A CLINICAL STUDY OF POSTERIOR CAPSULAR OPACIFICATION AND ITS VISUAL OUTCOME AFTER Nd: YAG LASER CAPSULOTOMY

By

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ABBREVIATIONS

BAB	:-	Blood Aqueous Barrier
BCVA	: -	Best Corrected Visual Acuity
CF	:-	Counting Fingers
CME	:-	Cystoid Macular Edema
D	: -	Diopter
ECCE	: -	Extra Capsular Catract Extraction
HM	:-	Hand Movements
IOL	: -	Intra Ocular Lens
IOP	: -	Intra Ocular Pressure
LEC	: -	Lens Epithelial Cells
mjs	:-	Milli Joule Second
Nd:YAG	:-	Neodymium : Yttrium Aluminum Garnet
OPD	: -	Out Patient Department
PCO	: -	Posterior Capsular Opacification/ Posterior Capsular Opacity
PCIOL	: -	Posterior Chamber Intra Ocular Lens
PoIVS	: -	Posterior Pole Visualization Score
PL	:-	Perception Of Light
PMMA	: -	Poly Methyl Methacrylate
RD	:-	Retinal Detachment
SCI	:-	Sealed Capsular Irrigation
µ	:-	Micron

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INTRODUCTION

Posterior capsular opacification is most common delayed complication following extra capsular cataract surgery and intraocular lens implantation[1].

Lens epithelial cells left behind in the capsular bag after extra capsular cataract surgery are mainly responsible for PCO development[2]. Clinically there are three morphological types of PCO- Fibrous, Pearl & Combined type[2]. Fibrous type of PCO is caused by proliferation & migration of LEC, which undergo epithelial to mesenchymal transition, resulting in fibrous metaplasia & leading to significant visual loss. Pearl type PCO is caused by LEC located at equatorial lens region, which following regeneration of lenticular fibers, form Elschnig pearls & Soemmering rings, responsible for most cases of PCO- related visual loss[2].

Young age is a significant risk factor for PCO, and its occurrence is a virtual certainty in pediatric patients. It remains the most common complication of cataract surgery.

Incidence of PCO ranges between 25% to 50% in adults[2]. PCO is a major problem following pediatric cataract surgery, and where the incidence is 100%.

Diabetic patients have significantly severe PCO, when compared with non-diabetic patients after one year of follow up[2].

The incidence of PCO is also high in eyes with uveitis and other ocular diseases like myotonic dystrophy and retinitis pigmentosa[2].

Eyes with hydrophobic acrylic IOLs have shown to provide a better visual outcome and lower incidence of PCO than silicone, PMMA or heparin surface modified PMMA IOLs[2].

In traumatic cataracts, the incidence of PCO is significantly higher and has been quoted to be as high as 92% at three year follow-up[2].

Any defect in the blood–aqueous barrier (BAB), possibly due to intraocular inflammation, preoperative high intraocular pressure (IOP), or excessive eye manipulation during surgery may lead to a disturbance in coagulation and fibrinolytic pathway, causing increased postoperative inflammation proposed as the cause of increased incidence of PCO in children[3].

Nd: YAG laser is the treatment of choice for the posterior capsular opacity. Visually significant PCO is usually managed by creating an opening in the opaque capsule using the Nd: YAG laser[4]. A surgical posterior capsulotomy may be indicated in children for dense PCO associated with secondary membrane formation.

Nd:YAG laser posterior capsulotomy is known to be associated with complications such as transient rise in IOP, enhanced risk of retinal detachment particularly marked in axial myopia, cystoid macular edema, IOL subluxation, lens optic damage/pitting, endophthalmitis, vitreous prolapse into the anterior chamber and anterior hyaloid disruption[2]. The current study is aimed to study the various clinical presentation, types and management of PCO and visual outcome and complications after Nd: YAG laser capsulotomy .

AIMS AND OBJECTIVES OF STUDY

To study the types of posterior capsular opacification, visual outcome and complication after Nd: YAG laser capsulotomy.

REVIEW OF LITERATURE

Posterior capsule opacification is a postsurgical complication following extra capsular cataract surgery with intraocular lens implantation. It is caused by lens epithelium cells proliferation and migration[1-4]. In addition to classic posterior capsule opacification, postoperative lens epithelial cell proliferation is involved in the pathogenesis of anterior capsule opacification and inter-lenticular opacification[1-4].

INCIDENCE

Clinical studies have noted that incidence of Posterior capsule opacification is 50% in adults and 100% in younger age group following Extra capsular cataract extraction[5].

LENS[6]

The adult human lens is a crystalline, avascular, biconvex colorless structure 4 mm thick and 9 mm in diameter. It lies posterior to the iris and anterior to vitreous body, suspended in position by the Zonules of Zinn.

Embryology[6-7]

The formation of the human crystalline lens begins at about 25th day of gestation. Two lateral outpouching called the optic vesicles form from the forebrain (diencephalon). The optic vesicles enlarge and become closely apposed to the surface ectoderm. The single layers of cuboidal cells of the surface ectoderm that overlie the optic vesicles become columnar and thickened at about 27 days of gestation forming the lens plate, or lens placode. The lens pit or fovea lentis appears at 29 days of gestation as a small indentation inferior to the center of the lens plate. The lens pit deepens by a process of cellular multiplication and invaginates. The resultant sphere with a single layer of cuboidal cells encased within a basement membrane (the lens

capsule), is called the lens vesicle. It is 0.2 mm in diameter at 33 days of gestation. The posterior cells of the lens vesicle become columnar and elongate to form primary lens fibers which progressively obliterate the lumen of the lens vesicle. The primary lens fibers make up the embryonic nucleus that will occupy the central area of the lens in adult life.

This monolayer of cuboidal cells is called as the lens epithelium. Subsequent differentiation and growth of the lens originates from the lens epithelium. At 7 weeks of gestation, the equatorial lens epithelial cells multiply rapidly and elongate to form secondary lens fibers. The anterior aspect of each developing lens fiber grows anteriorly toward the anterior pole of the lens. The posterior aspect of each developing lens fiber grows posteriorly toward the posterior pole of the lens, just inside the lens capsule. In this manner, new lens fibers are continually formed, layer upon layer. The secondary lens fibers formed between 2 and 8 months of gestation make up the fetal nucleus.

As lens fibers grow anteriorly and posteriorly, a pattern emerges where the fibers meet and interdigitate in the anterior and posterior portions of the lens. These patterns are known as sutures. Y-shaped sutures are recognizable at about 8 weeks of gestation. As the lens fibers continue to form and the lens continue to grow, the pattern of lens sutures becomes increasingly complex.

At birth, the human lens weighs approximately 90 mg, and it increases in mass at the rate of about 2 mg per year as new fibers form. After 20 years, the central, or oldest, lens fibers become less malleable and the lens nucleus becomes more rigid. After 40 years, the rigidity of the lens nucleus clinically reduces accommodation, and by age 60 nuclear sclerosis or discoloration often makes the lens sutures difficult to distinguish.

Anatomy – Histology[6-7]

The lens is a transparent, biconvex, elliptical, semisolid, avascular body of crystalline appearance located between the iris and the vitreous. Laterally, the equatorial zone of the lens projects into the posterior chamber and is attached by the zonules to the ciliary epithelium. The lens is unique among organs in that it contains cells solely of a single type, in various stages of cytodifferentiation, and retains within it all the cells formed during its lifetime. The oldest cells are contained within the core of the lens. New cells are added superficially to the cortex in a series of concentric layers throughout life.

As the cells become older and more embedded within the lens they undergo several changes, losing organelles, and to some extent their structural integrity, and become progressively more inert metabolically.

Lens dimensions[6-7]

The equatorial diameter is 9-10 mm in the adults. The lens has two surfaces, anterior and posterior, and a border where these surfaces meet is known as the equator. The anterior surface is less convex with radius of curvature of about 10 mm (8.0-14.0 mm). The centre of the anterior surface is known as the anterior pole, and is about 3 mm from the back of the cornea.

The posterior surface is more curved with a radius of curvature of about 6 mm (4.5-7.5 mm). The equator of the lens forms a circle lying 0.5 mm within the ciliary processes. It shows a number of dentations corresponding to the attachment of the zonular fibres.The refractive index of the lens is 1.39.The dioptric power is about 18D.

Structure[6-7]

The lens consists of

1) The lens capsule
2) The lens epithelium
3) The lens Substance

The lens capsule

The capsule is the basement membrane of the lens epithelium which completely envelops the lens and is the thickest basement membrane in the body. The thickness of the capsule depends upon the region of the capsule being measured and the age of the individual (thickness increases with age).

- It is thickest close to the equator on both the anterior and the posterior surfaces which is about 23μ.
- Intermediate thickness at the equator (I7μ) and at the anterior pole (9- 14μ).
- It is thinnest at the posterior pole (4μ).

The capsule is produced anteriorly by the lens epithelium and posteriorly by the elongating fiber cells. The capsule receives the insertion of the zonular fibers anteriorly and posteriorly at the lens periphery as well as at the lens equator.

Under the light microscope the capsule appears transparent and homogeneous. It shows birefringence under polarized light, with an indication of a lamellar structure with fibers arranged parallel to its surface. Its shows a positive reaction to Periodic acid—Schiff reagent which stains the glycoprotein matrix.

Under the electron microscope, the capsule appears to have a relatively amorphous appearance in which a lamellar structure is suggested by coarse scattered

filamentous elements. There are up to 40 lamellae, each of which is about 40 nm thick. At higher resolution fine fibrils may be identified which are about 2.5 nm in diameter. The lamellae run parallel to the capsular surface.

In the anterior and equatorial capsule there are electron dense inclusions which consist of collagen fibrils 15 nm in diameter and with periodicity of 50-60 nm. These are attributed to epithelial activity.

The capsule is rich in type IV collagen. It also contains types I and III collagen, in addition to a number of extracellular matrix components, which include laminin, fibronectin, heparin sulphate, proteoglycan and entactin. The capsule is freely permeable to water, ions and other small molecules, and acts as a barrier to protein molecules of the size of albumin.

The lens epithelium

The epithelium consists of a single sheet of cuboidal cells spread over the front of the lens. There are about 5,00,000 cells in the mature lens with a central density of about 5009/mm^2 in men, and 5781/mm^2 in women and an increased density towards the periphery. Cell density declines with age. There is no corresponding posterior layer because the posterior epithelium of the embryonic lens is involved in the formation of the primary lens fibers which come to occupy the centre of the lens nucleus. Epithelial cells can be divided in to three zones:-

1. **Central zone (A - Cells)**
2. **Intermediate zone**
3. **Germinative zone (E - Cells)**
4. **The Lens Substance**[6-7]

The lens substance which constitutes the main mass of the lens, is composed of densely packed fibers with very little extracellular space. The adult lens substance consists of the nucleus and the cortex. The nucleus accounts for approximately 84% of the diameter and thickness of the lens and the cortex the remaining 16%.

The nucleus is further subdivided into embryonic, fetal, infantile and adult nuclei. The embryonic nucleus contains the original primary lens fiber cells that are formed in the lens vesicle. The rest of the nuclei are composed of secondary fibers, which are added concentrically at different stages of growth by encircling the previously formed nucleus. The fetal nucleus contains the embryonic nucleus and all fibers added to the lens before birth. The embryonic and fetal nuclei, together with all the fibers added until 4 years of age, compose the infantile nucleus. The adult nucleus is composed of all fibers added before sexual maturation.

The cortex, which is located peripherally, is composed of all the secondary fibers continuously formed after sexual maturation and can be divided into the deep, intermediate, and superficial cortex. The region between the hardened embryonic and fetal nuclear core and the soft cortex sometimes is referred to as the epinucleus. The region between the deep cortex and adult nucleus is sometimes referred to as the perinuclear region.

PATHOGENESIS[5, 8]

In a normal lens, the LECs are confined to the anterior surface at the equatorial region and the equatorial lens bow. This single row of cuboidal cells can be divided into two different biological zones.

The anterior-central zone (corresponding to the zone of the anterior capsule) consists of a monolayer of flat cuboidal, epithelial cells with minimal mitotic activity.

In response to a variety of stimuli, the anterior epithelial cells ("A" cells) proliferate and undergo fibrous metaplasia. This has been termed "pseudofibrous metaplasia" by Font and Brownstein.

The second zone is important in the pathogenesis of "pearl" formation. This layer is a continuation of anterior lens cells around the equator, forming the equatorial lens bow ("E" cells). Unlike within the A-cell layer, cell mitosis, division, and multiplication are quite active in this region. New lens fibers are continuously produced in this zone throughout life.

In addition to classic PCO, postoperative LEC proliferation is also involved in the pathogenesis of other entities, such as anterior capsule opacification/fibrosis, a more recently described complication related to piggyback IOLs. Thus, there are three distinct anatomic locations within the capsular bag where clinically significant opacification may occur postoperatively.

Although both types of cells (from the anterior central zone and from the equatorial lens bow) have the potential to produce visually significant opacification, most cases of classic PCO are caused by proliferation of the equatorial cells. The term posterior capsule opacification is actually a misnomer, it is not the capsule which opacifies rather, an opaque membrane develops as retained cells proliferate and migrate onto the posterior capsular surface.

MORPHOLOGICAL FORMS OF POSTERIOR CAPSULE OPACIFICATION[5, 9]

1) Fibrosis-Type Posterior Capsule Opacification.
2) Pearl-Type Posterior Capsule Opacification.

Fibrosis-Type Posterior Capsule Opacification:-

Residual lens epithelial cells that are attached to the anterior capsule after ECCE are the predominant cells involved in the formation of fibrous membranes. Although cases of fibrosis tend to appear within 2-6 months of ECCE, many are clinically insignificant.

Remnant epithelial cells on the anterior capsule differentiate into spindle-shaped, fibroblast-like cells (myofibroblasts), which express smooth muscle actin and become highly contractile. These fibroblastic cells proliferate and migrate onto the posterior capsule to form a cellular layer that secretes extracellular matrix components and a basal lamina-like material. Cellular contraction results in the formation of numerous fine folds and wrinkles in the posterior capsule. At this stage the capsule is only mildly opacified. No significant visual loss occurs until the cells migrate into the visual axis.

More advanced stages of PCO result from further proliferation and multilayering of cells on the posterior capsule, and are associated with additional extracellular matrix production and the appearance of white fibrotic opacities. These cellular membranes increase the extent of capsular wrinkling which, in turn further increases visual distortion. This capsular contraction can cause further complications, such as decentration of the intraocular lens (IOL). In some cases the cells partially or completely degrade and leave the extracellular matrix components.

The extracellular matrix produced in the fibrosis-type of PCO is composed of types I and III fibrillar collagen with associated proteoglycans.

Differentiation of lens epithelial cells into fibroblast-like cells can also cause opacification of the anterior capsule. Although technically this is another form of

secondary cataract, it often is of no clinical significance because the opacification normally is away from the visual axis. In some cases of anterior capsular fibrosis the anterior capsule contracts centrifugally, which enlarges the capsular opening and may leave a sheet of cells attached to the IOL optic. Contraction of the anterior capsule, however, normally is centripetal, which shrinks the anterior capsular opening (capsule contraction syndrome). This reduction in size of the opening occurs more commonly in patients who have received a capsulorrhexis than in those who have undergone a "can-opener" capsulotomy. Vision is reduced only if the anterior capsular flap obscures the visual axis, but this can be corrected within a few weeks of the cataract surgery by making small radial incisions in the anterior capsule.

In cases where the cut edge of the anterior capsule rests on the IOL optic, residual anterior capsular cells may proliferate and extend from this cut edge onto the surface of the IOL, which results in the formation of a membranous outgrowth within approximately 1 week postoperatively. It is also possible that cells may migrate around onto the posterior surface of the IOL implant and therefore, contribute to the formation of PCO.

Growth factors present in both the aqueous and the vitreous humor have been implicated in the development of fibrous- type PCO. These include acidic and basic fibroblast growth factors, insulin-like growth factor-l, epidermal growth factor, platelet-derived growth factor, hepatocyte growth factor, and transforming growth factor - β.

Pearl-Type Posterior Capsule Opacification:

The pearls formed in this type of PCO are identical in appearance to Wedl (bladder) cells involved in the formation of posterior subcapsular cataract. Because Wedl cells are known to originate from equatorial lens epithelial cells, it is believed that residual cells in this region of the capsule are the predominant cells involved in the formation of pearls. Clinically, cases of pearl formation occur somewhat later than those of fibrosis (up to 5 years postoperatively).

Pearls were first observed by Hirschberg in 1901 and then by Elschnig in 1911 they now are referred to as Elschnig's pearls. Newly formed lens fibers therefore, are no longer forced in the anterior and posterior directions which results in the formation of a mass of cells (normally large and globular, but sometimes spindle shaped) loosely connected and piled on top of each other. The diameters of these cells are in the range 5—120μm. Each pearl represents the aberrant attempt of one epithelial cell to differentiate into a new lens fiber, possessing characteristics of both epithelial cells and fibers, and may be embedded in an extracellular matrix.

The fibrous granular cytoplasm and the possession of very few or no organelles are properties of lens fibers, but the pearls still have interdigitating processes and desmosomes between adjacent cells, which are traits of the original epithelial cells.

Soemmerring's Ring

Soemmerring first noticed PCO in humans in 1828. After ECCE, the cut edge of the remaining anterior capsular flap may attach itself to the posterior capsule within approximately 4 weeks postoperatively, through the production of fibrous tissue. Any residual cortical fibers and epithelial cells therefore, are trapped within this sealed

structure. The equatorial cells still retain the capacity to proliferate and differentiate into lens fibers. The increase in the volume of this lenticular material fills the space between the anterior and the posterior capsule, which results in the formation of a ring that often has the appearance of a string of sausages. Proliferating epithelial cells remain attached to the anterior capsule but also are found to a lesser extent on the posterior capsule, where they form small isolated groups.

In some cases the epithelial cells escape from the ring and migrate onto the anterior surface of the anterior capsule. Although the newly formed peripheral fibers appear almost normal, those that are forced inward sometimes degenerate, containing clusters of recrystallized proteins embedded in an amorphous material. These proteins normally are rod-shaped structures, but they may be spherical as well. In some cases the ring remains clear and yellow, but in others opacities develop and the ring looks cataractous. Because the ring forms at the periphery of the lens, vision is not affected.

The ring has two important functions. First, the haptics of an implanted IOL extend to the equator of the capsular bag held in place which prevents decentration. Second, the early fibrosis, which is known to seal the capsular surfaces, may help to contain the Elschnig's pearls by enhancing the seal between these two surfaces. The haptics of the IOL however, are a region of loose adherence through which epithelial cells may escape. The cells have to migrate only a small distance before the center of the posterior capsule is reached. Some surgeons believe that keeping the cut edge of the anterior capsular flap in front of the optic will ensure that the residual cells are kept further away from the center of the posterior capsule and thus reduce the incidence of PCO.

EVALUATION TECHNIQUES FOR POSTERIOR CAPSULE OPACIFICATION

Precise methods of evaluation are important to measure the progress of posterior capsular opacification. Most of the studies evaluate posterior capsule opacification in patients after ECCE/ phacoemulsification after full dilatation of pupil using slit lamp biomicroscopy. PCO is defined as opacification of the posterior capsule in the visual axis that is observed on silt lamp biomicroscopy, which includes Soemmering's ring (PCO peripheral to the IOL optic) and Elschnig's pearls and fibrous opacification behind the IOL optic.

The degree of opacification is assessed using:-

1) Visual acuity after surgery
2) Slit lamp biomicroscopy
3) Fundus visibility

Visual acuity after surgery

Visually significant posterior capsular opacification is defined as a decrease in the best-corrected postoperative vision by two lines in snellen's distant vision chart.

Slit lamp grading

1) Kruger grading:-

Kruger et al used a grading system of 0 to 3 to evaluate capsule opacification.

The criteria used were

0 = Absent.
1 = Very mild.
2 = Moderate.
3 = Dense white.

The capsule behind the optic was evaluated within a central area measuring 3 mm diameter, and also evaluated in the periphery. Distinction was given to grade Elschnig pearls and fibrosis.

2) **Sellman and Lindstrom grading:**

Sellman and Lindstrom graded fibrosis and Elschnig pearl formation on a similar four point scale. The original paper contains diagrams to illustrate the various grades for both fibrosis bands and pearls. These following grades were given:

1 = No or slight PCO without reduced red reflex, also no pearls at all or pearls not to the IOL edge.

2 = Mild PCO reducing the red reflex, Elschnig pearls to the IOL edge.

3 = Moderate fibrosis or Elschnig pearls inside IOL edge but with a clear visual axis.

4 = Severe fibrosis or Elschnig pearls covering the visual axis and severely reducing the red reflex.

Grading based on fundus visibility

Madurai intraocular lens study IV grading system:

It is based on visualization of the posterior pole assessed by examining the optic disc and macula using a Volk 90D lens.

Visualization of the optic disc was subjectively graded according to the following scale:-

1) Clear views of optic disc margin, but disc blood vessels and/or nerve fiber layer are not clearly seen.

2) Optic disc margin, as well as disc blood vessels and nerve fiber layer are not clearly seen.

Visualization of the macula was subjectively graded according to the following scale:-

0 - Clear view of foveal reflex, peri-foveal blood vessels and nerve fiber layer.

1 - Diminished foveal reflex, but clear view of peri-foveal blood vessels and nerve fiber layer.

2 - Blurred foveal reflex, peri-foveal blood vessels and/or nerve fiber layer.

The total scores of the visualization of the optic disc and the macula were combined to produce a total posterior pole visualization score (PoIVS) ranging from 0 to 4 in order of decreasing visualization.

IMAGING SYSTEMS

Scheimpflug system

Lasa et al showed in 1995 that Scheimpflug photography might be a useful tool for future assessment of PCO. The Schiempflug photography system was further developed by Hayashi in 1998. It is based on the use of the EAS-1000 anterior eye segment analysis system equipped with area densitometry to measure the scattering light intensity.

This principle is applied to obtain a cross sectional image of the anterior segment. An alignment system is coupled with a television monitor and the slit image of the best quality is transferred to the online image analysis computer.

The computer uses area densitometry to measure the scattering light intensity, which is deemed equal to the opacification density. To measure the central 3 mm

portion, three cross sections are taken at meridians of 0, 60, and 120 degrees and averaged out to give an approximate value of PCO. The value obtained was shown to have a good correlation with the visual acuity. The measurement method is easy to perform and can be done within a few minutes for each eye.

Digital photographic image acquisition systems

1) Brightness based analysis
2) Density map system
3) Computerized analysis of density boundaries
4) Texture analysis
5) Color coded grid system.

Tetz described a photographic image analysis system that can morphologically score posterior capsule opacification without dependence on visual acuity testing. Standardized slit lamp retroillumination photographs are analyzed. Posterior capsule opacification score is calculated by multiplying the density of opacification and graded from 1-4 by the fraction of capsule area behind the IOL optic that is opacified. This technique shows good inter-and intra-individual reliability.

Pandey et al reported a more sophisticated system of retro illumination imaging of the posterior capsule using a computerized high resolution digital system that can produce excellent images for objective documentation and quantitative measurement of posterior capsule opacification. Apple et al utilized Miyake-Apple posterior photographic technique for analyzing commonly used IOL model in eyes obtained postmortem to evaluate PCO and whether or not an eye had an Nd: YAG laser capsulotomy.

Prevention of Posterior Capsule Opacification[5, 10]

These measures can be divided into two categories. One strategy is to minimize the number of retained/regenerated LECs and cortex through thorough cortical cleanup. The second strategy is to prevent the remaining LECs from migrating posteriorly. The edge of the IOL optic is critical in the formation of such a physical barrier.

Three surgery-related factors and three IOL-related factors have been identified.

Surgery-related factors

1) Hydro dissection-enhanced cortical cleanup
2) In-the-bag (capsular) fixation
3) Capsulorrhexis edge on IOL surface

Hydro dissection-enhanced cortical cleanup:

This procedure was coined by Faust in 1984 and later on, in 1992 Howard Fine perfected the technique of subcapsular fluid injection and coined the term cortical cleavage hydrodissection. The cortical clean-up hydrodissection facilitate lens substance removal and enhance the safety of the surgery. The goal of hydrodissection is to remove equatorial cells and cortex, as opposed to single layer of anterior epithelium that does not migrate.

In-the-bag (capsular) fixation:

The advantage of the in-the-bag fixation is accomplishment of good centration. In-the-bag fixation of IOL functions primarily to enhance the IOL-optic barrier effect. When the IOL optic is fully in the capsular bag its contact is maximum with the posterior capsule and the barrier effect is functional. When one or both of the

haptics are out-of-the bag, a potential space exists that allows ingrowth of cells towards visual axis.

Capsulorrhexis edge on IOL surface:

A significant factor which helps in reducing PCO is creation of Capsulorrhexis with a diameter slightly smaller than that of IOL optic. This helps to provide a tight fit (analogous to a “shrink-wrap”) of the capsule around the optic.

IOL-Related Factors

1) IOL biocompatibility.
2) Maximal IOL Optic-Posterior Capsule Contact.
3) Barrier Effect of the IOL Optic.

IOL biocompatibility[5, 10]

Lens material biocompatibility is an often misunderstood term. It can be defined by many criteria e.g. the ability to inhibit stimulation of epithelial cellular proliferation. In large series of postmortem human eyes, the Alcon AcrySof IOLs presented with minimal to absent Soemmering’s ring formation PCO.

Hollick et al assessed PCO objectively by digital retro illumination imaging and found significant differences (P=0.0001) in percentage of PCO after 3 years between,

Acrylate (PCO 10%),

Silicone (PCO 40%) and

PMMA IOLs (PCO 56%).

Surface modification and coating of the intraocular lens:

The idea behind coating the IOL surface is to render it less attractive to cell attachment and cell growth. Heparin can be covalently bound to a PMMA IOL surface producing a heparin-surface-modified IOL with a hydrophilic surface.

Maximal IOL Optic-Posterior Capsule Contact[5, 10]

Other contributing factors in reducing PCO are posterior angulations of the IOL haptic and posterior convexity of the optic. This is due to the creation of a "shrink wrap", a tight fit of the posterior capsule against the back of the IOL optic. The relative "stickiness" of the IOL optic biomaterial probably helps to produce an adhesion between the capsule and IOL optic. There is preliminary evidence that the hydrophobic acrylic IOL biomaterial provides enhanced capsular adhesion, or "bioadhesion".

Barrier Effect of the IOL Optic[5, 10]

The IOL optic barrier effect plays an important role as a second line of defense against PCO, especially in cases where retained cortex and cells remain following ECCE. The concept of the barrier effect goes back to the original Ridley lens. If accurately implanted in the capsular bag it provides an excellent barrier effect, with almost complete filling of the capsular bag and contact of the posterior IOL optic to the posterior capsule ("no space, no cells"). A lens with one or both haptics "out-of-the-bag" has much less of a chance to produce a barrier effect. Indeed, the IOL optic's barrier function has been one of the main reasons that PC-IOLs implanted after ECCE throughout the decades did not produce an unacceptably high incidence of florid PCO.

The effect of a square-edge optic design as a barrier was first discussed by Nishi et al. A truncated, square-edged optic rim appears to cause a complete blockade of cells at the optic edge, preventing epithelial ingrowth over the posterior capsule. A major disadvantage of the truncated edge is the production of clinical visual aberrations, such as glare, halos and crescents.

Another example of design modification include introduction of sensor optic edge IOL manufactured by Advanced Medical Optics. This IOL has a squared posterior edge and a round anterior edge. Therefore, it avoids optical dysphotopsias, while retaining the PCO beneficial squared posterior edge.

Pharmacological Techniques and Immunological Inhibitors for PCO[5-10]

Efforts to prevent posterior capsule opacification after cataract surgery have included eliminating lens epithelial cells during the operation, or affecting the growth of these cells after the operation. Drugs used for these purposes can be applied during the operation. A long acting delivery system for these substances has also been tried by using poly-DL-lactid, a polymer of the monomeric lactid acid as a carrier substance, which slowly delivered specific drugs.

Three different groups of drugs have been tried:-

1) Anti- metabolites - designed to kill the remaining LECs.
2) Immunotoxins - for inhibiting the proliferation and migration.
3) Anti-inflammatory drugs - to hinder inflammatory reactions after cataract surgery.

1) Antimetabolites[11]

Many antimetabolites have been tried. Actinomycin D, methotrexate, daunomycin, 5-fluorouracil, colchicines, doxorubicin, cytosine arabinoside, and

rnitomycin C have all been tested. Colchicine an alkaloid destroys proliferating cells by arresting mitosis. Methotrexate-anticollagen conjugate was seen to hinder LEC growth in cell culture.

Daunomycin is the only one of all the anti- metabolites, to reach clinical human studies for this purpose.

2) Immunotoxins and lens epithelial cell adhesion molecules as blocking agents[11]

Lens epithelial necrosis factor (LENF) , antitransferrin receptor immunotoxin, ethylenediaminetetra-aceticacid , 4187X-ricin A immunoconjugate, lens epithelial cell adhesion molecule blocking agent, and immunotoxin MDX-RA have been tried as inhibitors of PCO after cataract surgery and IOL implantation.

LENF, antitransferrin receptor immunotoxin and 4187X-ricin A immunoconjugate could all inhibit LEC growth in cell cultures. Antitransferrin receptor immunotoxin consists of a monoclonal antibody of the IgG type directed against transferrin receptors, and it is chemically linked to protein synthesis inhibiting ricin-A. The idea is to destroy rapidly dividing LECs, which have a higher density of transferrin receptors as do all rapidly dividing cells. 4187X-ricin A immunoconjugate reacts specifically with an antigen on the human LEC membrane, and the ricin component inhibits protein synthesis.

Ethylenediaminetetra-acetic acid also disrupts the binding of LEC integrins to their ligands by chelating Ca^{2+} cations, and therefore it can inhibit LEC migration and proliferation. Lens epithelial cell adhesion molecule blocking agent had a synthetic ROD peptide, which was used to inhibit LEC integrins by competitive binding to the integrin beta chain.

The only immunotoxin, which has been tried in human cataract operations, is immunotoxin MDX-RA. It could significantly reduce PCO and was well tolerated.

Thapsigargin and caffeine inhibited LEC growth in cell culture. Thapsigargin-coated IOLs inhibited LEC growth in a human capsular bag culture system and total cell death of the residual anterior epithelial cells was achieved with higher concentrations. Thapsigargin and caffeine both inactivate Ca^{2+} cations, thapsigargin by blocking the Ca^{2+}-ATPase reuptake mechanism, and caffeine by blocking the release channel of $Ca^{2+}$from the cell.

3) Anti-inflammatory and immunomodulating drugs

Indomethacin, diclofenac sodium and cyclosporine-A inhibited LEC proliferation in cell culture. They all were shown to decrease the amount of prostaglandin E_2 produced by the LECs. Indomethacin-coated IOLs significantly reduced postoperative inflammation and PCO in rabbit eyes. Sustained release of indomethacin also significantly decreased inflammation, but it did not reduce PCO in rabbit eyes.

Toxicity to corneal endothelium and other ocular structures remains one of the major concerns for using cancer chemotherapeutic drugs, anti-inflammatory substances, hypo-osmolar drugs, and immunological agents, when the intralenticular compartment is in direct contact with the anterior chamber. However, with the development of a Sealed Capsular Irrigation (SCI) device, it is now possible to precisely deliver the pharmacological/ hypo-osmolar agents to the lens epithelial cells within the capsular bag, while minimising the potential for collateral ocular damage.

Management of Posterior Capsule Opacification

In the past, invasive surgical posterior capsulotomy was the primary treatment of posterior capsule opacification and it is still performed where the Nd: YAG laser facility is not available or in cases with very dense or fibrotic membrane.

The treatment of choice for clinically significant posterior capsule opacification is Nd: YAG laser posterior capsulotomy. It is an effective modality in the management of posterior capsule opacification.

There are several disadvantages of Nd: YAG laser capsulotomy. There are several vision-threatening complications such as damage to IOL optic, postoperative intraocular pressure elevation, cystoid macular edema, retinal detachment, IOL subluxation or dislocation and exacerbation of localized endophthalmitis. Nd: YAG laser posterior capsulotomy significantly increases the overall cost of cataract surgery beside burden on the health care.

Keeping in view several vision-threatening complication of Nd: YAG laser capsulotomy polishing of capsule is recommended in particularly high myopia patient where incidence of RD increases several folds after Nd: YAG capsulotomy.

Nd-YAG LASER POSTERIOR CAPSULOTOMY[9-10]

HISTORY

The recognition that the energy of sun could damage the human eye was a first step in the development of ocular phototherapy. Bonetus in Geneva in the 17th century was the first to describe the occurrence of a scotoma after sun gazing. Czerny and Deutschmann experimented on the retina of rabbits by focusing sunlight with a concave mirror and convex lens and noted the appearance of greyish burns, which later produced hyper pigmented scars.

Gerd-Meyer-Schwickerath demonstrated that the focused radiant energy could be used to create chorioretinal lesions of clinical value. Initially they developed a photo coagulator that used the sun as its light source and later developed the Xenon arc photo coagulator. Theodore Maiman in 1960 built the first laser which employed a ruby crystal as a medium. In 1968 L'Esperance developed ophthalmic Argon laser.

The development of Nd-YAG laser took place in the mid of 1970's, Beckman & sugar in 1973 used it for thermal cyclodestruction. Krasnov demonstrated that the high peak power pulses of Nd: YAG could be used to produce clinically desirable disruption of structures in the anterior segment of the eye.

In 1981 Frankhauser and his associates developed Q-switched (nanosecond) YAG laser and Aron Rosa and her associates developed the picoseconds mode-locked YAG laser.

Schematic display of a laser designed for the emission of short, intense pulses (Q-switched laser). The optical switch is essential for Q-switched laser action it becomes transparent permitting light to reach mirror, only during very short time interval.

Pulses may be manipulated by varying the parameters of the pumping flash lamp and the characteristics of the optical resonator and a continuous beam can be generated. This is called c-w mode and is not good enough for an optical breakdown which can be achieved only by Q switched or a mode locking mode.

Q-switched laser.

Q-switched high-power lasers emit pulses of much shorter duration ranging from 5 to 20 ns in duration. The generation of such pulses is achieved by temporary closing of the shutter within the cavity of laser rod, which places maximum energy in

the rod and leaves it in a highly amplifying condition if the pumping excitation is continued.

The sudden opening of the shutter results in a rapid build-up of circulating wave light intensity as it moves from mirror to mirror which reaches levels of 1000 times greater than a continuous wave laser. The beam which is coupled out is 106 watts. To couple out the light we need a very fast shutter which is not possible mechanically so we can use two types of shutters.

- **Pockets cell polarize assembly** - This is an electrically switchable optical assembly that in one state is quite transparent to light while in other it is opaque. Once the shutter is opened the stored energy in laser cavity is converted to a circulating light wave power. This change is called Q switching.
- **Use of bleachable dye -** A special organic dye either in a liquid solvent or in the form of a plastic film is interposed at the shutter. This dye is non transmissive when the incident light is weak, but when the intensities are sufficiently high it bleaches and becomes transparent, allowing the laser beam to go out and again the dye mold becomes opaque.

Pocket cells polarizers is more efficient then dye in converting the stored energy to laser output but has the inherent problem of being more complex and prone for malfunction.

Basic mechanism of mode locking the four-mode situation: Four waves of identical amplitude superimpose in such a way that at a time t all wave forms assume their maximum value. This event repeats periodically with time (T) intervals.

Mode-locked laser

The term mode, refers to two kinds of optical waves that are generated within the laser resonator. One of these is called the longitudinal mode, and the other the transverse electrical mode (TEM), depending on how the modes are oriented relative to the optical axis of the laser resonator.

The TEMs may significantly influence the optical properties of the Q-switched laser. In the mode-locked laser, the longitudinal modes play the decisive role, since they are coupled or synchronized together, resulting in a cumulative effect.

The mode-locked laser emits a train of very short pulses or spikes (often 7 to 20). Typically, each spike has duration of about 30 to 80 ps, and the interspike spacing is about 5 to 10 ns. The duration of the whole pulse train depends on the number of spikes contained in it and is usually of the order of less than 100 ns. The greater the number of longitudinal modes coupled together, the shorter the duration of the individual spikes. This in turn implies greater maximal irradiance of the single spikes.

PHOTODISRUPTION

Laser—tissue interactions may be divided into

1) Photochemical (Photo radiation ,Photo ablation)
2) Thermal (photocoagulation ,Photo vaporization)
3) Ionizing effects (Photo disruption) most often interactions include a mix of these.

Photo disruption can be defined as the use of high peak power ionizing laser pulses to disrupt tissue. Light energy is concentrated in time and space of lasers capable of emitting high power through very short light pulses.

The unique properties of the Nd: YAG lasers depend on the infrared wavelength and the ultra short duration of the laser pulse. It penetrates through the aqueous with μminimum absorption. The Nd: YAG laser beam can be focused sharply inside non-pigmented ocular tissue. The ultra short pulse of the Q switched Nd: YAG laser results in tissue ionization. This causes a “blasting effect” resulting in tissue destruction with minimal thermal effect.

Photo disruptors focus such an intense electromagnetic radiation into a small area during a brief period of time that the electromagnetic field of the laser pulse strips electrons from atoms and molecules at the focal point creating plasma. Plasma is a mixture of ions and free electrons rarely existing in nature outside the atmosphere of the sun or other stars. The rapid expansion produces shock and pressure waves which create additional mechanical damage around the target site.

Because of extremely short pulse length, the short pulsed YAG lasers, with 50 times less energy than an Argon laser pulse, can produce power densities greater than a billion watts per sq cm compared with 50,000 watts per sq cm for the Argon laser. The p second-long pulse is 10 billion times shorter than a typical Argon application. Current ophthalmic Q-switched Nd: YAG lasers have a maximum output of 10 to 20 mj, while mode-locked units have a maximum output of approximately 4.5 mj.

MECHANISMS OF TISSUE DAMAGE BY LASERS

Many mechanisms of damage by laser have been described. The micro plasma temperature reaches 15,000°C focally causing vaporization and melting of liquids and solids in a small volume near the focal point. In biologic systems, thermal denaturation of protein and nucleic acid is confined to a radius of 0.1 mm for 1 mj pulse. Multiple mechanisms combine to generate pressure waves radiating from the

zone of optical breakdown. Foremost among these is the rapid plasma expansion that begins as a hypersonic shock wave. The shock wave begins immediately with plasma formation and expands at a hypersonic velocity of 4 km/second.

The wave front propagation falls to sonic velocity (1.5 km/sec in water) within 200µ .The acoustic transient lasts 15 n/sec at location 300µ from the focal point. The pressure falls from maximum of 1000 atm to 100 atm within a distance of 1 mm. The shock wave is followed by cavitations or vapours bubble formation.

Cavitation begins within 50 to 150 n sec after breakdown in water. The cavitations are too rapid to be visible and should be distinguished from bubble formation. Persistent bubbles probably consist of hydrogen and oxygen gases.

BEAM OPTICS

A pulsed Nd: YAG system requires separate aiming system because there is no emission between pulses and because of the invisible infrared wavelength. Most Nd: YAG system has a single continuous-wave. Low-power Helium-Neon laser coaxial with the Nd: YAG pathway to locate the focal point. The He-Ne Laser emits a red beam at 632.8 nm with an irradiance level below the threshold for retinal injury. An aperture or prism arrangements maybe used to modify the He-Ne beam into annulus (circle) pattern or into two or more separate beams.

The cone angle of the beam delivered to the patient is the major factor in retinal protection. The divergence of the beam after the focal point will reduce the irradiance at the retina below the threshold for retinal damage. Many current ophthalmic Nd: YAG lasers are specified to have a solid cone angle to about 16 degrees (max 20 degrees). The larger the cone angle, the lower the irradiance at a

given distance in front of and behind the focal point. The divergence at the cone angle may reduce the irradiance at the retinal level by a factor of nine.

OPTICAL BREAKDOWN

When the intensity of light propagating in transparent medium is sufficiently high, atoms are ionized and the originally transparent medium turns opaque. This is called optical breakdown and is observed as a bright light flash accompanied by a sharp sound. This commonly occurs once a certain threshold power density is achieved by focusing Q-switched or mode-locked laser radiation.

Immediately after the optical breakdown has been initiated laser energy is absorbed by the plasma which causes intensive local heating up to 15,000 degrees. The inherent high pressure leads to rapid expansion of the plasma with the consequent emission of the shock waves which expands spherically. This phenomenon results in tissue destruction which is utilized for cutting iris or the posterior capsule or for creating a shock wave which helps to sweep away deposits on IOL (as in YAG sweeping).

Optical breakdown is a nonlinear process. It occurs suddenly when the density of power exceeds the threshold for optical breakdown. Increasing the energy input causes the breakdown zone to move anteriorly. Suprathreshold energies result in an elongated breakdown region that extends anteriorly along the laser beam axis.

Factors influencing optical breakdown

1) Optical breakdown is degraded by induced astigmatism. Whenever possible, the laser pathway should be through central-clear cornea, perpendicular to the cornea and the IOL. When marked corneal astigmatism or irregularity is present, use of a YAG contact lens reduces this problem by shifting the air

interface from the cornea to the optically superior contact lens front surface. It also acts as a plus lens, increasing beam convergence, reducing focal spot size, and magnifying the surgeon's view.

2) The threshold for optical breakdown is also lower at optical interfaces. Breakdown occurs if the laser is accurately focused on the capsule, but no breakdown is seen if the laser is slightly misfocused in the aqueous or vitreous. The He-Ne focal point must be well-defined. The smaller the depth of focus of the He-Ne aiming beam, the more reproducible is the localization of the zone of the YAG optical breakdown.

Indications for YAG Capsulotomy

A gradual decrease in visual acuity, after successful surgery or optical degradation of visual quality like glare, altered contrast and color sensitivity.

Contraindications to Laser Capsulotomy

Absolute

1) Difficulty in target visualization like in corneal opacity irregularities or edema
2) Inadequate stability of eye

Relative

1) Glass IOL
2) Known or suspected CME
3) Active iridocyclitis
4) High risk for retinal detachment (RD)

Timing of Capsulotomy

A complete ophthalmic history and a detailed examination is an essential prerequisite to the procedure. The best clinical tool is the direct ophthalmoscope and the evaluation of the red reflex. The laser interferometer, the potential acuity meter (PAM), the lens opacity meter and contrast sensitivity testing are quite unreliable. Direct ophthalmoscopy may show a disturbed red reflex and wrinkling of the posterior capsule. Fluorescein angiography is indicated in cases where cystoid macular edema is suspected.

According to most authors there is no reason to wait at least three months before opening the capsule, and capsulotomy maybe performed as early as three weeks after surgery. However, some authors advise to wait till the time when the IOL optic has fixed to the posterior capsule, so that YAG capsulotomy will not disrupt this apposition. Y Levin et al suggest to delay the capsulotomy for six months in eyes with diabetic retinopathy

TECHNIQUE OF Nd: YAG LASER POSTERIOR CAPSULOTOMY[8-11]

Preparation of the Patient

1) Explanation of the Procedure and Informed Consent.
2) Anti-glaucoma Medication should be administered one hour before procedure.
3) Dilatation of pupil may not be required, except for the beginners and in mitotic pupil. One may mark the centre of the pupillary axis in the capsule with a single laser shot. When the pupil later dilates the marker reminds the surgeon of the patient's true visual axis. Dilating the pupil results in a larger capsular window, causing more debris liberation with more risk of complications.

4) Topical Anesthesia is required only if contact lens is employed. This can be achieved by proparacaine 1 percent, 1 to 2 drops immediately before the capsulotomy. A retro bulbar injection for akinesia may be required in rare cases of nystagmus and extremely apprehensive patient.
5) Patient must be seated comfortably with properly adjusted stool, table and chin rest. A darkened/semi darkened room is preferable as it improves surgeon's visualization of the target. An illuminated target is provided to the patient for maintaining steady fixation.
6) A head strap may be used to maintain forehead positioning.

Procedure

A contact lens such as Peyman or Abraham lens may be used. Contact lens helps in stabilizing the eye, improve laser optics and facilitate accurate focusing.

Abraham lens increases the convergence angle from 16 to 24°, decreases the area of laser at posterior capsule to 14µ from 21µ and increases beam diameter at both the cornea and retina. The laser may get focused on the retina if laser is not delivered through the lens button.

The capsutotomy is started eccentrically with minimal energy 1 to 2 mJ/pulse. This eccentric position is preferred to predict the behavior of posterior capsule to photodisruptive forces and avoid pitting of IOL in central position.

The shots are placed across the tension lines to obtain largest opening per shot. The usual strategy is to create a cruciate opening beginning superiorly near the 12 O'clock position and progressing down towards the 6 O'clock position. This is followed by placing shots at 3 and 9 O'clock position. Any flaps created during the procedure are cut, so as to cause them to retract and fall back to periphery. Free

floating fragments should be avoided. A posterior defocusing of the laser beam should be done to avoid lens pitting.

Christmas tree pattern should be used in case of increased tendency to lens pitting. In this technique shots are placed nasally and temporally rather than progressing from 12 O'clock to the 6 O'clock position.

In case of aphakia an anterior defocusing is done to avoid breaking the anterior hyaloid face.

The size of the capsulotomy should match the size of the pupil in the physiologic state, about 4 mm.

Postoperative Care

Topical beta-blocker or apraclonidine is used immediately after the procedure and continued for at least 1 week.

Topical steroids and cycloplegics are used to counter iritis postoperatively.

COMPLICATIONS

1) Intraocular pressure rise
2) Iritis/ uveitis/vitritis
3) Iris hemorrhage
4) IOL Pitting/ cracking
5) Retinal detachment
6) Cystoid Macular Edema
7) Other Complications –corneal edema, pupillary glaucoma, macula hole, retinal haemorrhage, hyphema, re-opacity.

1) Intraocular pressure rise [12]

IOP rise has been well documented, and there are several postulated mechanisms. Obstruction of the trabecular meshwork by inflammatory cells and debris from the capsulotomy is thought to cause a reduced drainage capacity. Lynch et al showed a quantifiable reduction in outflow facility in monkey eyes following Nd: YAG capsulotomy and histopathological studies showed the anterior chamber trabecular meshwork to contain fibrin, lens material, inflammatory cells, pigment, and erythrocytes. Studies using laser flare cell meter-on human subjects also show that acute intraocular hypertension after capsulotomy is related to elevated aqueous particles.

Schubert postulates specifically that the loss of dialyzable protein from disrupted vitreous is responsible for the IOP increase. Other postulated mechanisms include pupillary block due to the forward movement of vitreous and shockwave damage to the trabecular endothelial cells, release of inflammatory-mediators, and direct effect on trabecular cells. Studies in the 1980s, soon after the inception of laser capsulotomy, were varied in their reported levels of IOP rise but tended to show that a large proportion of patients had some degree of pressure rise (25- 90%). The level of pressure rise was very variable among studies. In prospective trial of 526 patients, Keates et al found that IOP increased to 30 mm Hg or more in 5.7% and returned to pretreatment level in 89% by 1 week post-treatment. Stark et al showed that out of a total patient population of 17,911 that 28% had an IOP increase of over 30mm Hg from pre-treatment values. He also found a persistent increase in intraocular pressure in about 1% of patients. Intraocular pressure begins rising in the period immediately after laser posterior

capsulotomy. The pressure rise tends to maximize at 3 to 4 hours after laser treatment, and it returns to within 5 mm Hg of pre-laser values by 24 hours.

Risk Factors

Channell et al found that smaller capsulotomies lead to smaller IOP increase, this is due to the smaller amount of debris released, and hence less trabecular meshwork obstruction.

Aron-Rosa reported that in 200 patients studied, all the pressure rise were when the energy per pulse was 2.5 mJ or higher.

Patient-dependant risk factors include aphakia, glaucoma, pre-existing IOP of greater than 20 mm Hg, high myopia, and vitreoretinal disease.

Treatment

1) Apraclonidine 1%
2) 0.5% topical Timolol
3) Topical Levobunolol0.5%
4) Topical 2% Pilocarpine
5) Oral Acetazolamide

2) Iritis/ uveitis/vitritis[12]

In a study of 526 patients, Keates et al found iritis persisting in 0.4% and vitritis persisting in 0.7% after a 6-month postoperative period. Chambless, in a study with an average follow-up period of 7 months, found persistent anterior uveitis in 1.4%. Thus, although transient anterior chamber flare may be seen post-laser treatment, persistent iritis or vitritis is rare.

3) Iris hemorrhage[12]

The Nd: YAG laser does not photocoagulate tissues so when it cuts them, small vessels on the iris that are ruptured will bleed. Gardner et al found a 3% incidence of iris bleeding with resulting minimal hyphema which cleared completely by 1 week post laser.

4) IOL Pitting/ cracking[12]

Rates of damage have been quoted at between 4% to 40% of cases. Lathe cut polymethylmethacralate (PMMA) lenses had stellate crater patterns while silicone lenses had a smooth splash-like pattern.

5) Retinal detachment[12]

Many studies on incidence of retinal detachment after Nd: YAG capsulotomies have shown increased rates (incidence of 0.5% to 3.6%) compared to patients who have not had laser treatment.

Koch et al found that risk factors for retinal detachment include high myopia, lattice degeneration with associated holes, greater use of laser energy, and larger capsulotomy size.

6) Cystoid Macular Edema[12]

The etiology of CME following Nd: YAG capsulotomy most likely involves movement of the vitreous cavity and vitreous damage, which results in the release of inflammatory mediators. Vitreoretinal traction caused by the procedure may also play a part.

Many studies have shown the incidence of CME following laser capsulotomy between 0.7% to 4.9%.

7) Other Complications

1) Corneal edema,
2) Pupillary block glaucoma
3) Macula hole
4) Retinal hemorrhage
5) Endophthalmitis: Propionibacterium acnes causes an indolent endophthalmitis which cocoons the lens and presents as PCO. Nd:YAG capsulotomy in such cases may cause their spread into vitreous producing endophthalmitis.

ADVANCEMENTS IN Nd: YAG LASER CAPSULOTOMY

Pitting of intraocular lens occurs in 15% to 33% of eyes during Nd: YAG laser posterior capsulotomy. The pitting usually is not visually significant, although rarely the damage may cause sufficient glare and image degradation that the damaged intraocular lens must be explanted. In 1984 Bath PE, Hoffer KJ, Aroner-Rose D et al has done study on glare disability after Nd: YAG laser capsulotomy.

Naeser, Rask and Hansen reported 3.0% posterior capsule opacity requiring YAG capsulotomy and 12.1% moderate posterior capsule opacity not requiring YAG. Sellman and Lindstrom in 1988 reported 7.1 - 16.1 % moderate/severe Elschnigs pearl migration and 2.4 - 4.4% capsular fibrosis. Watts have reported in 1986 of 2 - 4 % capsular membrane thickening.

Moisseiev, Bartov, Schochat et al reported in 1989 41 % posterior capsule opacity requiring capsulotomy.

Cystoid Macular edema has been reported in 1991 to develop in 0.55% to 2.5% of eyes after the Nd: YAG laser capsulotomy by Keates RH, Steinert, Pulioilto, et al who studied long term follow up after Nd: YAG laser[13-14].

Shaumberg et al conducted an important Meta analysis in 1998 of published articles on posterior capsule opacity and generated pooled estimate of eyes developing posterior capsule opacity over three postoperative points viz. 1, 3 and 5 years[15-16]. They noted that the rate of posterior capsule opacity remains high over 25% during the 5 years postoperative period.

Javitt JC, Teilsch JM et al in 1999 had followed up patients after Nd: YAG capsulotomy and found that cases of iritis are less than 1 % of eyes[17].

Findl O,Drexher W et al in 1999 studied the changes in IOL position caused by Nd:YAG laser capsulotomy with 3 IOL styles, 1 piece PMMA , 3 piece foldable and plate haptic in 32 pseudophakic eyes with PCO .The capsulotomy included backward IOL movement in 32 eyes . It was more pronounced with plate-haptic IOLs than with 1 piece PMMA and 3 piece foldable IOLs[18] .

Jayne G , Martin Wand et al in 2000 studied the long term effect of Nd:YAG capsulotomy on intraocular pressure in 100 patients who were followed up for 1.5 years .The changes in IOP were significantly higher in eyes with capsulotomy than those in non-capsulotomy eyes. Patients with glaucoma require long term additional antiglaucoma medication than with non-glaucoma paients who require initial glaucoma therapy after capsulotomy[19].

Wang J et al in september 2002 measured visual acuity , contrast sensivity and glare sensivity in 73 pseudophakia eyes before and after Nd:YAG laser posterior capsulotomy. It concluded that visual acuity,contrast sensivity curve and glare sensivity curve improved in all cases post operatively [20].

Hayashi K, Hayashi H et al in Oct 2003 studied correlation between visual acuity ,contrast sensivity and contrast sensivity with glare source before and

after Nd:YAG laser capsulotomy in 90 pseudophakic eyes . Before laser capsulotomy visual acuity,correlated with PCO. After capsulotomy,visual function improved markedly and no longer correlated with PCO[21] .

Sheard RM, Goodburn SF , et al , in 2003 studied the incidence of retinal detachment after Nd:YAG laser posterior capsulotomy in patients with PVD The prevalence of PVD was significantly higher in eyes after ECCE and IOL implantation than in phakic eyes independent of Nd:YAG laser posterior capsulotomy .Presence or absence of PVD at the time of capsulotomy is not helpful in assessing the risk for RD in first year after laser tratment[22] .

BillotteC, Berdeaux G in October 2004 evaluated the clinical consequences of complication from Nd: YAG laser capsulotomy for PCO in 70 year old population over 9 years . It concluded that reducing PCO and associated use of Nd:YAG laser capsulotomy contribute to preserving visual acuity[23] .

Ratana P et al in 2004 studied the incidence of retinal breaks and retinal detachment after Nd:YAG laser posterior capsulotomy and prophylactic treatment in preoperative retinal breaks in 341 patients (350eyes) . By 5 yrs, the overall cumulative proportion of RD was seen in the 341 patients .2% Patients were in close follow up and prophylactic photocoagulation was done for pre-existing retinal breaks in high risk eyes [24].

Hucy Woung LC et al in 2004 conducted a study on a 68 years old woman who had cataract surgery, presented with decreased visual acuity and posterior capsule opacification .She underwent Nd:YAG laser capsulotomy .Two months later , she developed decreased visual acuity secondary to cystoid macular edema . She was treated with 0.5%ketorolac drops for 3 weeks, later her vision improved .This

indicates CME related to Nd:YAG laser capsulotomy is caused by prostaglandin release and responsive to topical non-steroidal drops[25] .

Yilmaz S et al in september 2006 conducted a study on 128 pseudophakic eyes to determine the effect of Nd:YAG laser posterior capsulotomy size on refraction and visual acuity .Patients were divided in to 2 groups depending upon capsulotomy size , 80 had small size (<4 mm) and 48 had large size (>4mm) .Visual acuity and refractive error were measured post operatively and 1, 14, 30, and 90 days post operative . It concluded that size of posterior capsulotomy does not affect refractive error and visual acuity[26].

Cinal A, Demirok A et al in December 2007 evaluated Nd:YAG laser posterior capsulotomy procedure with or without anesthesia in 35 pediatric eyes and 51 adult eyes .He concluded that Nd:YAG laser capsulotomy can be performed safely and effectively in children under topical anesthesia[27].

Zemaitiene R et al in May 2007 evaluated the impact of sharp edged intraocular lenses with different haptic designs made from same hydrophobic acrylic material on posterior and anterior lens capsule opacification. He concluded that patients with one piece acrylic hydrophobic IOL group more frequently presented with capsular folds behind the IOL optic area than those in three piece IOL group[28].

Lin N, Chex X, et al, in May 2008 studied the effect of Acrysof intraocular lenses (IOL) on the development of posterior capsule opacification compared with silicon or polymethyl methacrylate (PMMA) IOLs for patients with senile cataracts. Acrysof and sharp edged silicon IOLs are similarly effective in inhibition of PCO after cataract surgery[29].

Varga A, Sacu S et al in 2008 studied the effect of posterior capsule opacification on macular sensivity and BCVA before and after Nd:YAG laser capsulotomy in pseudophakia eyes. After Nd:YAG laser capsulotomy BCVA and macular sensivity improved. The PCO scores correlated well with induced loss of macular sensivity. Functional macular mapping indicated that there is no significant associated between PCO values and macular sensivity in eyes with ARMD[30].

Rasool W, Raza A and Ali SI in 2012 studied Nd: YAG laser capsulotomy and opined it is an effective treatment of posterior capsule opacification, but is not without attendant risks.[31]

MATERIALS AND METHOD

Source of data

This is hospital based study of 58 eyes of 56 patients with posterior capsular opacification following cataract surgery.

All patients were out patients in the Department of Ophthalmology of Shri B M Patil Medical College Hospital and Research Center during the study period between October 2010 to April 2012.

Method of collection of data

Sample size :- The average rate of posterior capsular opacity is 75% and allowable error of 15 % with 95 % confidence limit and worked out sample size is 57, using the following statistical formula :-

$$n = \frac{(1.96)^2 \times p \times q}{L^2}$$

Statistical analysis:-

1. Diagrammatic presentation
2. Mean +/- Standard Deviation
3. Suitable statistical list like MC numers , x^2 test ,or chi square test

Notification of cases:-

Patient with significant PCO after cataract extraction were included for the study. A total number of 58 eyes were studied. The data collected were categorized into sex, age, type of PCO, time interval between cataract surgery and PCO development, energy level used, visual acuity before and after Nd: YAG laser, IOP during the follow up after the procedure.

Detailed history was taken regarding the duration of symptoms and treatment previously taken. Ocular examination included visual acuity, intraocular pressure, and fundoscopy.

Slit lamp examination of cornea, anterior chamber, Position of IOL and type of PCO based on morphology was done.

Once the posterior capsule opacification was determined to be the cause of loss of vision, Nd: YAG laser posterior capsulotomy was advised. Relevant patient details were recorded in the proforma. Informed consent was obtained from all the patients after explaining the need, risk, complication and other available treatment options in their vernacular language. Visual acuity, IOP was recorded. Fundus examination was done to rule out any retinal pathology. Q- Switched Nd: YAG laser was performed under topical anesthesia. Power setting and number of exposures were varied depending on the thickness of the posterior capsule. The goal was to achieve an opening slightly larger than the pupillary size in room light with as few exposures as possible.

Visual acuity and IOP was recorded after 1 hr, 1week, 1month and 3 months after the procedure. Antibioticsteroid eye drops 4 times a day for 1week were

prescribed. Anti glaucomatous drugs, Tab. Acetazolamide 250 mg BD was advised when needed.

The cases were carefully followed up and looked for any incidence of IOL pitting, IOL crack, iritis, and raise in IOP, hyphaema, vitritis, RD, CME, iris bleed and reopacity.

The following parameters were studied

- Visual acuity
- IOP
- Complication

Inclusion criteria

Those patients were included who underwent routine extracapsular cataract extraction with IOL with visually significant PCO.

Exclusion criteria

- Corneal edema or haze
- Glaucoma
- Vitreous opacities and haze
- Myopic degeneration
- Old cystoids macular edema
- Optic atrophy

Pre laser preparation: - All patients were anaesthetized with topical anesthesia.

Preliminary examination:-

- Preliminary examination under torch light and visual acuity testing by Snellen's chart was done for all patients participating in the study
- Slit lamp examination to study the morphology of PCO
- Tonometry
- Direct and indirect ophthalmoscopy

Procedure:-

After adequate topical anesthesia, patient was made to sit at the YAG laser set up with chin rested against the chin rest, Peyman or Abraham contact lens was used to stabilize the eye. Peyman or Abraham contact lens was used to improve the laser beam optics, and facilitate accurate focusing. The Abraham contact lens increases the convergence angle to 24° from 16°, decreases the area of laser at the posterior capsule to 14µm from 21µm, and increases the beam diameter at both the cornea and the retina. The Abraham contact lens must be used with care because it is a modified posterior pole lens, if the Nd: YAG laser is not sent through the lens button, but rather the peripheral "carrier" portion of the lens, the Nd: YAG laser may be focused on the retina and cause damage[32].

The minimal amount of energy necessary to obtain breakdown and rupture the capsule is desired. In most cases capsule can be opened by using 1–2mJ/pulse.

The capsule was examined for wrinkles that indicate tension lines. Shots placed across tension lines results in the largest opening per pulse because the tension causes the initial opening to widen.

In the cruciate pattern, the first spot was placed superiorly and peripherally at 12 o' clock position and extended towards 6 o'clock. Then to complete the cross, shots were extended from the center to 3 o'clock and 9 o' clock. Laser spots unavoidably hit the IOL because of the close apposition of capsule and lens. Some ophthalmologists recommends a Christmas tree approach, in this the first spot starts at 12 o'clock and is swept down towards 4:30 and 7:30; this will avoid the risk of central lens damage [32].

Post laser treatment

- Antibiotic -steroid 4 times a day for 1week were prescribed.
- Anti glaucoma drugs, Tab Acetazolamide 250 mg BD was advised when needed.

Follow up

After laser visual acuity and IOP were recorded at 1 hr, 1week, 1 month and 3 month. During the follow up patients were examined for complications like hyphema, aqueous flare, IOL pitting, IOL crack, iritis, raise in IOP, vitritis, RD, CME, iris bleed and reopacity.

RESULTS

During the study period 58 eyes of 56 pseudophakic persons with significant PCO, who attended OPD, were assessed for PCO and consent was taken. The following observations were made which are depicted below in tabular and graphical formats

Table No.1: - Gender Distribution

Sex	No. of cases
Male	**28**
Female	**28**
Total	**58**

Note: 1 Male-both eyes; 1 Female-both eyes.

The table showing gender distribution, where in PCO was found to be 1:1.

Figure1:- Shows Gender Distribution

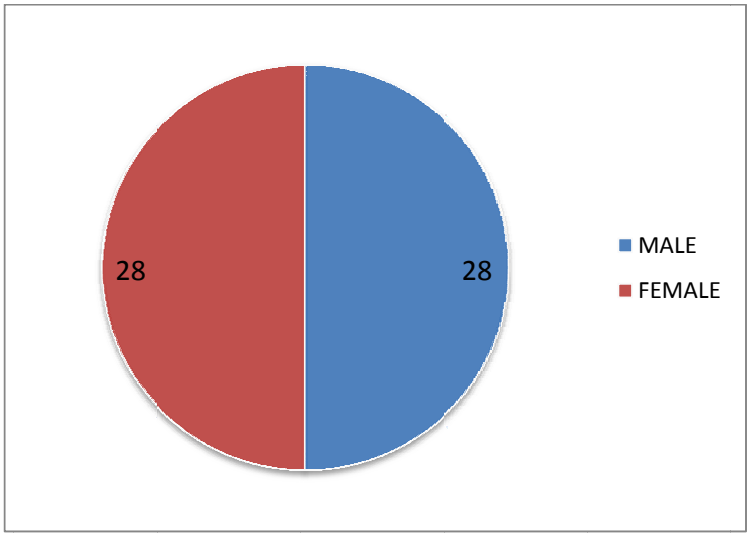


Table No 2:- Age Distribution

Age (Years)	No. of Patients	% Age
<10	1	1.18%
10-20	2	3.57%
21-30	3	7.14%
31-40	0	0%
41-50	9	16.07%
51-60	14	25%
61-70	18	32.14%
71-80	6	10.71%
81-90	2	3.57%
TOTAL	56	100%

Note: 1 Male-both eyes;1 Female-both eyes

Above table shows, Out of 56 patients, 18 (32.14%) patients were in the group of 61-70 years of age and 14(25%) patients were in the group of age 51-60 years . 9 (16.07%) patients were in 41-50 years of age ,6 (10.71%) patients were in the age group of 71-80, 4 (7.14%) patients were in 21-30 years of group, 2 patients were in age group of 10-20 and 81-90years each.

Figure 2:-Shows Age Distribution

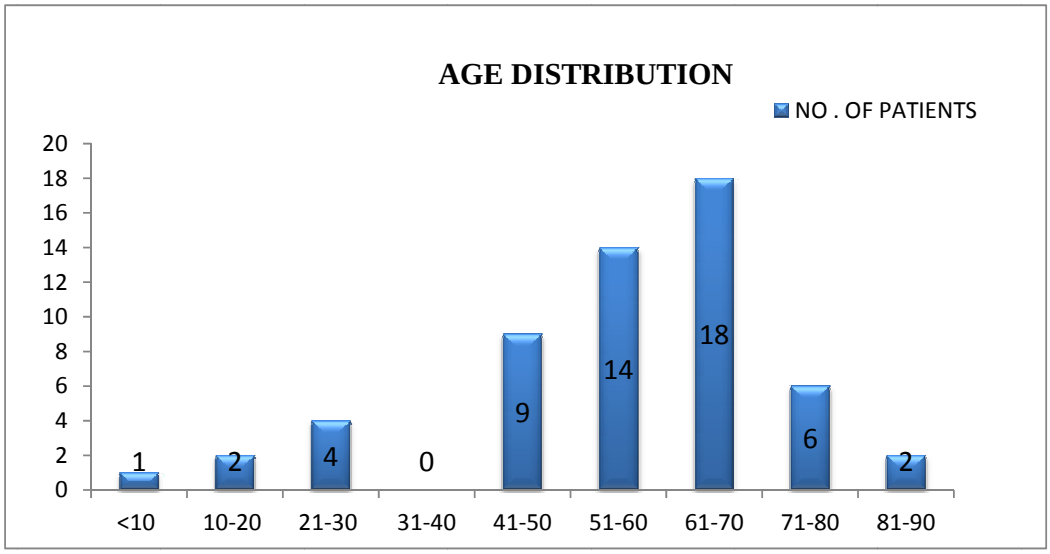


Table No. 3:- Time Interval Between Cataract Surgery And PCO Development

Time Interval (Years)	No. Of cases
> 6 Months	5
6 Months – 1 yr	13
2 - 3 yr	12
3 - 4 yr	9
3 – 4 yr	9
4 – 5 yr	3
> 5 yrs	7
Total	58

Above table shows, the mean time interval between cataract surgery and PCO development in our study was 13 patients(22.4%) developed PCO in 6 months -1 year ,12(20.68%) patients developed PCO in 1-2 years ,9(15.51%) patients had time interval of 2-3 years and 3-4 years , 3(5.17%) patients developed in 4-5years ,7 patients(12.06%) developed PCO more than 5 years and 5 developed PCO in less than 6 months .

Figure 3:- Shows time interval between cataract surgery and PCO development

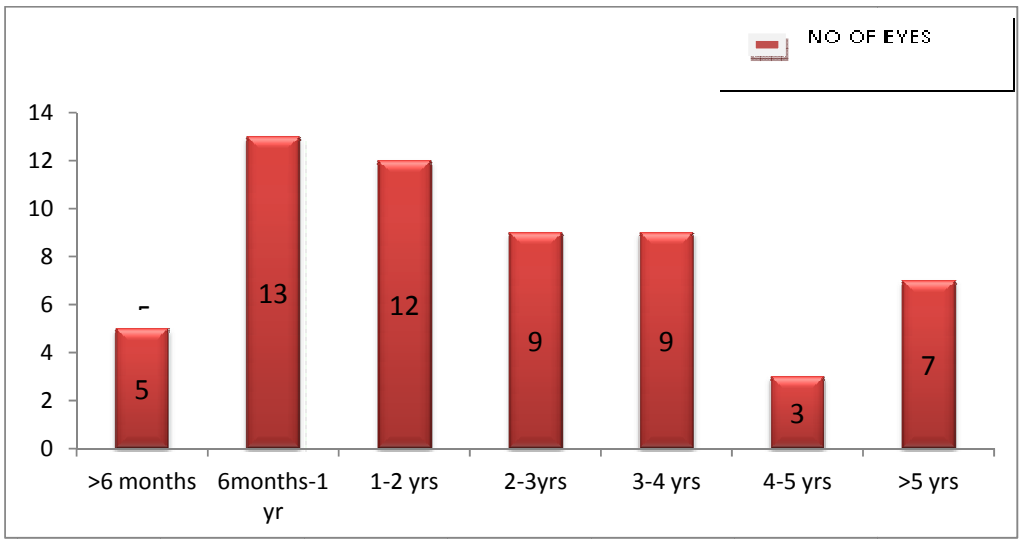


Table No.4:- Summary Of Energy Level Used For Capsulotomy In mjs

Energy level (mjs)	No of Eyes
0.5	1
1	13
2	3
3	41
4	0
5	0
Total	58

Table showing Energy level used was maximum of 3mjs in 41(70.68%) eyes. In 13 eyes (22.41%) energy of 1 mjs was used. In 3 eyes 2mjs was used and in one eye 0.5 mjs was used.

Figure 4:- Shows Energy level used For Capsulotomy in mjs

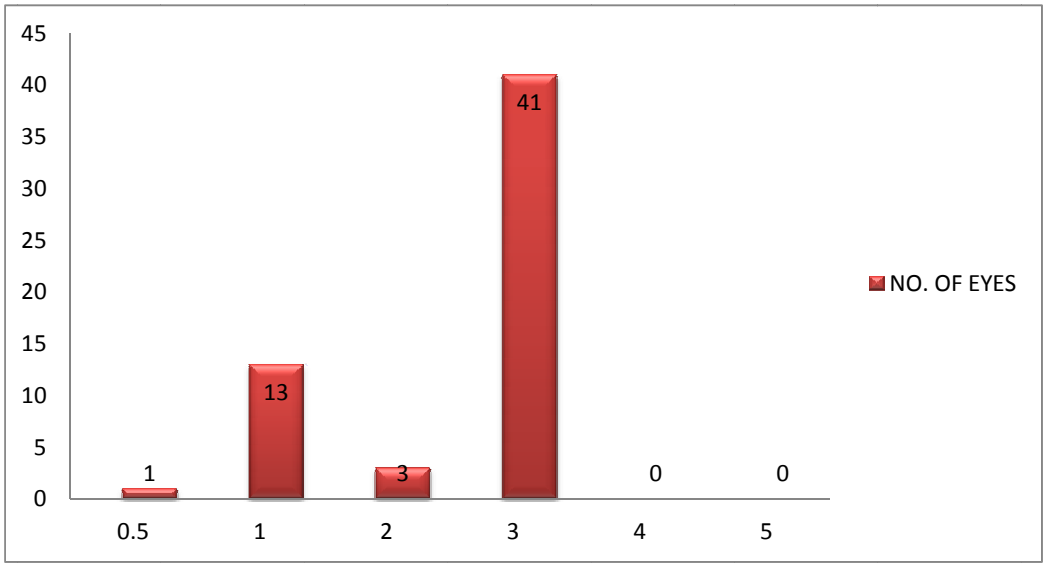


Table no. 5:- Showing Best Corrected Visual Acuity before YAG Laser

Best corrected visual acuity	No of Eye	% of Eyes
PL	1	1.72%
Hand movements	2	3.44%
Counting fingers	11	18.96%
6/60	18	31.03%
6/36	18	31.03%
6/24	3	5.17%
6/18 P	4	6.89%
6/6 P	1	1.72%
Total	58	100%

Results of table 5 showed patients having best corrected visual acuity ranged from PL to 6/6P. 6/60 and 6/36 vision was found in 18 eyes (31.03%) each. 3(5.17%) eyes had vision 6/24 and 4(6.89%) eyes had vision 6/18P. 11 (18.96%) eyes had CF(1 m to 3m), 2 (3.44%) eyes had HM and 1 (1.72%) eye had PL and 6/6P each.

Figure 5:- Shows BCVA before YAG laser.

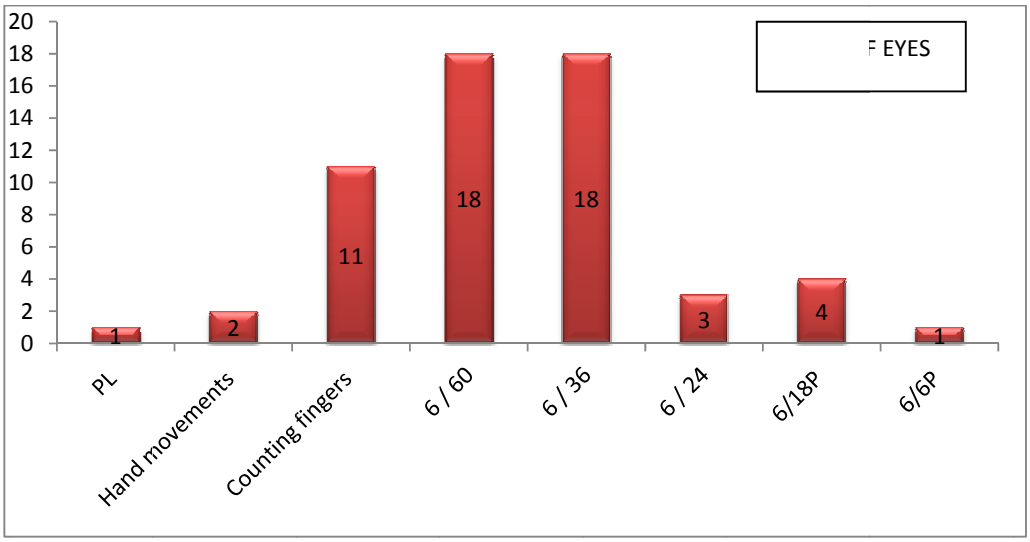


Table No 6:- Showing Best Corrected Visual Acuity after YAG Laser

Visual Acuity	No. of Patients	% Age
6/6	1	1.72%
6/9	4	6.29%
6/12	10	17.24%
6/18	17	29.31%
6/24	12	20.68%
6/36	7	12.06%
6/60	4	6.29%
Counting fingers	3	5.17%
Total	58	100%

In our study majority of eyes had shown improvement of 6/18 in 17 eyes (29.31%), 12 (20.68%) eyes showed improvement of 6/24 , 10 (17.24%) eyes showed improvement of 6/12 ,7 (12.06%) eyes showed improvement of 6/36.1 eye showed 6/6 improvement,4 eyes showed 6/9 improvement.

Figure 6:- Shows BCVA after YAG laser

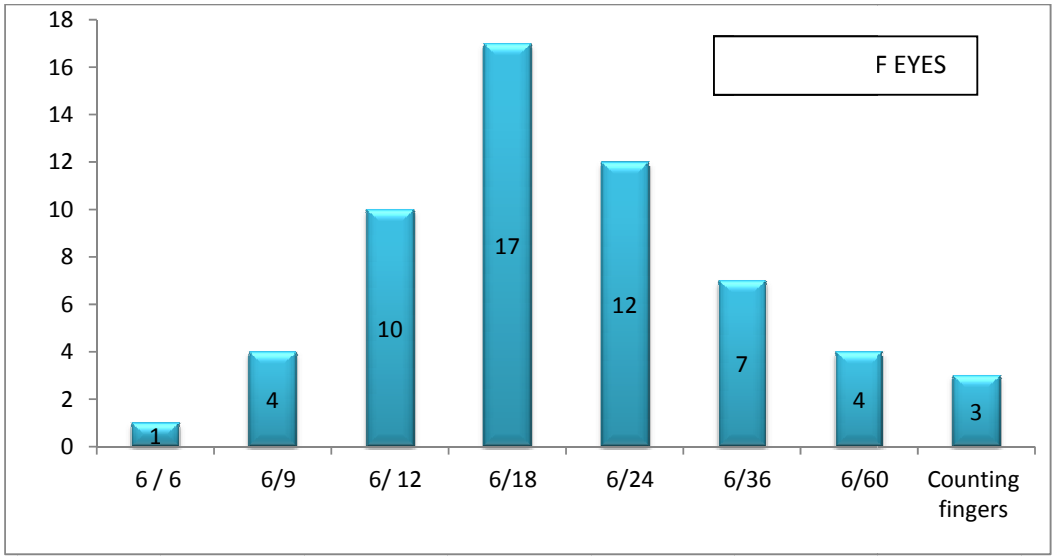


Table No 7:- Comparison Of Visual Acuity Before And After Laser.

Visual acuity	No. of patients before laser	No. of patients after laser
6/6	1	1
6/9	0	4
6/12	0	10
6/18	4	17
6/24	3	12
6/36	18	7
6/60	18	4
CF	11	3
HM	2	0
PL	1	0
TOTAL	58	58

Chi-square =48.768 and p<0.001, this p value showed highly statistically significant difference.

Out of 58 eyes, maximum number of eyes showed improvement in the range of 6/12 to 6/9. 17 eyes showed improvement of 6/18, 12 eyes showed BCVA 6/24, 10 eyes showed BCVA 6/12, 7 eyes showed improvement of 6/36, 4 eyes showed improvement of 6/9. One eye showed an improvement of 6/6.

Figure 7:- Comparison of Visual Acuity before and after laser

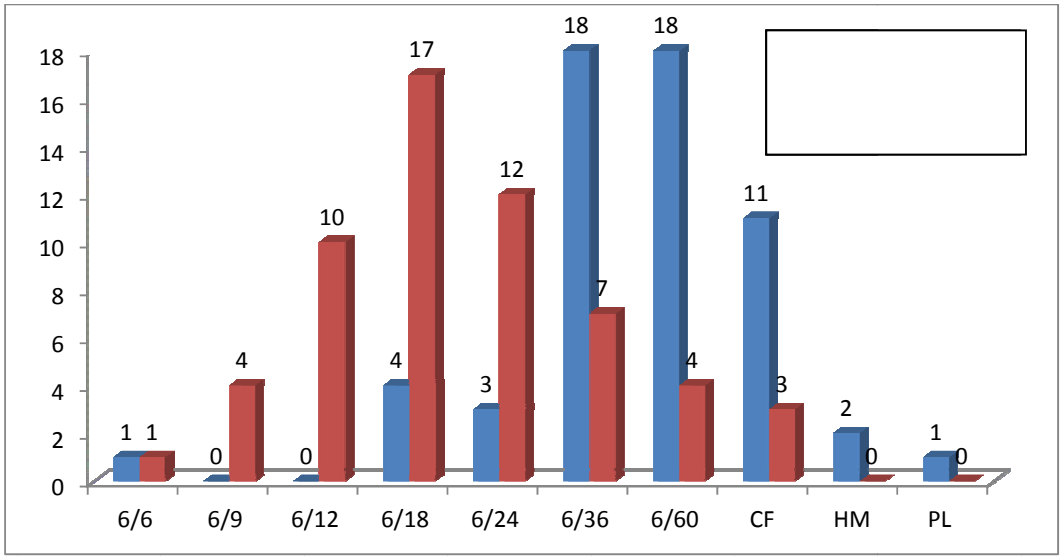


Table No 8a:- Fluctuation of IOP after 1 Month of Follow Up

Pre Laser IOP	IOP after 1 month	Fluctuation
12.2	14.6	2.4
17.3	21.9	4.6
14.6	17.3	2.7
12.2	14.6	2.4
12.2	13.4	1.2

t =4.844, p=0.0084, highly statistically significant.

In our study the IOP fluctuated in the range of 2-4 mm of Hg in the follow up after 1 month and after 3 months. On applying paired 't' test = 4.844 and p value =0.0084 after the follow up of 1 month, which was statistically highly significant

Figure 8a:-Fluctuation of IOP after 1 month

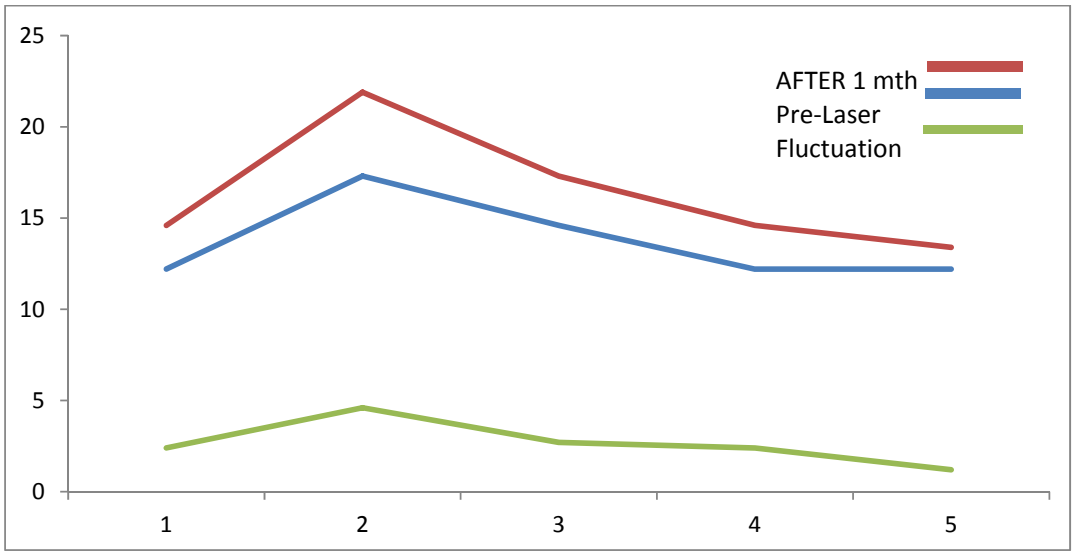


Table No 8b:- Fluctuation of IOP after 3 Months of Follow Up

Pre-Laser IOP	IOP after 3 months	Fluctuation
12.2	14.6	2.4
14.6	17.3	2.7

t=17.00, p=0.0374, statistically highly significant.

In our present study on applying the paired‘t’ test t = 17.00 and p value is 0.0374 after the follow up of 3 months which is highly statistically significant.

Figure 8b:-Fluctuation of IOP after 3 months.

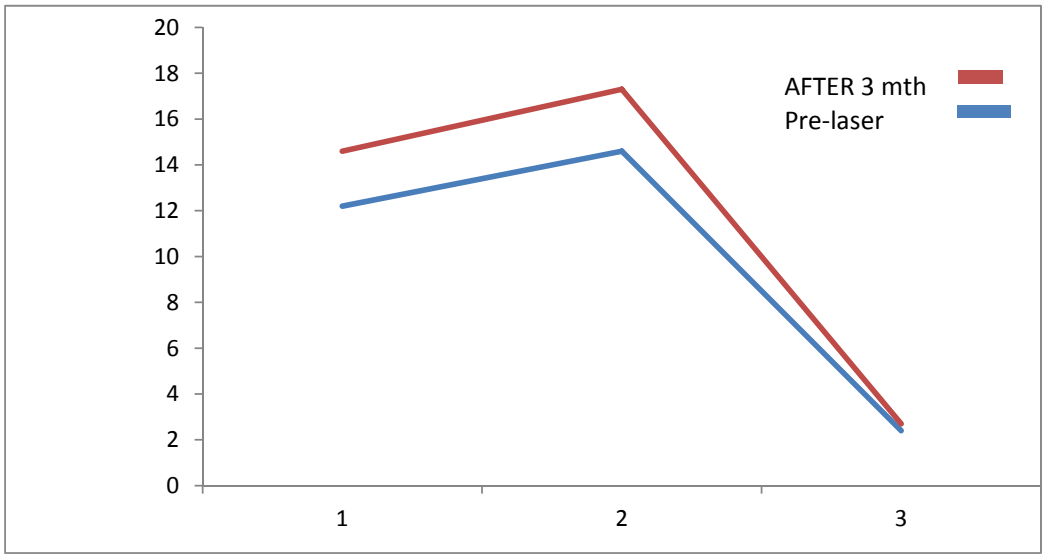
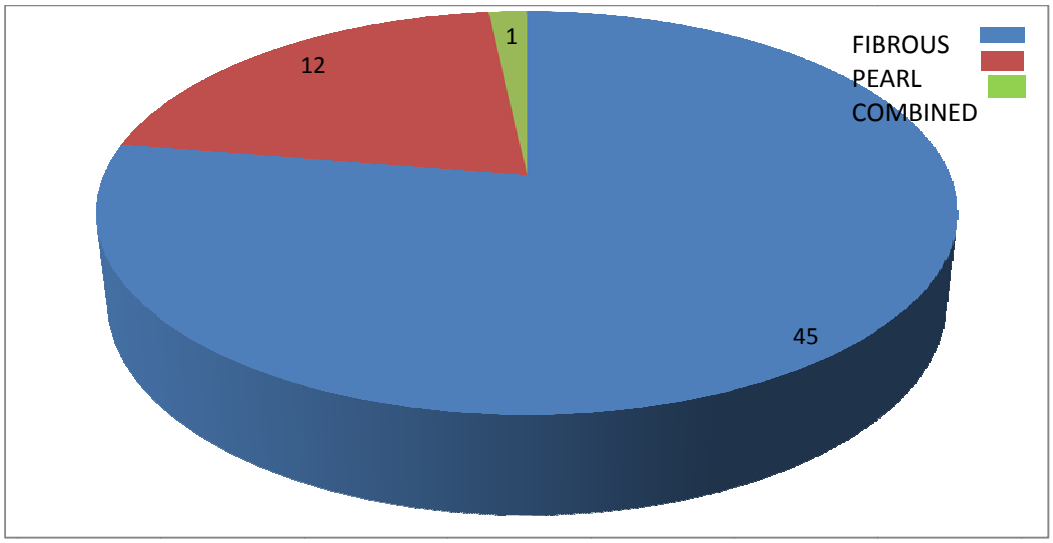


Table No 9:- Type of PCO

Type of PCO	No. of eyes	Percentage
Fibrous	45	77.58%
Pearl	12	20.68%
Combined	1	1.72%

In our study 45 (77.58%) eyes had fibrous type, 12 (20.6%) eyes had pearl type and 1 (1.72%) had combined type of PCO.

Figure 9:- Type of PCO



DISCUSSION

PCO is the most common post surgical complication following extra capsular cataract surgery and intraocular lens implantation. Nd: YAG laser availability has lead to an effective treatment for PCO.

In our study 58 eyes of 56 patients were evaluated. The ratio of male to female among patients who had significant PCO was found to be 1:1. Mian et al compared Nd: YAG capsulotomy rates in their study on 434 eyes of 329 patients who underwent cataract extraction and IOL implantation, reported that there was no gender distribution difference in Nd: YAG capsulotomy rates[33].

Out of 56 patients, 18 (32.14%) patients were in the group of 61-70 years of age and 14(25%) patients were in the group of age 51-60 years. Mian et al compared their study on 434 eyes of 329 patients reported that there was no difference in Nd: YAG capsulotomy rates when comparing age[35]. Wajeeha Rasool, Ali Raza et al showed that the mean age of the patients was 66 years $\pm$ 9.8, with a range of 50-85 years[33].

The mean time interval between cataract surgery and PCO development in our study was 6 months to 1 year in 13 patients(22.4%) ,12(20.68%) patients developed PCO in 1-2 years ,9(15.51%) patients had time interval of 2-3 years and 3-4 years before development of PCO, 3(5.17%) patients developed in 4-5years ,7 patients(12.06%) developed PCO more than 5 years and 5 developed PCO in less than 6 months. In our study the time interval between surgery and development of PCO ranged from less than 6 month to more than 5years. Apple et al reported that the time interval between surgery and PCO development varies widely with the range from 3 months to 4 years after surgery[34].

Mahtab Alam Khanzada, Shafi Muhammad Jatoi showed that the mean time interval between cataract surgery and Nd: YAG laser posterior capsulotomy was 2.5 years[35].

In our study the energy level was used in the range of 0.5 -3 mjs with a mean of 1.75 mjs. Mahtab Alam Khanzada et al reported the energy level required ranged from 1.5 to 5 mjs and mean was 3.2 mj[35]. Gore VS reported in 2012 that the laser power setting required is between 1 to 2.5 mjs or if mode is locked then between 3to5 mjs[36].

The study showed that 41(77.58%) eyes had developed fibrous type of PCO and 12(20.6%) eyes had developed pearl type of PCO. Rafiq M et al reported 62% eyes with fibrous type of PCO and 35%eyes with pearl type of PCO[37].

Out of 58 eyes, maximum number of 44(75.8%) eyes in our study, showed improvement in the range of 6/24 to 6/6. 17 eyes showed improvement of 6/18, 12 eyes showed BCVA 6/24, 10 eyes showed eyes showed BCVA 6/12, 7 eyes showed improvement of 6/36, 4 eyes showed improvement of 6/9. One eye showed an improvement of 6/6. After applying chi-square $p<0.001$ was obtained, which is statistically highly significant. Buehl et al investigated in a prospective interventional case series, the difference in the vision before and after Nd: YAG capsulotomy and reported the gain after Nd: YAG capsulotomy[38]. Wajeeha Rasool, Ali Raza et al showed that prior to Nd: YAG laser capsulotomy only 7% (14) patients had good best corrected visual acuity (6/18). After one week follow-up there was significant improvement of 6/18 in 73% patients[31]. Congdon et al reported improvement in best corrected visual acuity after Nd: YAG laser capsulotomy[39].

The present study showed IOL pitting in 1(1.72%) eye out of 58 eyes of 56 patients. Aurangzeb S, Faheemullah S, Jai R A et al found prevalence of 7% for IOL

damage during YAG laser posterior capsulotomy highest in group 3 than in group 1(4.49%), group 2(4.1%) and group 4 (1.27%)[40].

The present study showed the signs of iritis in 3 (5.17%) eyes out of 58 eyes of 56 patients in the follow up of 1 hr and 1 week following Nd: YAG posterior capsulotomy. Burq et al in a prospective study studied complications of laser capsulotomy on 104 eyes subjected to Nd: YAG capsulotomy for the treatment of PCO and noted iritis in 27.9% eyes[12]. Josef et al reported complications after Nd: YAG capsulotomy among which iritis was found in 1% of the eyes[41].

Intraocular pressure fluctuation after Nd: YAG capsulotomy has been well documented in earlier studies. In our study, 25(43.10%) eyes showed fluctuation of IOP in the range of 2-4 mm of Hg during the follow up in 1 hr, 1week, 1 month and 3 months.

However, it was found that the fluctuation of IOP was statistically not significant during 1 hr and 1 week of the follow up. The fluctuation of IOP during the follow up of 1 month and 3 months was statistically significant with p value of 0.0084 and 0.0374. Burq et al in a prospective study studied complications of laser capsulotomy on 104 eyes subjected to Nd: YAG capsulotomy for the treatment of PCO and noted transient rise in IOP-46.2% eyes[12]. J Ge, M Wand reported that the changes in IOP in eyes treated with capsulotomy were significantly higher than those in non capsulotomy eyes at each time interval following capsulotomy[42].

The study showed, the effectiveness of the procedure in the visual outcome which was statically highly significant, using chi-square test, with p value <0.001. This study also showed the effectiveness and minimal complications of Nd: YAG laser posterior capsulotomy in contrast to previous documented studies. Nd: YAG laser is the effective and universally accepted procedure for removal of posterior capsular opacity.

SUMMARY

The average rate of PCO is 75% and worked out sample was 57. 58 eyes of 56 patients were taken in the study. The aim of the study was to study the types of posterior capsular opacification on the basis of its morphology, visual outcome and complications after Nd: YAG laser capsulotomy. These cases were treated and followed up between Oct 2010 to April 2012. In this study 58 eyes of significant PCO were evaluated for visual acuity and IOP was recorded after 1 hr, 1 week, 1month and 3 months.

The present study showed M: F ratio is 1:1. Maximum number of the patients with significant PCO was in the age group of 61-70 years.

The time interval between cataract surgery and PCO development was found in the range of less than 6 months to more than 5 years. Energy level was used in the range of 0.5 to 3 mjs to treat PCO.

In our study visual acuity before Nd: YAG laser was in the range of PL to 6/18P, improvement in the visual acuity after laser in the range of CF to 6/6.

The present study also showed the signs of iritis in 3 eyes out of 58 eyes of 56 patients in the follow up of 1 hr and 1 week following Nd:YAG posterior capsulotomy. IOL pitting was found in 1 eye out of 58 eyes of 56 patients. Fluctuation of IOP in 25 eyes.

The study showed that 41(77.58%) eyes had developed fibrous type of PCO and 12(20.6%) eyes had developed pearl type of PCO.

The effectiveness of the procedure in the visual outcome which was statistically significant, using chi-square test = 48.768 and $p<0.001$.

This study also shows the effectiveness and reports minimal complications of Nd:YAG Laser posterior capsulotomy. Nd:YAG laser is the effective and universally accepted procedure for removal of posterior capsular opacity.

CONCLUSION

Posterior capsular opacification is the commonest cause of blindness and visual impairment in pseudophakic eyes with average rate of 75% in the hospital based study.

Multiple factors have been studied like Gender, Age, type of PCO, time interval between cataract surgery and PCO development, energy level. Visual improvement after YAG laser was in the range of 6/6 to 6/24. This outcome is also associated with minimum complications such as fluctuation in intraocular pressure, iritis and IOL pitting during the follow up period of the study.

Statistically significant visual outcome in terms of improved visual acuity was noted after Nd:YAG laser capsulotomy .Hence in conclusion Nd:YAG laser capsulotomy proved to be highly effective in improving visual acuity in eyes with PCO. Nd:YAG laser capsulotomy is easy ,non-invasive outpatient procedure, less time consuming and causes less discomfort.

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ANNEXURE – I

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT: A clinical study of posterior capsular opacification and its visual outcome after Nd:YAG laser capsulotopmy .

PRINCIPAL INVESTEGATOR: Dr. Varun Kumar
Postgraduate student,
Department of Ophthalmology
B.L.D.E. University's
Shri B.M. Patil Medical College &
Research Centre, Solapur Road,
BIJAPUR - 586103

PG GUIDE: Dr. Sunil G Biradar M.S.,
Professor of Ophthalmology,
B.L.D.E. University's
Shri B.M. Patil Medical College &
Research Centre, Solapur Road,
BIJAPUR - 586103

PURPOSE OF RESEARCH:

I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

This study is for better understanding and safety of Nd:YAG laser for the treatment of posterior capsular opacity .

PROCEDURE:

I understand that I will undergo Nd:YAG laser posterior capsulotomy.

BENEFITS:

I understand my participation in this study will help in the treatment of posterior capsular opacity with the help of Nd:YAG laser .

RISKS AND DISCOMFORTS:

I clearly understand the involved risk in the procedure.

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or

video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr. Varun Kumar is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.

And that a copy of this consent form will be given to me for keep for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Varun Kumar will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

_____	_____	_____
Date	Dr. Sunil G Biradar	Dr. Varun Kumar
	(Guide)	(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Varun Kumar has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

ANNEXURE - II

CASE SHEET PROFORMA

NAME: **AGE:** **SEX:** **O.P.NO:**

ADDRESS:

DATE OF EXAMINATION:

DATE OF PROCEDURE:

DATE OF SURGERY:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

PAST HISTORY:

FAMILY HISTORY:

TREATMENT HISTORY:

GENERAL PHYSICAL EXAMINATION:

LOCAL EXAMINATION: RE LE

EYE LIDS:

CONJUNCTIVA:

CORNEA:

ANTERIOR CHAMBER:

IRIS:

PUPIL:

LENS:

TYPE OF POSTERIOR CAPSULAR OPACITY:

INTRA OCULAR PRESSURE:

VISUAL ACUITY:

PIN HOLE:

FUNDUS EXAMINATION:

DIAGNOSIS:

TREATMENT:

Nd:YAG LASER :

ENERGY USED:

NUMBERS OF SHOTS:

POST LASER MEDICATION:

1. Tab Acetazolamide 250 mg yes/no
2. Antibiotic steroid eye drops yes/no
3. NSAIDS Eye drops q.i.d yes/no
4. Any other treatment yes/no

FOLLOW UP VISION	1 hr	1week	1month	3 months
		RE		LE
INTRA OCULAR PRESSURE:				

SLIT LAMP EXAMINATION:

FUNDUS EXAMINATION:

INTRAOCULAR PRESSURE

INTRAOCULAR CRACK

INTRAOCULAR LENS PITTING

IRIS BLEED

IRITIS

VITRITIS

REOPACITY

RETINAL DETACHMENT

CYSTOID MACULAR EDEMA

ANNEXURE - III

COLOUR PLATES

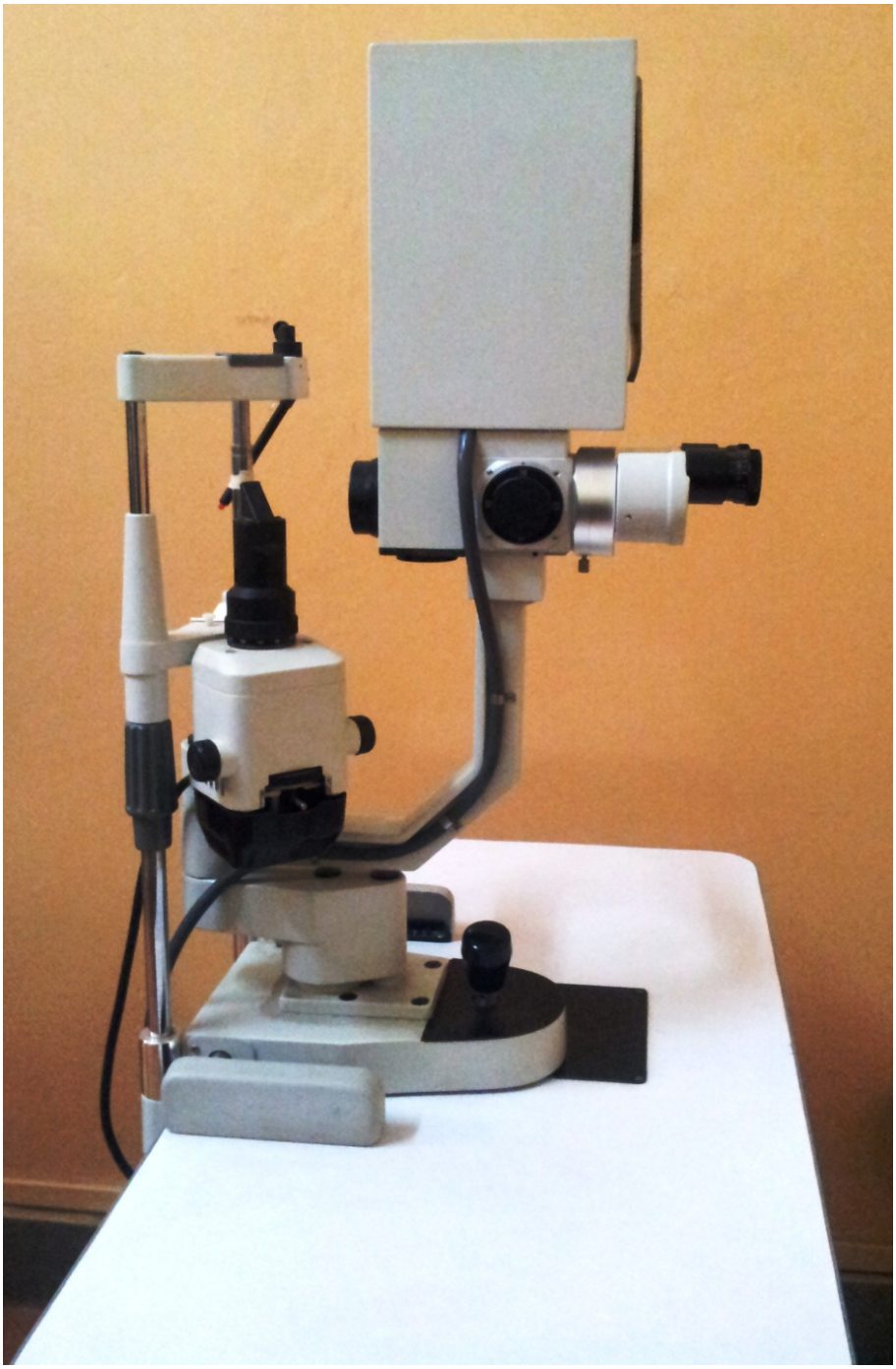

FIG. 1 Nd:YAG LASER

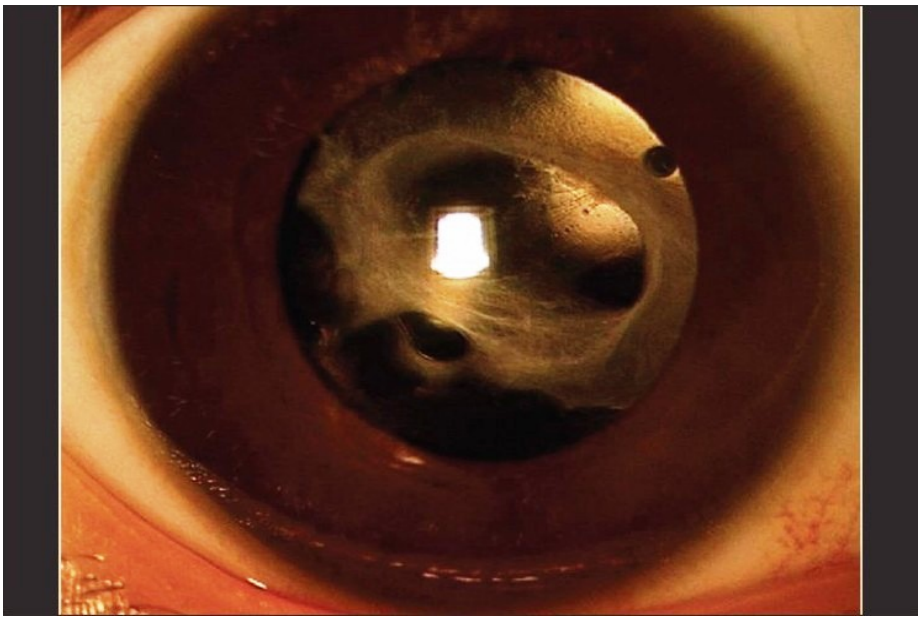

FIG.2 FIBBROUS TYPE PCO

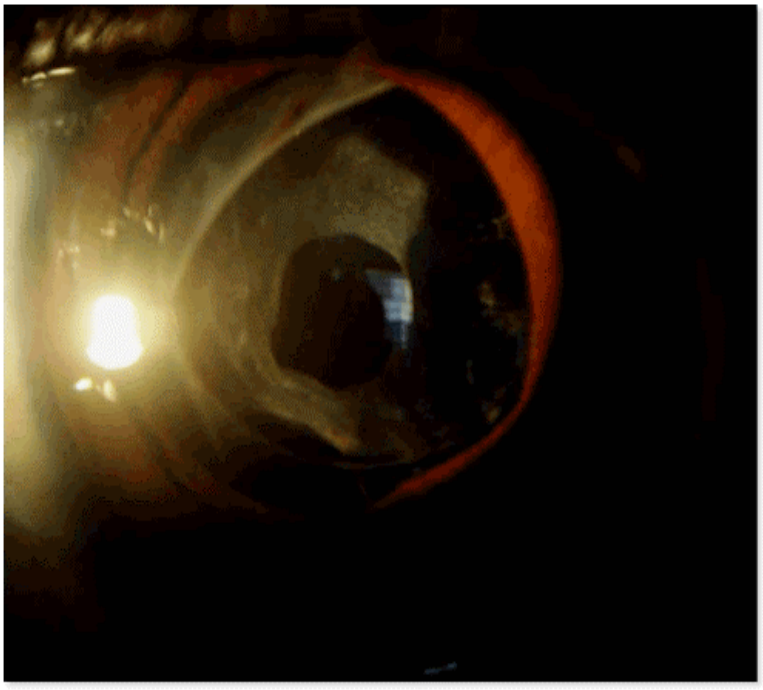

FIG. 3 FIBBROUS TYPE PCO AFTER Nd:YAG CAPSULOTOMY

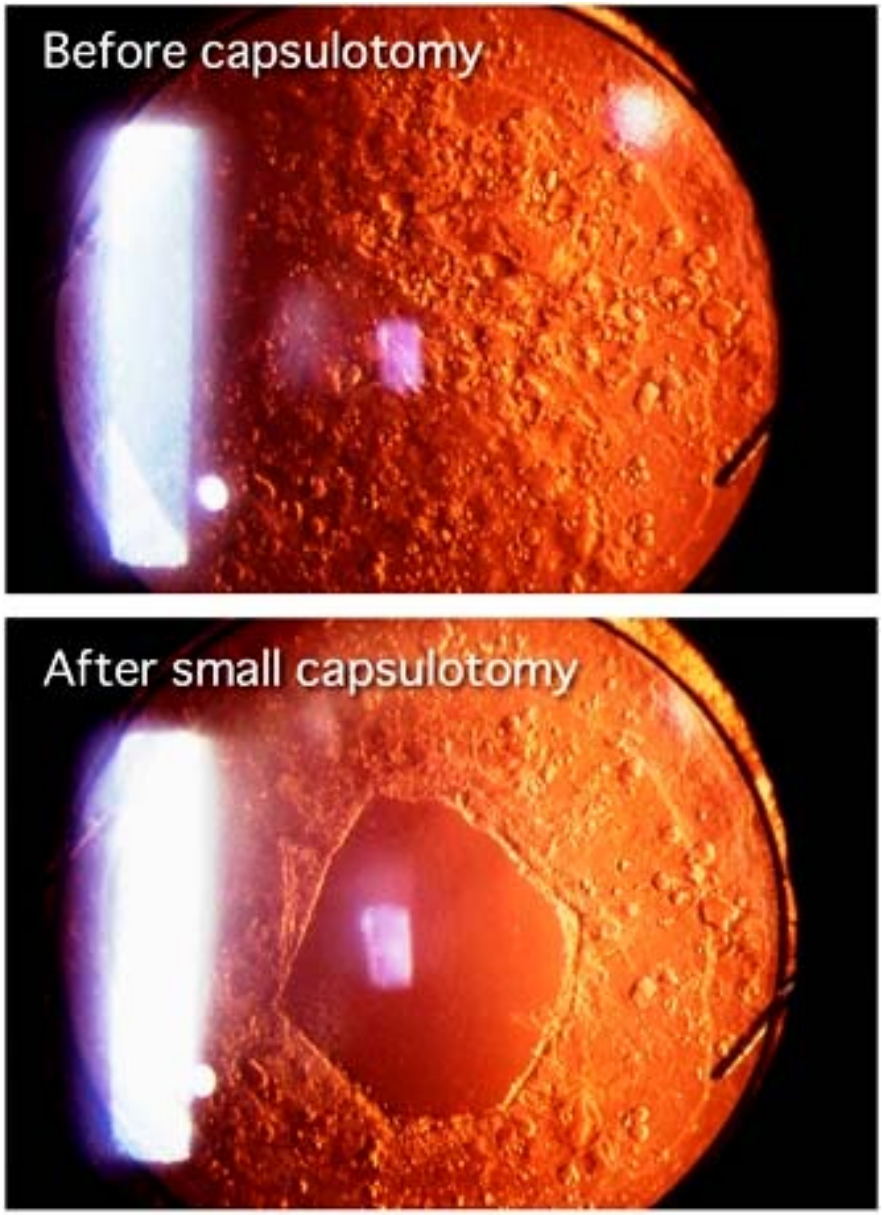

FIG. 4 PEARL TYPE PCO BEFORE AND AFTER Nd:YAG CAPSULOTOMY

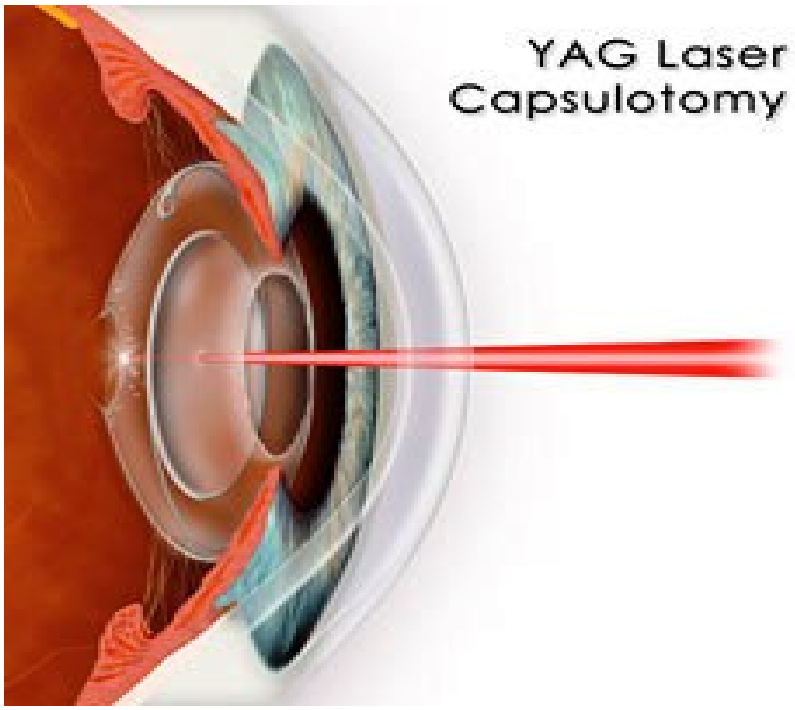

FIG. 5 YAG LASER CAPSULOTOMY

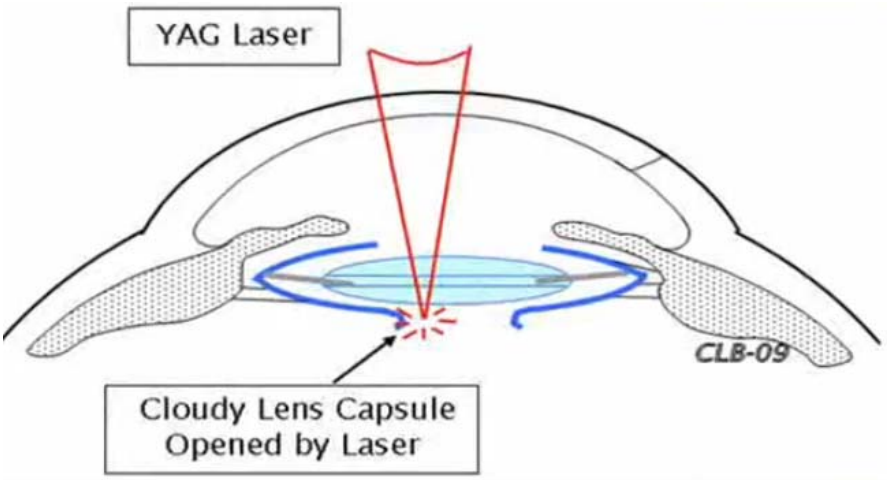

FIG. 6 YAG LASER

COMPLICATIONS

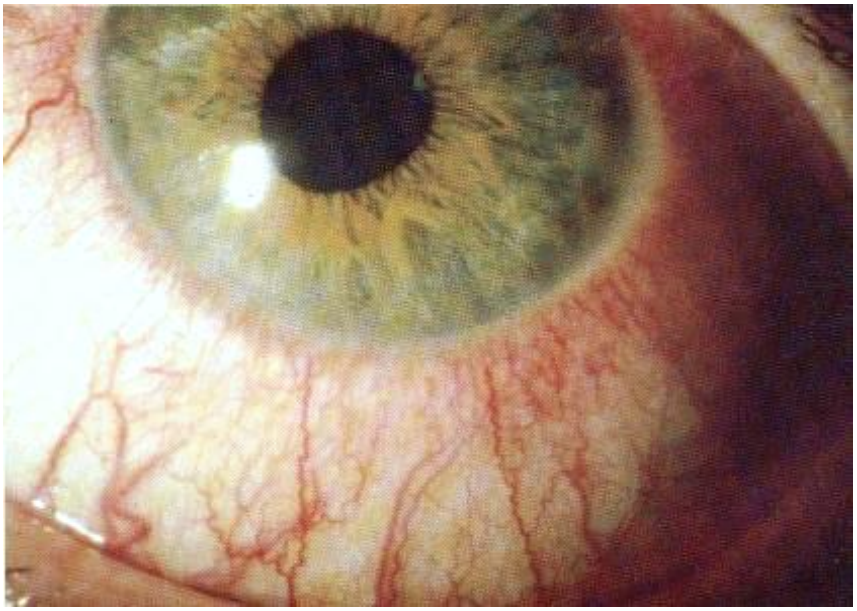

FIG.7 IRITIS

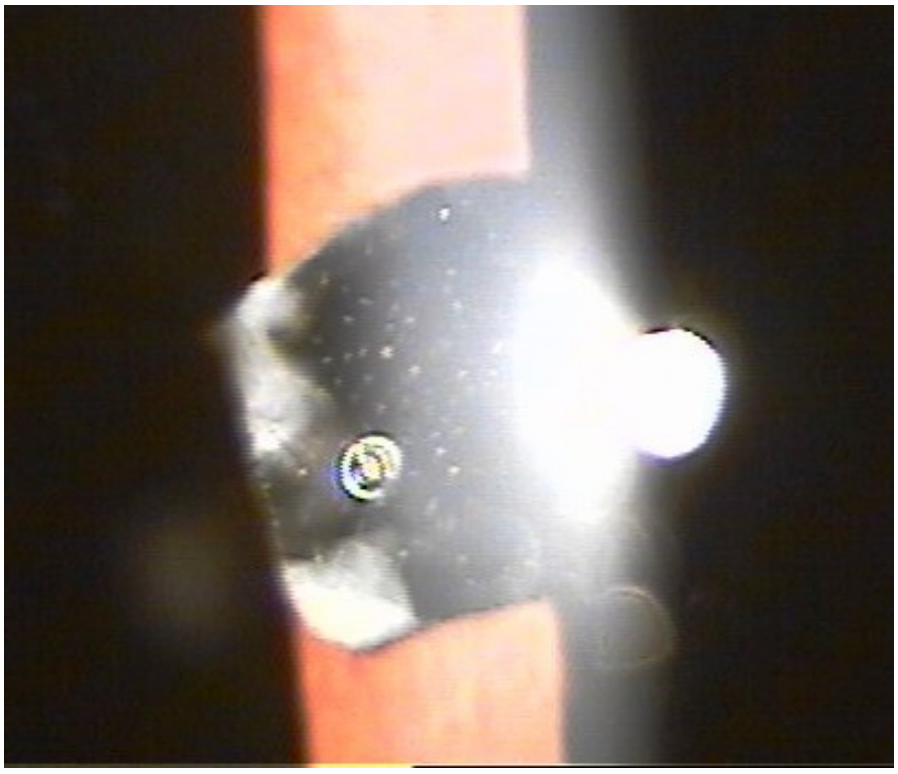

FIG. 8 IOL PITTING

ANNEXURE –IV

KEYS FOR MASTER CHART

1. Nd: YAG laser-Neodymium Yttrium Aluminium Garnet.

2. PCO-Posterior Capsular Opacity

3. VA-Visual Acuity

4. IOP-Intra ocular pressure

5. IOL-Intraocular Lens

6. RD-Retinal Detachment

7. CME-Cystoid macular edema.

MASTER CHART

Sl. No.	Name	Age/ Sex	OP. NO.	Eye	Type of PCO	Date of Surgery	Pre-laser VA	Energy	Pre-Laser IOP	Follow up 1hr		Follow up after 1wk		Follow up after 1mth		Follow up after 3 month		Complications of IOL		Complication of YAG laser							
										VA	IOP	VA	IOP	VA	IOP	VA	IOP	IOL Pitting	IOL Crack	Iritis	Raise in IOP	Hyphaema	Vitritis	RD	CME	Iris bleed	Reopacity
1	H.B Dodamani	50/M	247871	LE	Fibrous	6/05/08	CF3mts	3	12.2	6/36P	12.2	6/36P	12.2	6/36	12.2	6/36	12.2	A	A	A	A	A	A	A	A	A	A
2	Shivagangamma	60/F	265458	RE	Pearl	3/8/09	6/60	3	14.6	6/24P	14.6	6/24	14.6	6/18P	14.6	6/18	14.6	A	A	A	A	A	A	A	A	A	A
3	Lakshmibai	60/F	281631	LE	Fibrous	10/10/07	6/24	3	17.3	6/18P	17.3	6/18P	17.3	6/18	17.3	6/18	17.3	A	A	A	A	A	A	A	A	A	A
4	Santosh Biradar	12/M	287652	RE	Fibrous	7/6/08	6/60	3	14.6	6/36P	12.2	6/36	14.6	6/24	12.2	6/24	12.2	A	A	A	A	A	A	A	A	A	A
5	Urmesh	48/M	298630	RE	Fibrous	5/10/06	6/36	3	14.6	6/18P	14.6	6/12P	14.6	6/12	14.6	6/12	14.6	A	A	A	A	A	A	A	A	A	A
6	B A Shelidar	57/M	297923	RE	Pearl	3/10/09	6/60	3	17.3	6/18P	14.6	6/18	17.3	6/18	14.6	6/18	14.6	A	A	A	A	A	A	A	A	A	A
7	Payangouda	75/M	300620	LE	Fibrous	10/10/05	6/60P	3	17.3	6/12	14.6	6/9P	17.3	6/9P	17.3	6/9P	17.3	A	A	A	A	A	A	A	A	A	A
8	Payangouda	75/M	300620	RE	Fibrous	10/12/06	6/60P	3	14.6	6/36	14.6	6/24P	14.6	6/24	14.6	6/24	14.6	A	A	A	A	A	A	A	A	A	A
9	Neelamma	68/F	377910	LE	Fibrous	13/1/10	6/36P	3	17.3	6/18P	12.2	6/18	14.6	6/18	12.2	6/18	14.6	A	A	A	A	A	A	A	A	A	A
10	J A Inamdar	29/M	113827	LE	Fibrous	10/2/09	6/36	3	14.6	6/18P	14.6	6/18	14.6	6/18	14.6	6/18	14.6	A	A	A	A	A	A	A	A	A	A
11	Guduma	75/M	115563	RE	Fibrous	3/2/04	6/36P	3	14.6	6/24	14.6	6/18P	14.6	6/18	14.6	6/18	14.6	A	A	A	A	A	A	A	A	A	A
12	S M Biradar	60/M	119134	LE	Fibrous	20/4/02	CF3mts	3	14.6	6/60	14.6	6/36P	14.6	6/36	14.6	6/36	14.6	A	A	A	A	A	A	A	A	A	A
13	Fatima	45/F	120514	RE	Fibrous	20/4/10	6/36P	3	14.6	6/24	14.6	6/18	14.6	6/18	12.2	6/18	14.6	A	A	A	A	A	A	A	A	A	A
14	Verendra pattar	24/M	234456	LE	Fibrous	12/5/07	HM	3	14.6	CF2mts	12.2	CF2mts	12.2	CF 2mts	12.2	CF 3mts	12.2	A	A	P	A	A	A	A	A	A	A
15	Bhuveneshwari	17/F	89348	LE	Fibrous	5/7/10	6/6P	3	17.3	6/6P	17.3	6/6	17.3	6/6	17.3	6/6	17.3	A	A	A	A	A	A	A	A	A	A
16	Amenabi	65/F	115464	LE	Combined	10/7/05	6/60P	3	14.6	6/24P	14.6	6/24	14.6	6/18P	14.6	6/18P	14.6	A	A	A	A	A	A	A	A	A	A
17	Nimbewwa	65/F	139413	RE	Pearl	5/04/10	6/60P	3	17.3	6/36P	17.3	6/36	17.3	6/36	17.3	6/36	17.3	A	A	A	A	A	A	A	A	A	A
18	Nimbewwa	65/F	139413	LE	Fibrous	2/5/07	HM	3	12.2	CF2mts	14.6	CF2mts	14.6	CF3mts	14.6	CF3mts	14.6	A	A	A	P	A	A	A	A	A	A
19	Bhageerati	76/F	141344	RE	Pearl	10/5/02	6/18P	3	17.3	6/12P	17.3	6/12	17.3	6/12	17.3	6/9P	17.3	A	A	A	A	A	A	A	A	A	A
20	Mahadevi	70/F	115594	RE	Pearl	14/8/06	CF3mts	3	20.6	6/60P	20.6	6/36P	20.6	6/36	17.3	6/24P	20.6	A	A	A	A	A	A	A	A	A	A
21	B B Patil	78/F	145019	RE	Fibrous	15/5/10	6/36P	3	17.3	6/24P	30.4	6/24	30.4	6/18	21.9	6/18	12.2	A	A	A	P	A	A	A	A	A	A
22	Basappa	88/M	147609	RE	Fibrous	10/1/08	6/36	3	17.3	6/9P	17.3	6/9	17.3	6/9	17.3	6/9	17.3	A	A	A	A	A	A	A	A	A	A
23	Bheemappa	65/M	147611	RE	Fibrous	3/3/8	6/60	3	17.3	6/18P	17.3	6/18	17.3	6/18	17.3	6/18	17.3	A	A	A	A	A	A	A	A	A	A
24	Shivaputaraya	70/M	152356	LE	Fibrous	28/5/10	6/60	3	14.6	6/18P	12.2	6/18	14.6	6/18	12.2	6/18	12.2	A	A	A	A	A	A	A	A	A	A
25	Basavant raj	60/M	154927	LE	Fibrous	6/6/9	6/36P	3	17.3	6/12P	14.6	6/12	14.6	6/12	14.6	6/12	14.6	A	A	A	A	A	A	A	A	A	A
26	Jaytubai	60/F	165367	LE	Fibrous	8/6/10	6/60P	3	17.3	6/18P	17.3	6/18	17.3	6/18	17.3	6/18	17.3	A	A	A	A	A	A	A	A	A	A
27	Padmavati	65/F	169143	RE	Pearl	7/7/05	CF3mts	3	14.6	6/36P	14.6	6/36	17.3	6/24P	14.6	6/24P	14.6	A	A	A	P	A	A	A	A	A	A
28	Shaniabai	70/F	171006	LE	Fibrous	16/4/09	6/36	3	17.3	6/18P	14.6	6/18	14.6	6/18	14.6	6/18	14.6	A	A	A	A	A	A	A	A	A	A
29	Shivagondappa	60/M	171334	RE	Fibrous	6/4/06	PL+/PR+	3	21.9	CF3mts	25.8	CF 2mts	25.8	CF 2mts	20.6	CF 2mts	13.4	A	A	A	P	A	A	A	A	A	A
30	Suvarna	55/F	166278	LE	Pearl	24/6/07	6/24P	3	12.2	6/12P	12.2	6/12	12.2	6/12	12.2	6/12	12.2	A	A	A	A	A	A	A	A	A	A
31	Devendra	45/M	15333	RE	Pearl	26/7/10	6/36	3	14.6	6/12P	17.3	6/12	17.3	6/12	17.3	6/12	17.3	A	A	P	P	A	A	A	A	A	A
32	Vajantha	55/F	181110	RE	Fibrous	14/8/09	6/24	3	14.6	6/18P	14.6	6/18	14.6	6/18	14.6	6/12P	14.6	A	A	A	A	A	A	A	A	A	A

33	Gangamma	60/F	183411	RE	Fibrous	3/8/07	CF3mts	3	17.3	6/60P	17.3	6/60	17.3	6/60	17.3	6/60	17.3	A	A	A	A	A	A	A	A	A	A
34	Kamalabai	45/F	183740	LE	Pearl	12/8/09	CF3mts	3	17.3	6/24P	14.6	6/24	17.3	6/24	14.6	6/24	14.6	A	A	A	A	A	A	A	A	A	A
35	I R patil	83/M	192879	RE	Fibrous	6/3/11	6/36P	3	14.6	6/12	14.6	6/12	14.6	6/12	14.6	6/12	14.6	A	A	A	A	A	A	A	A	A	A
36	Itabai	60/F	16424	LE	Fibrous	11/8/07	CF2mts	3	12.2	6/36P	12.2	6/36	12.2	6/36	14.6	6/36	12.2	A	A	A	A	A	A	A	A	A	A
37	Siddappa	64/M	198857	RE	Fibrous	15/6/08	6/60P	3	17.3	6/24P	12.2	6/24	12.2	6/18	13.4	6/18	12.2	A	A	A	A	A	A	A	A	A	A
38	Parvati	50/F	218474	LE	Fibrous	20/7/8	6/60P	0.5	12.2	6/36P	12.2	6/36	12.2	6/24	12.2	6/24	12.2	A	A	A	A	A	A	A	A	A	A
39	Shantesh	27/M	236755	RE	Fibrous	5/5/09	6/36P	1.5	17.3	6/24P	17.3	6/24P	17.3	6/24	14.6	6/18P	17.3	A	A	A	A	A	A	A	A	A	A
40	Bhimaraya	66/M	22567	LE	Fibrous	5/3/3	6/36P	3	14.6	6/24P	14.6	6/24P	14.6	6/24P	14.6	6/24P	14.6	A	A	A	A	A	A	A	A	A	A
41	Tamanna	60/M	163033	RE	Fibrous	14/2/09	CF1mts	1.5	14.6	6/9P	14.6	6/9	14.6	6/9	12.2	6/9	12.2	A	A	A	A	A	A	A	A	A	A
42	Kashibai	65/F	155648	RE	Fibrous	3/3/11	CF2.5mts	1.5	17.3	6/24P	13.4	6/24P	13.4	6/24P	12.2	6/24P	12.2	A	A	A	A	A	A	A	A	A	A
43	Siddamma	48/F	154379	LE	Fibrous	5/3/09	6/60P	3	14.6	6/18P	14.6	6/18P	14.6	6/18P	14.6	6/18P	14.6	A	A	A	A	A	A	A	A	A	A
44	Bassappa	56/M	103329	RE	Pearl	15/4/09	6/60	2	17.3	6/18P	17.3	6/18	17.3	6/12P	17.3	6/12P	17.3	A	A	A	A	A	A	A	A	A	A
45	Shanta bai	60/F	129992	LE	Fibrous	6/10/10	6/36P	1.4	17.3	6/18	17.3	6/18	17.3	6/18	13.4	6/18	17.3	A	A	A	A	A	A	A	A	A	A
46	Jana bai	50/F	130304	RE	Fibrous	10/02/08	6/60	2	12.2	6/12P	12.2	6/12P	12.2	6/12P	12.2	6/12P	12.2	A	A	A	A	A	A	A	A	A	A
47	Yellapa	65/M	134284	LE	Fibrous	13/2/11	6/18P	3	17.3	6/9P	17.3	6/9P	17.3	6/9	17.3	6/9	17.3	A	A	A	A	A	A	A	A	A	A
48	Subhash kulkarni	68/M	111948	RE	Fibrous	15/11/11	6/36P	3	17.3	6/18P	12.2	6/18P	12.2	6/18P	13.4	6/18	12.2	A	A	A	A	A	A	A	A	A	A
49	Siddanna	61/M	29767	LE	Fibrous	12/1/04	6/60P	2	13.4	6/12	13.4	6/12	13.4	6/12	13.4	6/12	13.4	A	A	A	A	A	A	A	A	A	A
50	Amit kamba	7/M	134560	RE	Fibrous	18/4/10	6/60P	1.4	-	6/60P		6/60P		6/60		6/60	-	A	A	P	A	A	A	A	A	A	A
51	Bassappa	71/M	115596	LE	Fibrous	24/4/11	6/36	1.5	17.3	6/18	17.3	6/18	17.3	6/18	17.3	6/18	17.3	A	A	A	A	A	A	A	A	A	A
52	Noorjan	55/F	134699	RE	Fibrous	16/2/12	6/36P	1.4	14.6	6/24P	14.6	6/24P	12.2	6/24	14.6	6/24	14.6	P	A	A	A	A	A	A	A	A	A
53	Naagubai	50/F	123889	LE	Fibrous	9/6/11	CF1mts	1.5	12.2	6/12P	12.2	6/12P	12.2	6/12	13.4	6/12	12.2	A	A	A	A	A	A	A	A	A	A
54	Sabhudra	75/F	150319	RE	Pearl	12/10/11	6/18P	1.4	14.6	6/9P	14.6	6/9	14.6	6/9	13.4	6/9	14.6	A	A	A	A	A	A	A	A	A	A
55	Dareyappa	21/M	148843	RE	Fibrous	25/5/02	6/18P	1.5	14.6	6/12	14.6	6/12	14.6	6/12	14.6	6/12	14.6	A	A	A	A	A	A	A	A	A	A
56	Irabai	67/F	162762	LE	Pearl	12/5/10	CF1mts	1.4	17.3	6/24P	17.3	6/24	17.3	6/18	13.4	6/18	17.3	A	A	A	A	A	A	A	A	A	A
57	Chandamma	65/F	143925	RE	Fibrous	25/8/9	6/60P	1.5	12.2	6/18	12.2	6/18	12.2	6/18	12.2	6/18	12.2	A	A	A	A	A	A	A	A	A	A
58	Kushma	68/F	139875	RE	Fibrous	26/5/08	6/36P	1.5	14.6	6/18	14.6	6/18	14.6	6/18	14.6	6/18	14.6	A	A	A	A	A	A	A	A	A	A