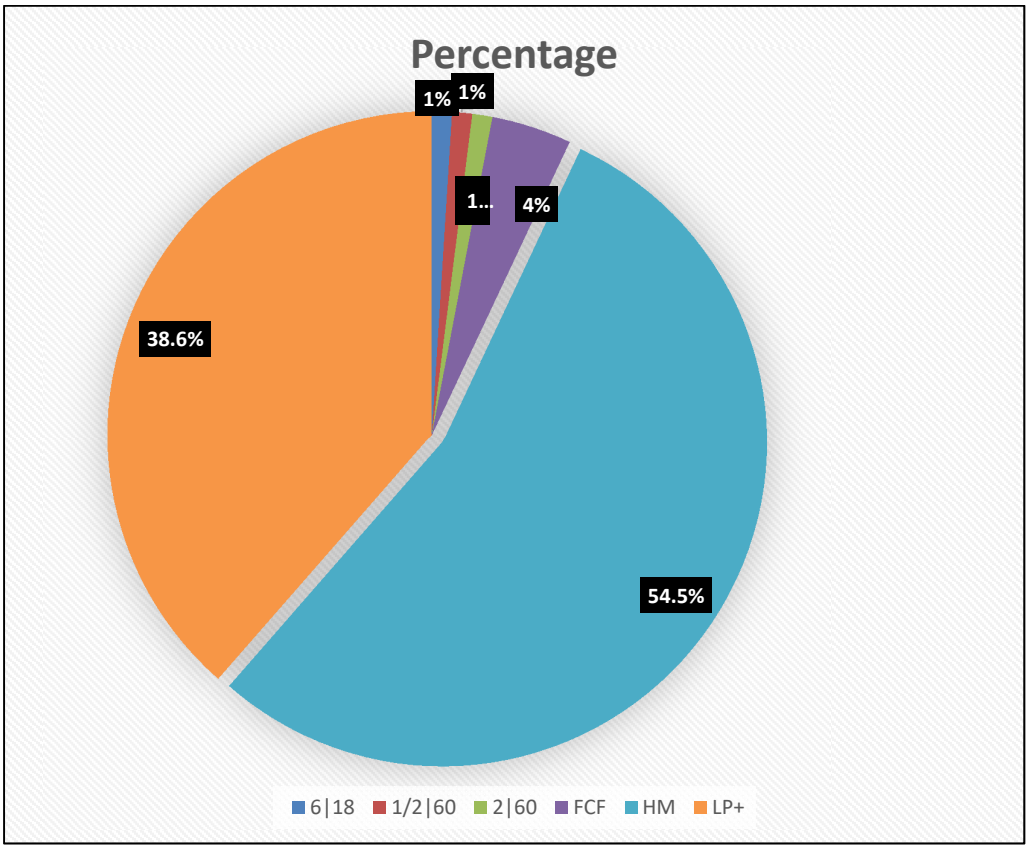

Fig 16: Percentage of preoperative BCVA

Table:10 Pre- Operative IOP (baseline)

IOP	n	%
<=20	9	8.9
21 to 30	17	16.8
31 to 40	27	26.7
41 to 50	32	31.7
More than 50	16	15.8
Overall	101	100

Table 10 shows the frequency of pre-operative IOP. **91.1%** of patients presented with initial IOP of ≥ 20mm/Hg pre- operatively whereas **74.2%** of patients presented with IOP of > 31mm/hg

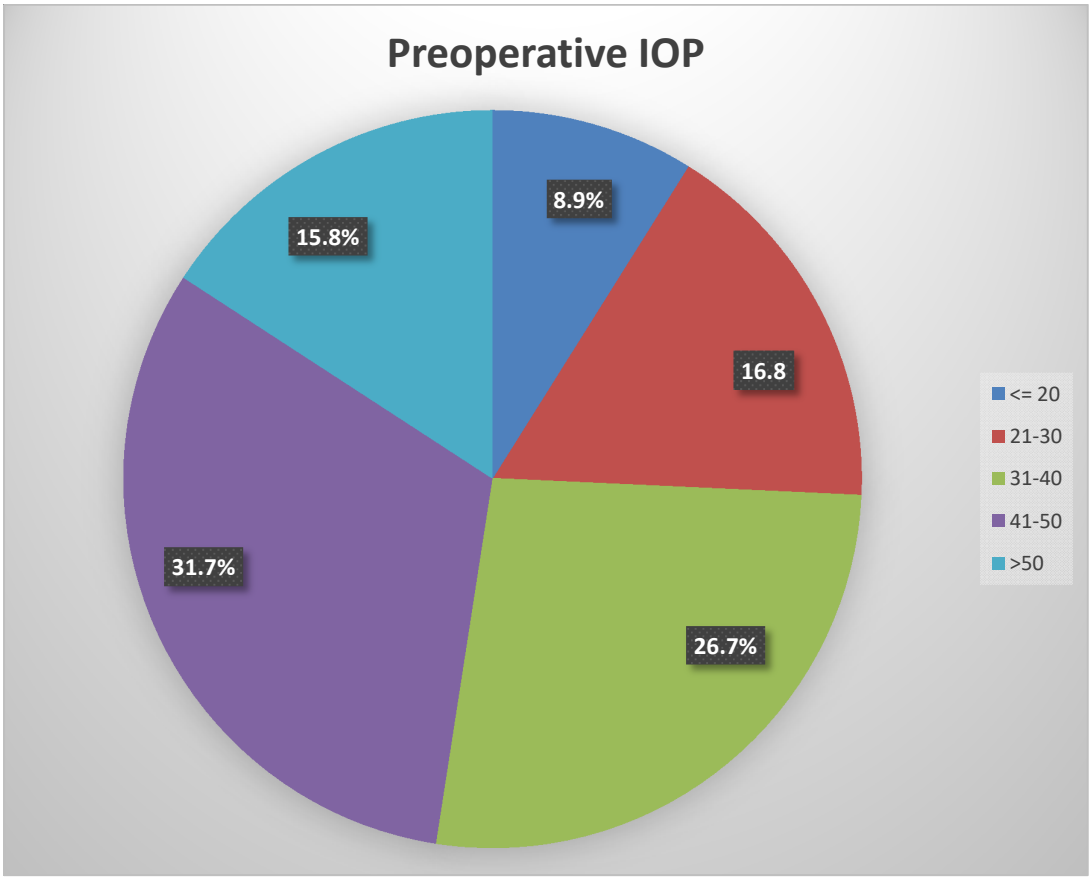

Fig 17: Preoperative IOP

Table 11: Post-Operative Day 1 BCVA

BCVA	n	%
6/9	9	8.9
6/12	29	28.7
6/18	8	7.9
6/24	18	17.8
6/36	19	18.8
6/60	6	5.9
5/60	5	4.9
1/60	2	2.0
HM	3	3.0
LP+	2	2.0
Total	101	100

BCVA was <6/60 for 17.8% of patient at the first day post operatively. 82.2% of patients presented with the vision >6/60.

Table 12: Post – Operative day 1 IOP

IOP	n	%
≤21	90	89.1
>21	11	10.9
Total	101	100

For 89.1% of patient the IOP was ≤ 21 at the first day of follow up and for 10.9% of patients the IOP was > 21.

Table 13: Post-Operative day 1 complication

Complication	n	%
Iritis	101	100.0
Fibrin membrane	15	14.9
Corneal edema	13	12.9
Minimal Residual Cortex	2	2.0
Hyphema	3	3.0
Vitreous in AC	3	3.0

In Table 13, we can see the post-operative complications at day 1. 14.9% of patients presented with Fibrin membrane at day 1 post – operatively and 12.9% of patients presented with corneal edema. 2% of the patients presented with minimal residual cortex. Re-surgery was not done for any of these patients.

Table:14 Tabulation of IOP at various follow up visit:

IOP	Baseline n (%)	1 month n (%)	3month n (%)
<=10	1 (1.0)	29 (28.7)	15 (17.0)
11 to 20	8 (7.9)	59 (58.4)	68 (77.3)
>20	92 (91.1)	13 (12.9)	5 (5.7)
Total	101	101	88

The baseline, 1st month and the 3rd month IOP is shown in Table 14. Reduction of IOP approximately 20 mmHg has been noted between baseline and post-operative IOP.

TABLE:15 Comparison of pre-op and post-op IOP

IOP	n	Mean (SD)	Min – Max	P-value [b]
Baseline	101	39.71 (12.91)	6 – 70	-
1month	101	15.37 (6.86)	8 – 54	<0.001
3month	88	14.70 (4.56)	8 – 34	<0.001

b – Paired t test

Comparison of the Pre-op and Post- op IOP was done using Paired T- Test. High level of statistical significance *(P<0.001)* was found between baseline and 1 month IOP *(Mean (SD) = 39.71(12.91)* and also between baseline and 3 month IOP *(Mean (SD) = 14.70(4.56).* When comparing the post-op IOP (1st, 3rd month) data with the pre-op (Baseline) IOP it is found that there was reduction in the IOP after treatment. Among 101 patients, 13 patients were lost to follow up in the 3rd month.

Table:16 Relationship between duration of symptoms and postoperative IOP

Duration of symptoms	IOP (3rd month)			P-value [e]
	n	Mean (SD)	Min – Max	
<1 week	49	13.80 (3.61)	8 – 28	
1 to 2 weeks	22	14.82 (5.00)	10 – 34	**0.029**
>2 weeks	17	17.18 (5.66)	8 – 30	
Total	**88**	14.70 (4.56)	8 – 34	-

[e] ANOVA test

The table 16 shows the post-op comparison of IOP at end of 3rd month. To find out the significant difference between IOP and duration of symptoms ANOVA test was used. The *p-value (0.029)* shows that there is a high significant difference between two categories. The mean (SD) IOP at third month was **17.18 (5.66)** and it was found to be high in patients with duration > 2 weeks of symptoms when comparing between three groups.

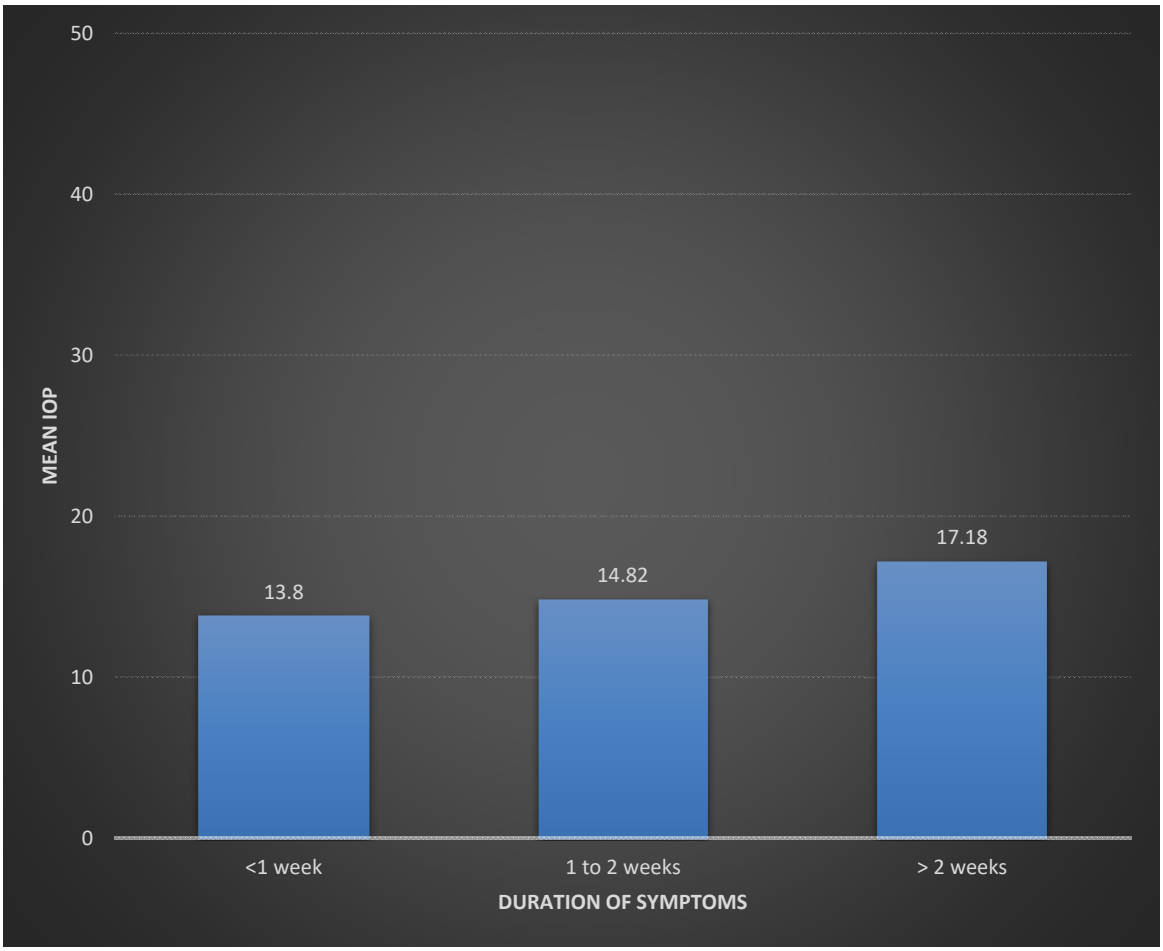

Fig 18: Relationship between duration of symptoms and IOP

Table:17 Bonferroni pairwise comparison test

	<1 week	1 to 2 week
1 to 2 week		
Mean difference	1.02	-
p-value	0.99	
>2 week		
Mean difference	3.38	2.36
p-value	**0.02**	0.31

Table 17 shows the Bonferroni Pairwise comparison. It states that there is a significant difference in IOP in patient presenting between <1 week and >2 weeks duration of symptoms (Mean difference = 3.38, p-value = 0.02).

Table:18 Comparison of Preoperative and postoperative visual acuity (VA)

Visual acuity	n	Median (Snellen's equivalent)	IQR	P-value [a]
Baseline	101	2.60 (HM)	2.60 – 2.90	-
1 month	101	0.30 (6/12)	0.18 – 0.48	<0.001
3 month	88	0.18 (6/9)	0.18 – 0.30	<0.001

a – Wilcoxon sign rank test, IQR – Interquartile range

Wilcoxon sign rank (Non – Parametric) test was used to find out the significant difference between pre-op and post-op visual acuity. P-value less than 0.05 was considered as statistically significant. The p-value shows that there is a significant improvement in VA between baseline and 1month (p-value <0.001), 3month (p-value <0.001).

Table:19 Relationship between duration of symptoms and postoperative BCVA

Duration of symptoms	BCVA (3rd month)				P-value[d]
	6/6 to 6/18	6/24 to 6/36	<6/36	Total	
<1 week	46 (93.9)	3 (6.1)	0	49	<0.001
1 to 2 weeks	18 (81.8)	3 (13.6)	1 (4.5)	22	
>2 weeks	10 (58.8)	0	7 (41.2)	17	
Total	74	6	8	**88**	-

d – Fisher's exact test

Fisher's exact test was used to find out the association between 3rd month BCVA and duration of symptoms. Our study revealed that when our patients presented to hospital within one week of onset of symptoms, there was a significant improvement in vision post – operatively, as compared to those patients who presented after 2 weeks. The p-value (<0.001) shows that there is an association between the duration of symptoms and BCVA.

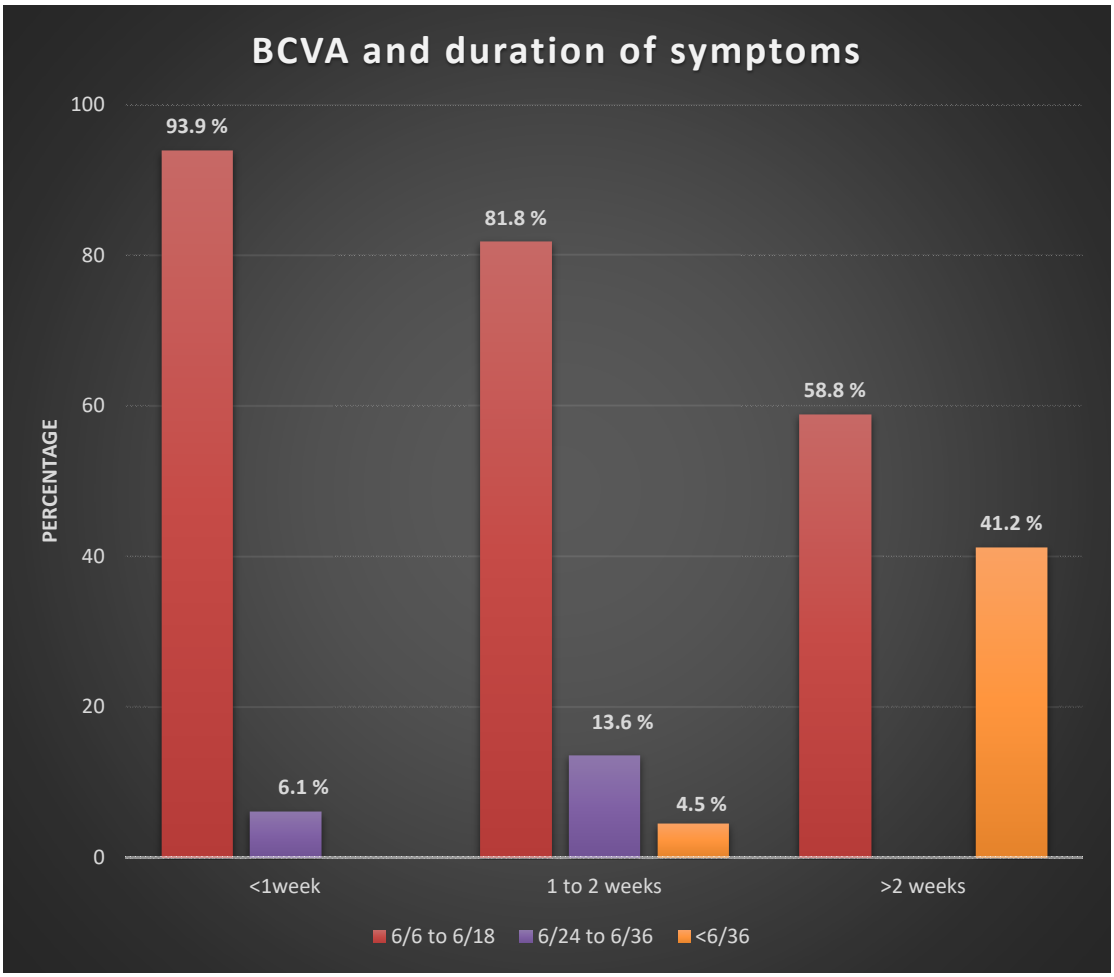

Fig 19: Postoperative BCVA compared with the duration of symptoms

Table:20 Tabulation of BCVA at various follow up visit

BCVA	Baseline n (%)	1 month n (%)	3 month n (%)
6/6 to 6/18	1 (1.0)	78 (77.2)	74 (84.1)
6/24 to 6/36	-	11 (10.9)	6 (6.8)
<6/36	100 (99.0)	12 (11.9)	8 (9.1)
Total	**101**	**101**	**88**

Table 20 shows the distribution of BCVA at various follow up visits. When compared to the baseline data the BCVA has significantly improved postoperatively. 84.1% of patients achieved BCVA > 6/18 at 3 months post – operatively.

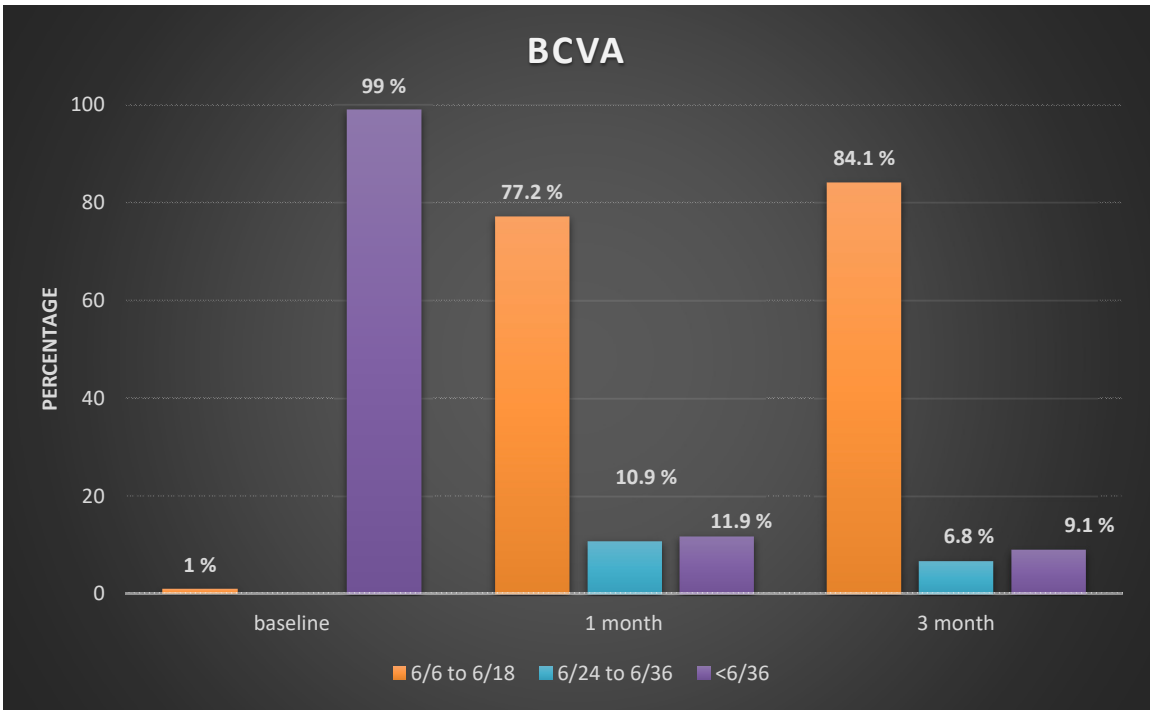

Fig 20: comparison of BCVA during various follow ups

Table:21 Procedure done

Procedure done	n	%
SICS -Aphakia	7	7.0
SICS with PCIOL	91	90.0
IFIOL	1	1.0
SFIOL	2	2.0
Total	**101**	**100**

90% of patients underwent Manual SICS with PCIOL implantation whereas 7% of patients underwent lens extraction. 1% of patient underwent Iris Fixated Intra Ocular Lens Implantation (IFIOL) and 2% of patient underwent Scleral Fixated Intra Ocular Lens Implantation (SFIOL).

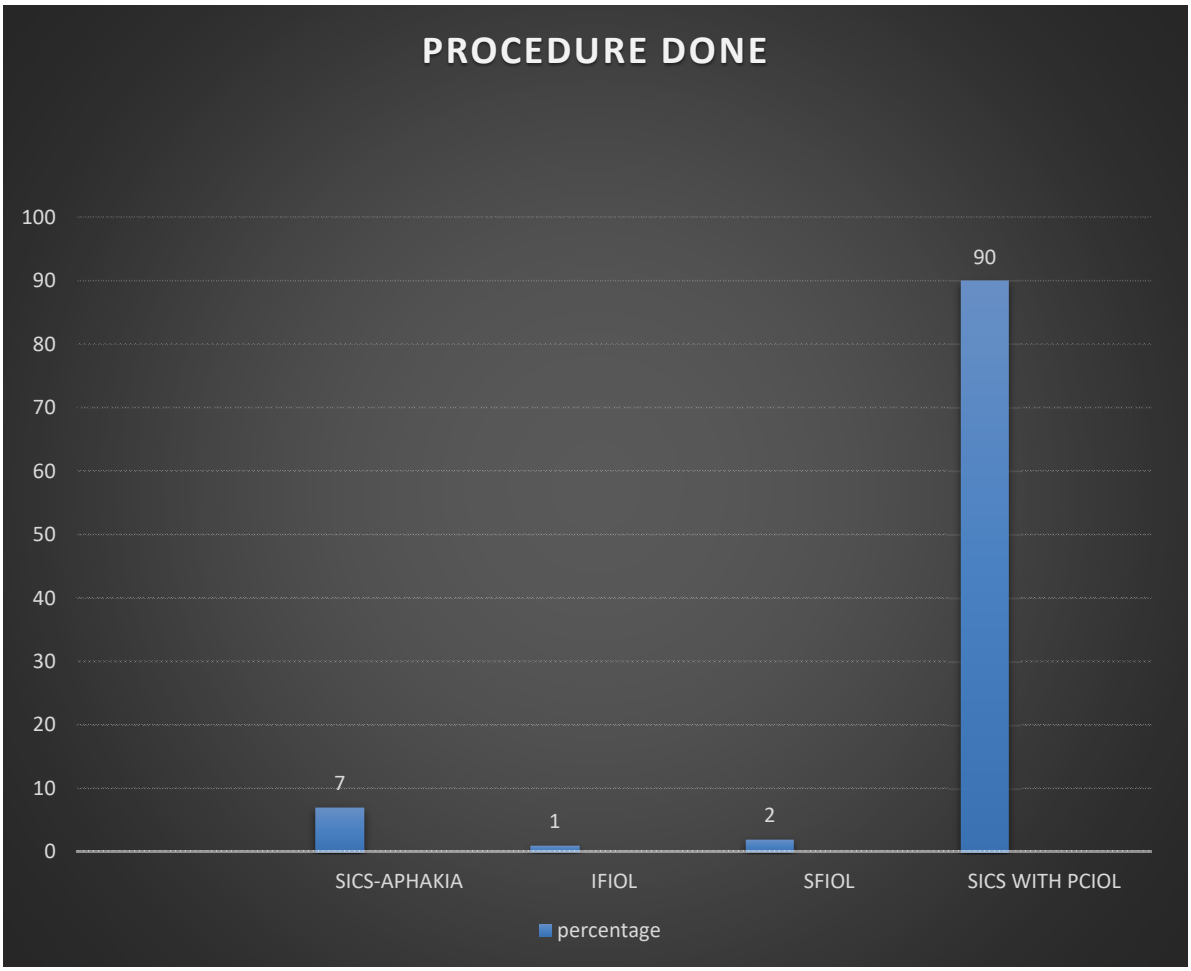

Fig 21: Procedures done for LIG

Table:22 Intraoperative Complications

Intra-op Complications

	n	%
PCR with Vitreous Disturbance (VD)	5	4.9
Zonular dialysis with VD	5	4.9
Whole bag removal/ Aphakia	5	4.9

Among 101 patients who were operated it was found that 4.9% of patients had PCR with VD, 4.9% of patient had Zonular dialysis with VD and 4.9% had whole bag removal with Aphakia.

Table:23 Additional procedure

Additional procedure	n	%
CTR used	3	3.0
Automated vitrectomy	12	11.9
IFIOL	1	1.0
PPV + SFIOL	2	2.0

Additional procedures were done in 18 patients. 11.9% of the total who had additional procedure underwent automated vitrectomy.

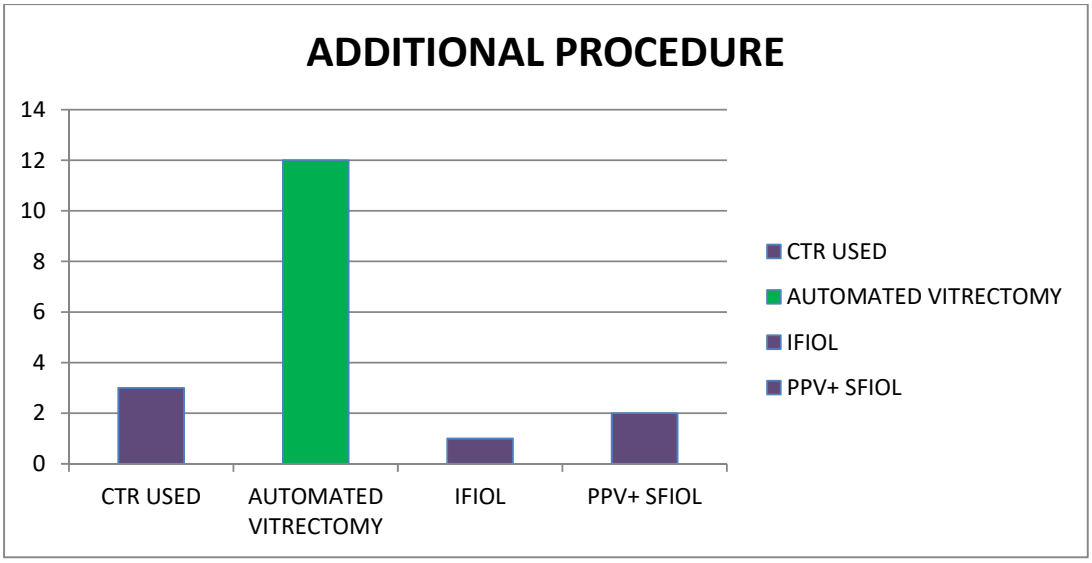

Fig 22: Additional procedures

Table:24 Association between Complications and duration of symptoms

Duration of symptoms	Complications 1 month		Complications 3 month	
	Yes **n (%)**	**No** **n (%)**	**Yes** **n (%)**	**No** **n (%)**
<1 week	11 (39.3)	42 (58.3)	6 (33.3)	43 (61.4)
1 to 2 weeks	5 (17.9)	21 (29.2)	3 (16.7)	19 (27.1)
>2 weeks	12 (42.9)	9 (12.5)	9 (50.0)	8 (11.4)
Total	28	72	18	70
P-value	**0.004** [c]		**0.002** [d]	

c – Chi square test; d – Fisher's exact test

Chi- square test and Fishers exact test was used to measure the association between the durations of symptoms at 1st month and in the 3rd month. When the patients presented to the hospital within 1 week of symptoms the post-operative complications were less compared to the other two groups. At the first month there was a statistical significance *p<0.004* and also at the third month there is a statistical significance *p<0.002.* This states that there is an association between the duration of symptoms and the complications.

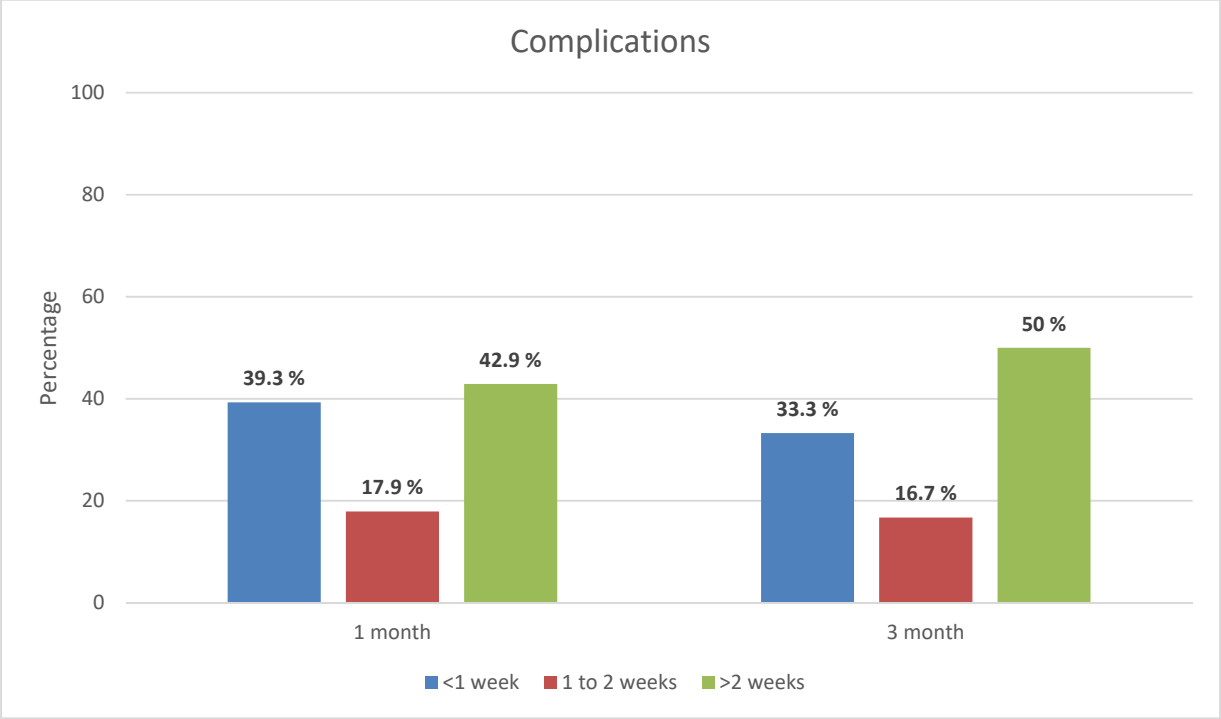

Fig 23: Complications and durations of symptoms

Table:25 Complication frequency table at 1st month and 3rd month

Complications	1st Month	3rd Month
Secondary glaucoma	11(10.9)	10(9.9)
GOA	4(4.0)	4(4.0)
Absolute glaucoma	1(1.0)	1(1.0)
Vitreous in AC	3(3.0)	-
Post op IRITIS	4(4.0)	-
Corticosteroid induced ocular HTN	-	1(3.0)

Few people developed complications at the first and the third month after surgery. At the first month of follow up it was found that among 101 people who were operated, 11 cases developed secondary glaucoma in which 1 case further progressed to absolute glaucoma at the third month of follow up.

Patient with absolute glaucoma who was noted at the first month was lost to follow up at the third month.

4 cases of post op iritis were noted at 1st month, and they were subsequently treated with steroids of which one patient developed steroid induced ocular hypertension and Anti Glaucoma Medication (AGM) was started for this patient.

Table:26 Complication of LIG at final visit (3rd month)

LIG	n	%
Phacomorphic	9	42.9
Phacolytic	4	33.3
Overall	13	39.4

At the 3rd month of follow up, it was found that patients presenting with phacomorphic glaucoma had higher rate of complications as compared to those presenting with Phacolytic glaucoma.

DISCUSSION

LIG are not uncommon in India. This study was done to find out whether a good post-operative visual outcome and IOP control in patients with LIG is obtained using appropriate early medical and surgical management. 101 patients were enrolled for the study, 51 male and 50 female. The mean age group was 64.36 years. 13 patients were lost to follow – up at the third month postoperatively.

The age group in our study ranged from 41 to 82 years with a mean age of 64.36 years and 61% were aged above 60 years. When our study has been compared with the Lahan Study, where the age group of the patients ranged from 40 to 80 years of which 43.1% were aged between 60-69 years. Whereas the study done by Dr.Damodhar Pradhan, et al, in Nepal states that 35% of LIG cases occur in patients' age under 60 years.

In our studyit was found that the incidence of Phacolytic glaucoma was slightly more common (54.5%) than Phacomorphic glaucoma (43.5%). Comparing our study with the Eastern Nepal study, it has been found that the percentage of Phacomorphic (57.5%) Glaucoma is higher than the Phacolytic (42.5%) glaucoma similar to that of the studies done by Pradhan et al., Yaakub A et al, also shows that the incidence of Phacomorphic cases were higher than Phacolytic cases.

In our study it was found that 63.6% of females presented with Phacomorphic glaucoma whereas 60.0% of males were affected by Phacolytic glaucoma. The male is to the female ratio for the LIG is almost equal in this study 50.5: 49.5. In previous studies on LIG it is found that the females (55.45%) outnumbered males and also the incidence of Phacomorphic Glaucoma was found to be higher than the Phacolytic Glaucoma.

51 male and 50 female were enrolled for this study. The mean age group of the patient was 64.36. In our study equal preponderance of males and females has a chance of getting affected by LIG. 50.5% of male and 49.5% of female were affected by LIG. Study done by Yaakub A et al, states that women's have a higher chance of getting LIG than men due to the higher prevalence of cataract in them. The mean age group in the study was found to be 70.2 years.

Our study shows a drastic improvement in visual outcome after surgery and there is a control in the IOP and when compared with the baseline data. There was a good prognosis in the vision when the patients presented to the hospital within 1 week of acute symptoms. Among 101 patients who underwent surgery most of the patients had underwent the procedure SICS with PCIOL. Ramakrishanan R et al, in their study at Tirunelveli, India (March 2006-April 2007) on outcomes of SICS in patients with Phacomorphic glaucoma. MSICS is the appropriate surgical procedure for treatment of advanced cataract in developing world.

The overall result from this study states that the LIG is more common in people presented with advanced cataracts. Delay in presentation more than one week from onset of symptoms is associated with poor visual outcome increased rate of complication and uncontrolled IOP. Both male and female have an equal chance of getting affected by LIG. Early diagnosis and treatment of the disease may result in good prognosis. In most of the patients, who presented to the hospital within one week of acute symptoms had good post-operative outcome than patients who presented lately. The loss of follow up post-operatively was due to the lack of awareness among the people and this still remains as a bottleneck these days.

STRENGTH AND LIMITATION OF THE STUDY

Strength

- This study helped to have an in-depth knowledge about the patients and understand the factors responsible for delayed presentations of patients with LIG

Limitation

- The major limitation of this study was that there was a short follow up period and there were also loss in the follow up post – operatively.
- Long term follow up is needed to see the post-operative outcomes and quality of life for these patients.
- 13 patients were lost to follow up in third month of our study.

CONCLUSION

This study highlights that lack of awareness in the population about early treatment of cataract which is a major detrimental factor in the visual outcome after cataract surgery. The analysis of 101 cases of lens-induced glaucoma who received intraocular lens implants with various procedures has shown that visual acuity has improved after surgery and with the reduction in the IOP. The loss of follow-up after one month postoperatively is a major bottleneck today. Despite the robust services that are provided by the hospital there is a lack of public awareness on the benefits of early detection and treatment of cataract. This is more important in the prevention of LIG. Both the rural and the urban population should be aware of the poor outcome if the treatment is delayed.

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ABBREVATION

LIG	Lens Induced Glaucoma
IOP	Intra Ocular Pressure
RE	Right Eye
LE	Left eye
SICS	Small Incision Cataract Surgery
MSICS	Manual Small Incision Cataract Surgery
BCVA	Best Corrected Visual Acuity
PCR	Posterior Capsular Rent
HM	Hand Movements
LP+	Light Perception
PCIOL	Posterior Chamber Intra Ocular Lens
SFIOL	Scleral- Fixated Intra Ocular Lens
IFIOL	Iris Fixated Intra Ocular Lens
CNS	Central Nervous System
RS	Respiratory System
GIT	Gastro Intestinal Tract

CVS	Cardio Vascular System
ECCE	Extra Capsular Cataract Extraction
GAT	Goldmann Applanation Tonometry
HMW	High Molecular Weight
c AMP	Cyclic Adenosine Mono Phosphate
ALC	Anterior Lens Capsule
BSS	Balance Salt Solution
MIP	Major Intrinsic Protein
AGM	Anti – Glaucoma Medication

CONSENT FORM

Informed Consent form to participate in a clinical trial

Study Title: A prospective observational study to assess the post-surgical outcomes following cataract surgery in patients with Lens Induced Glaucoma.

Protocol Number:

Subject Name: _____ Subject Initials:_____

- Subject ID No:
- Date of Birth/ Age:

		Please put initial
1.	I confirm that I have understood the information about the study, procedures and treatments for the above study and have had the opportunity to ask questions and I have received satisfactory answers to all of my questions. I have been given a copy of the informed consent form to take home.	[ ]
2.	I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving my medical care or legal rights being affected. However this may not be possible for certain surgical procedures.	[ ]

3.	I understand that the Investigator of the study wants to access my health records for research purpose. However I understand that my identity will not be revealed or information released to third parties or published.	[ ]
4.	I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose (s).	[ ]
5.	I agree to take part in the above study.	[ ]

Signature (or Thumb impression) of the Subject:

_____

Date: __/__/____

Subject's Name: _____

_____

Signature (or Thumb impression) of Legally Acceptable Representative (LAR):

_____

Date: __/__/____

Investigator's Name: _____

Signature of the Witness: _____

Date: __/__/___

Name of the Witness:

_____

PROFORMA

A prospective observational study to assess the post-surgical outcomes following cataract surgery in patients with lens induced glaucoma.

Glaucoma Clinic, Aravind Eye Hospital, Madurai

Name: M.R.No:

Age: UID No:

Sex: Study No:

Mobile No:

Vitals: ☐ SBP ☐ DBP

Systemic disease: ☐ DM ☐ Hypertension ☐ Asthma ☐ Cardiac ☐ Others

History: ☐ Defective vision ☐ Redness ☐ Pain ☐ Trauma ☐ Others (if others specify)

Duration of symptoms: ☐ <1 Weeks ☐ 1 to 2 Weeks ☐ >2 Weeks

H/o usage of anti-glaucoma medications: Yes ☐ No ☐

	Right Eye	Left Eye
Laterality		
BCVA		
UCVA		
IOP (mm of Hg) by GAT		

Slit Lamp Examination

		Right	Left
Conjunctiva	[1- Normal, 2- Others] if others specify		
Cornea	[1- Clear, 2-Ohters] if others specify		
Anterior Chamber Depth	[1-Normal, 2-Shallow, 3-Deep]		
Iris	[1- Normal, 2-Atrophic patches, 3- Others] if others specify		
Pupil Shape	[1-Regular, 2-Irregular, 3-Others]If others specify		
Pupil Reaction	[1-Brisk, 2-Sluggish, 3-Fixed, 4-Rapfd]		
Lens Status	[1-Phacomorphic, 2-Phacolytic, 3-Others] if others specify		
Fundus	[1-Assessable, 2 -Not Assessable] if assessable specify details		

Postoperative Examination [1st day]

1.	Wound [1-Well opposed, 2-Wound leak, 3-Iris prolapse]	
2.	Cornea [1-Clear,2-Epithelial edema ,3-Epithelial defect ,4-DM strip, 5-SK,6-DM fold]	
3.	Anterior chamber [1-Normal, 2-Shallow, 3- Deep]	
4.	Ac content [1-Clear, 2-Cortex, 3-Vitreous in AC, 4-Hyphema, 5-Fibrin membrane, 6-Hypopyon]	
5.	Iritis [1-Yes, 2- No]	
6.	Pupil [1-Regular, 2-Irregular, 3-Irido dialysis]	
7.	PCIOL [1-Inbag, 2- Insulcus, 3-Aphakia, 4- Pupillary capture, 5-Decentered, 6- Dislocated, 6-Not assessable	
8.	Pc [1-Intact, 2- Rent , 3-Zonular dialysis, 4- Whole bag removal, 5- Not assessable]	

	Right Eye	Left Eye
UCVA		
BCVA		
IOP (mm of Hg) by GAT		

Post-Operative Follow-up

	1st month	**3rd month**
BCVA		
IOP (mm of Hg) by GAT		

ETHICS COMMITTEE APPROVAL

ARAVIND MEDICAL RESEARCH FOUNDATION
Institutional Ethics Committee
(REGISTRATION NO. ECR/182/INST/TN/2013 DATED 20.04.2013)

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Mrs.Premalatha Panneerselvam M.A., M.Ed

20th December 2017

To
DR. P. PRAVEEN KUMAR
MS Resident
Aravind Eye Hospital
Madurai

Dear Dr. PRAVEEN KUMAR,

Thesis Title: A Prospective Observational Study To Assess The Post-Surgical Outcomes Following Cataract Surgery In Patients With Lens Induced Glaucoma

IEC Code: IEC201800254

Thank you for submitting your thesis and seeking the approval from the ethics committee. The documents provided by you for consideration which include the thesis protocol and informed consent forms were reviewed for the research methodology and scientific content. The Ethical committee did not find any correction and has recommended the thesis to go ahead in the present form.

Thanking you

Yours Sincerely,

Dr.R.Sharmila
Member Secretary
Institutional Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
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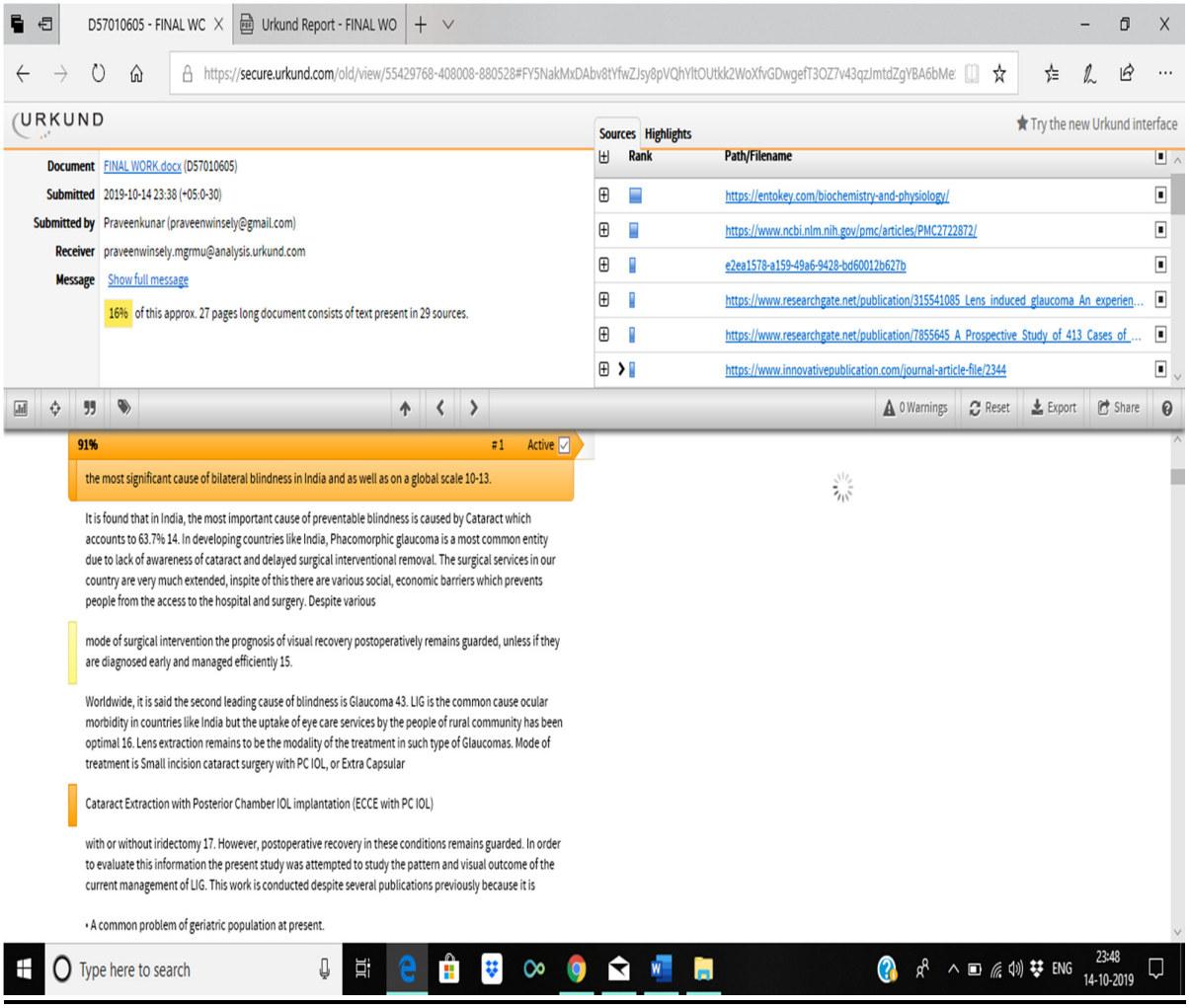
PLAGIARISM REPORT



URKUND

Urkund Analysis Result

Analysed Document: FINAL WORK.docx (D57010605)
Submitted: 10/14/2019 8:08:00 PM
Submitted By: praveenwinsely@gmail.com
Significance: 16 %

Sources included in the report:

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CERTIFICATE - II

This is to certify that this dissertation work titled **"A Prospective Observational study to assess the post- surgical outcomes following Cataract Surgery in patients with Lens Induced Glaucoma"** of the candidate **Dr. Praveen Kumar P** with registration Number **221713456** for the award of **M.S Ophthalmology** in the **branch of III**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 16% percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

MASTER CHART

AFFECTED_EYE	BCVA_PREOP	UCVA_PREOP	IOP_PREOP	CONJUCTIVA	CONJUCTIVA_SPECIFY	CORNEA	CORNEA_SPECIFY	ANT-CHAMBER_DEPTH	VH GRADING	FLARE_GRADE	CELL_GRADE	LENS_PARTICLE	IRIS	IRIS_SPECIFY	PUPIL_SHAPE	PUPIL_SHAPE_SPECIFY	PUPIL_REACTION	PUPIL_REACTION_SPECIFY	LENS_STATUS
1	HM	6/24	48	2	circumcorneal congestion	2	epithelial edema	1	vh- 3	2+	1+	1	1		1		3		2
1	LP+	LP+	40	2	circumcorneal congestion	2	epithelial edema	1	vh-3			1	1		1		3		2
1	LP+	LP+	56	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
1	HM	HM	26	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		2		1
1	HM	HM	36	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		3		1
2	HM	HM	28	2	circumcorneal congestion	2	corneal edema , pigments on endothelium	2	vh-2			0	1		1		3		1
2	HM	HM	48	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
1	HM	HM	34	1		2	kps at endothelium	1	vh-3	1+	1+	1	1		3	posterior synechiae	2		2
2	LP+	LP+	40	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		2
1	FCF	FCF	28	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		3		2
2	1/2 60	1/2 60	48	2	circumcorneal congestion	2	epithelial edema	2	vh-3			0	1		1		3		1
2	LP+	LP+	44	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
2	HM	HM	50	2	circumcorneal congestion	2	epithelial edema ,endothelial dusting	2	vh-1			0	1		1		3		1
2	LP+	LP+	26	2	circumcorneal congestion	1		1	vh-3			0	1		2		2		2
2	HM	HM	58	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
2	LP+	LP+	38	2	circumcorneal congestion	2	epithelilal edema ,stromal edema	1	vh-3	1+		1	3	no niew	3	no view	5	no view	2
2	HM	HM	50	1	circumcorneal congestion	1		2	vh-2			0	1		1		3		1
2	LP+	LP+	30	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		2		1
1	HM	HM	26	2	circumcorneal congestion	2	epithelial edema	1	vh-3	1+		1	1		1		2		2
2	HM	HM	42	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
1	HM	HM	48	2	circumcorneal congestion	1		1	vh-3			0	1		1		2		2
2	LP+	LP+	30	1		2	epithelial edema	1	vh-3	1+	1+	1	3	no niew	3	no view	3	no view	2
1	HM	HM	56	1		2	epithelila edema , bullae	1	vh-3	2+	1+	1	1		1		2		2
2	HM	HM	40	2	circumcorneal congestion	2	epithelial edema	1	vh-3			1	1		1		1		2
1	LP+	LP+	48	2	circumcorneal congestion	2	epithelial edema	2	vh-2	2+	1+	0	1		1		3		1
2	HM	HM	40	2	circumcorneal congestion	2	epithelial edema	1	vh-3			1	1		1		1		2
1	LP+	LP+	48	2	circumcorneal congestion	2	epithelial edema	2	vh-2	2+	1+	0	1		1		3		1
2	HM	HM	48	2	circumcorneal congestion	2	epithelial edema ,stromal edema	1	vh-3		2+	1	1		1		3		2
2	LP+	LP+	42	2	circumcorneal congestion	2	epithelial edema ,bullae	1	vh-3	1+		1	1		1		2		2
2	HM	HM	68	2	circumcorneal congestion	2	epithelial edema	1	vh-3	1+		0	1		1		2		2
1	HM	HM	70	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
2	HM	HM	14	2	circumcorneal congestion	1		2	vh-2			0	1		1		1		1
1	LP+	LP+	30	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		2		1
2	LP+	LP+	40	2	circumcorneal congestion	2	epithelial edema , lens cortex adherent to endothelium	1	vh-3	2+	4+	1	1		1		3		2
1	HM	HM	23	2	circumcorneal congestion	1		2	vh-2			0	1		1		2		1
1	LP+	LP+	38	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
1	HM	HM	52	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
1	LP+	LP+	50	2	circumcorneal congestion	2	epithelial edema	1	vh-3		2+	1	1		1		3		2
1	LP+	LP+	46	1		2	epithelial edema	2	vh-2			1	1		1		2		2
1	HM	HM	56	2	circumcorneal congestion	1		1	vh-3			1	1		1		3		2
2	6/18	6/18	24	2	circumcorneal congestion	2	epithelial edema	1	vh-3			1	1		1		1		2
1	HM	HM	38	1		2	inflammatory deposits on endothelium	1	vh-3			0	1		1		1		2
1	HM	HM	38	2	diffuse cingestion	2	stromal haze	1	vh-3			1	1		1		3		2
1	HM	HM	20	2	circumcorneal congestion	2	stromal haze	1	vh-3	2+	3+	1	1		1		3		2
1	HM	HM	32	1		2	epithelial edema	2	vh-2			0	1		1		3		1
2	LP+	LP+	20	2	circumcorneal congestion	1	epithelial edema	1	vh-3			1	1		1		3		2
1	HM	HM	56	1	circumcorneal congestion	2	epithelial edema	1	vh-3	3+		0	1		1		3		2
2	HM	HM	48	2	circumcorneal congestion	2	epithelial edema	1	vh-3			0	1		1		1		2
1	HM	HM	34	2	circumcorneal congestion	2	pigments on endothelium	1	vh-3		1+	0	1		3	posterior synechiae	2		2
2	LP+	LP+	50	2	circumcorneal congestion	2	microcytic edema , stromal bullae , pigments on endothelium	2	vh-2			0	1		1		3		1
1	HM	HM	20	1		2	epithelial edema	1	vh-3			0	1		1		3		2

1	HM	HM	36	1	circumcorneal congestion	2	epithelial edema	1	vh-3			1	1		1		2		2
1	HM	HM	70	2	circumcorneal congestion	2	epithelial edema	1	vh-3			0	1		1		3		2
1	LP+	LP+	46	2	circumcorneal congestion	2	epithelial edema,bullae, lens particles on endothelium	1	vh-3	2+		1	1		1		3		2
1	HM	HM	48	2	circumcorneal congestion	1	epithelial edema	3	vh-4			0	1		1		3		2
2	LP+	LP+	24	2	circumcorneal congestion	2	epithelial edema	1	vh-3			1	1		1		3		2
2	HM	HM	40	2	circumcorneal congestion	2	epithelial edema	3	vh-3		2+	1	1		1		3		2
2	HM	HM	30	2	circumcorneal congestion	2	epithelial edema	1	vh-3		3+	1	1		1		2		2
1	2/60	2/60	6	2	circumcorneal congestion	2	Descemets fold	1	Vh-3			1	1		1		2		3
2	FCF	FCF	30	2	circumcorneal congestion	2	epithelial edema	1	vh-3	1+	1+	1	1		1		2		2
2	LP+	LP+	42	2	circumcorneal congestion	2	epithelial edema	1	vh-3	1+	1+	0	1		1		1		2
2	FCF	FCF	14	2	circumcorneal congestion	2	epithelial edema , bullae, descemets folds	3	vh-4			1	1		1		1		3
2	FCF	FCF	42	2	circumcorneal congestion	2	epithelial edema	3	vh-3		2+	1	1		1		3		2
2	HM	HM	32	2	circumcorneal congestion	1		1	vh-3	2+	1+	0	1		1		1		2
2	LP+	LP+	44	2	circumcorneal congestion	2	epithelial edema	1	vh-3			0	1		1		3		2
1	HM	HM	44	2	circumcorneal congestion	2	epithelial edema	1	vh-3			0	1		1		3		2
1	LP+	LP+	32	2	circumcorneal congestion	2	epithelial edema	3	vh-3			1	1		1		1		2
1	LP+	LP+	42	2	circumcorneal congestion	2	epithelial edema	3	vh-3	2+	1+	0	1		1		3		2
2	LP+	LP+	38	2	circumcorneal congestion	2	epithelial edema	1	vh-3			0	1		1		2		2
1	LP+	LP+	32	1		1		2	vh-2	2+	2+	0	1		1		2		2
2	LP+	LP+	14	2	circumcorneal congestion	1		2	vh-2			0	1		1		2		2
2	LP+	LP+	28	2	circumcorneal congestion	2	epithelial edema	1	vh-4	1+		0	1		1		2		2
2	HM	HM	42	2	circumcorneal congestion	2	epithelial edema	1	vh-3	2+	1+	0	1		1		3		2
2	LP+	LP+	52	2	circumcorneal congestion	2	epithelilal edema	1	vh-3			1	1		1		3		2
1	LP+	LP+	54	2	circumcorneal congestion	2	epithelial edema	1	vh-3			1	1		1		1		2
1	LP+	LP+	50	2	circumcorneal congestion	2	epithelial edema	1	vh-3	1+	1+	0	1		1		2		2
2	HM	HM	48	2	circumcorneal congestion	2	epithelial edema	1	vh-4	1+	1+	0	1		1		2		2
1	HM	HM	44	2	circumcorneal congestion	2	epithelial edema	1	vh-3		1+	0	1		1		2		2
1	HM	HM	40	2	circumcorneal congestion	1		1	VH-3		1+	0	1		1		2		2
1	LP+	LP+	40	2	circumcorneal congestion	1	epithelial edema , lens particles touches endothelium	1	vh-3			1	1		1		2		2
1	LP+	LP+	48	2	circumcorneal congestion	2	epithelial edema	1	vh-3			0	1		1		3		1
1	HM	HM	44	2	circumcorneal congestion	2	epithelila edema	2	vh-2			0	1		1		2		1
1	HM	HM	50	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		3		1
1	LP+	LP+	30	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		3		1
1	HM	HM	38	1		2	epithelial edema	2	vh-1			0	1		1		1		1
1	LP+	LP+	56	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		2		1
1	LP+	LP+	60	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		2		1
1	HM	HM	42	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
1	HM	HM	36	2	circumcorneal congestion	1		2	vh-2			0	1		1		3		1
1	HM	HM	46	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		2		1
2	LP+	LP+	56	2	circumcorneal congestion	1		2	vh-2			0	1		1		2		1
1	HM	HM	20	2	circumcorneal congestion	1		2	vh-2			0	1		1		1		1
1	LP+	LP+	38	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
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1	HM	HM	38	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		2		1
1	HM	HM	14	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		3		1
1	HM	HM	24	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		2		1
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2	HM	HM	42	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		3		1
1	HM	HM	34	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		3		1
0	HM	HM	64	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		2		1
2	HM	HM	60	2	circumcorneal congestion	2	epithelial edema	2	vh-1			0	1		1		3		1
2	LP+	LP+	32	2	circumcorneal congestion	2	epithelial edema	2	vh-2			0	1		1		2		1