"VISUAL OUTCOMES AND COMPLICATIONS AFTER NEODYNIUM-DOPED YTTRIUM ALUMINIUM GARNET (ND YAG) LASER CAPSULOTOMY IN POSTERIOR CAPSULAR OPACIFICATION"

By

DR. GONA SOUJANYA

Dissertation Submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH TAMAKA, KOLAR
In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY
IN
OPHTHALMOLOGY

Under the guidance of

DR. KANTHAMANI.K M.B.B.S., M.S.,

DEPARTMENT OF OPHTHALMOLOGY

SRI DEVARAJ URS MEDICAL COLLEGE

TAMAKA, KOLAR.

(APRIL – 2019)

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**VISUAL OUTCOMES AND COMPLICATIONS AFTER NEODYNIUM DOPED YTTRIUM ALUMINUM GARNET LASER CAPSULTOMY IN POSTERIOR CAPSULAR OPACIFICATION**" is a bonafide and genuine research work carried out by me under the guidance of DR.KANTHAMANI,M.S. Professor and H.O.D Department of ophthalmology, Sri Devaraj Urs Medical College, kolar , Karnataka.

DATE: SIGNATURE OF THE CANDIDATE

PLACE: **DR.GONA SOUJANYA**

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled

"VISUAL OUTCOMES AND COMPLICATIONS AFTER NEODYNIUM DOPED YTTRIUM ALUMINUM GARNET LASER CAPSULOTOMY IN POSTERIOR CAPSULAR OPACIFICATION"

is a bonafide research work done by

DR.GONA SOUJANYA

Under my guidance and supervision,
in partial fulfilment of the requirement for the degree of

M.S.OPHTHALMOLOGY.

DATE:
PLACE:

SIGNATURE OF THE GUIDE
DR.K.KANTHAMANI,*M.S*
Professor and H.O.D.
Department of ophthalmology
S.D.U.M.C, Kolar.

ENDORSEMENT BY THE HOD, PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"VISUAL OUTCOMES AND COMPLICATIONS AFTER NEODYNIUM DOPED YTTRIUM ALUMINUM GARNET LASER CAPSULOTOMY IN POSTERIOR CAPSULAR OPACIFICATION"** is a bonafide and genuine research work carried out by **DR.GONA SOUJANYA** under the guidance of **DR. K. KANTHAMANI,** M.S, Professor and H.O.D, Department of Ophthalmology, Sri Devaraj Urs Medical College.

Signature of HOD
Dr.K.Kanthamani.
Professor and H.O.D.
Department of ophthalmology.
Sri Devaraj urs medical college
Kolar, Karnataka

DATE:

PLACE: KOLAR

Signature of Principal
Dr.M.L.Harendra Kumar,M.D.
Principal
Sri devaraj urs medical college
Kolar, Karnataka

DATE:

PLACE: KOLAR

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION &

RESEARCH, TAMAKA

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that Sri Devaraj Urs Academy of Higher Education and Research, Kolar shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic purpose.

Date:

Place:

DR.GONA SOUJANYA
Post Graduate student
Department of Ophthalmology
Sri Devaraj Urs Medical College

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH

ETHICAL COMMITTEE CERTIFICATE

This is to certify , the ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved, **DR.GONA SOUJANYA** Post Graduate student of ophthalmology at Sri Devaraj Urs Medical College, Kolar to take up the dissertation work titled **"VISUAL OUTCOMES AND COMPLICATIONS AFTER NEODYNIUM DOPED YTTRIUM ALUMINUM GARNET LASER CAPSULOTOMY IN POSTERIOR CAPSULAR OPACIFICATION"** to be submitted to Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka.

Signature of Member Secretary

Signature and Seal of the Principal

SDUAHER

Sri Devaraj Urs Academy of Higher Education and Research

Certificate of Plagiarism Check

Author Name	Dr.Gona Soujanya
Course of Study	Synopsis / Thesis / Dissertation
Name of Supervisor	DR. KANTHAMANI
Department	OPHTHALMOLOGY
Acceptable Maximum Limit	10%
Submitted By	librarian@sduu.ac.in
Paper Title	visualnoutcomes and complications after neodynium doped yttrium aluminium garnet laser capsulotomy in posterior capsular opacificatio
Similarity	07 %
Paper ID	181201031623
Submission Date	2018-12-01 03:16:23

* This report has been generated by DrillBit Anti-Plagiarism Software

G. Soujanya	
Signature of Student	Signature of Supervisor
Head of the Department	
University Librarian	Post Graduate Director

Senior Librarian, 1/12/18
Library and Information Centre
Sri Devaraj Urs Medical College
Tamaka, KOLAR-563 101.

ACKNOWLEDGEMENT

*First and foremost I would like to thank the almighty "**Saibaba"** for giving me strength at everystep of my life. Without my faith in you, I would not have been where iam now & I would have never got through it.*

Though I take the credit of being the author of this dissertation, there are many people who have contributed to it. I owe my gratitude to all those people who made this dissertation possible &because of whom my post graduate experience has been the one I will cherish forever.

*I would like to express my sincere gratitude to my guide **DR.KANTHAMANI**, professor & HOD in the department of ophthalmology, SDUMC, Kolar, for her continuous support, patience, motivation, and immense knowledge. Her precious advice on both the dissertation as well as the path of my career has been priceless. I could not have imagined having a better advisor & mentor. I thank you mam for correcting all the mistakes of mine and answering to my silliest questions at times.*

*I would also like to thank **DR.M.S.PADMAJOTHI** and **DR.MOHAN KUMAR,** my professors, Department of ophthalmology, Sri Devaraj Urs Medical College, Kolar. Without your support, initiative and constant encouragement this study would not have been possible.*

*I would like to express my heartful thanks to my Assistant Professors, **DR. SANGEETHA T, DR. USHA B.R., DR. RASHMI, DR. INCHARA, DR. CHAITRA,** Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamaka, kolar for their help and suggestions rendered to me during this study. I thank all my teachers throughout my life for having made me what I am today.*

*My gratitude and thanks to **DR. M.L. HARENDRA KUMAR**, Principal, Sri Devaraj Urs Medical College, Tamaka, kolar, for letting me use the college and hospital facilities and resources.*

*I would like to specially thank **DR.HARISH, DR.NUTHAN** and **DR.MEGHANA** for all their help during this study and making my journey through it smooth & joyful.*

*Words cannot express how grateful I am to my parents, **MRS.GONA JHANSI & MR.GONA SATYANARAYANA**, for all the sacrifices that they have made on my behalf. Their prayer for me is what has sustained me this far. I would like to thank my husband **K.TEJ KIRAN** for his never ending love, patience and immense encouragement which enabled me to complete this work succesfully and my in-laws **K.VENKATESWARA RAO & K.PADMAVATHI** for their constant support and encouragement, without whom my journey wouldn't have been so smooth.*

*I would like to thank my sister **GONA.SWATHI** & my bro-in-law **C.RAM** for their constant support. Swathi has been my constant source of support since my childhood. I would like to thank our cute BABLOO for being my stress buster many times & loving me.*

I would like to thank all my juniors for their help and cooperation in any work that we do.

*I would like to thank sister **Margare**t and sister **Parvathi** for their patience, care & competence.*

My heartful gratitude to all my patients who submitted themselves most gracefuuly & whole heartedly participated in this study. I sincerely thank my institute Sri Devaraj Urs Medical College, Tamka, Kolar for giving me a wonderful foundation and forum of knowledge in the field of ophthalmology which stands for the rest of my life.

LIST OF ABBREVIATIONS USED

ACIOL	---	Anterior chamber Intra ocular lens
AC	---	Anterior chamber
ATP	---	Adenosine triphosphate
b.c	---	Before Christ
BCVA	---	Best corrected visual acuity
Ca^{+}	---	Calcium
CCC	---	Continuos curvilinear capsulorrhexis
CME	---	Cystoid macular oedema
DFCS	---	Duration from cataract surgery
DO	---	Direct ophthalmoscopy
ECCE	---	Extra capsular cataract extraction
EP	---	Elschnigs pearls
FIB	---	Fibrous
gz	---	Germinative zone
GSH	---	Glutathione
GSSG	---	Glutathione disulphide
HMP	---	Hexose Mono phosphate shunt
ICCE	---	IntraCapsular cataract extraction
IOP	---	Intraocular pressure
IOL	---	Intra Ocular lens
IDO	---	Indirect ophthalmoscopy
K^{+}	---	Potassium
LOCS	---	Lens opacity classification system
LEC	---	Lens equatorial cells
MH	---	Macular hole

MIP	---	Major intrinsic protein
Na^+	---	Sodium
Nd-YAG	---	Neodynium doped yttrium aluminium garnet
NADPH	---	Nicotinamide adenine dinucleotide phosphate
NSAID	---	Non-steroidal anti-inflammatory drugs
PCO	---	Posterior capsular opacification
PCIOL	---	Posterior chamber Intra ocular lens
PMMA	---	Poly methyl methracrylate
RRD	---	Rhegmatogenous retinal detachment
RD	---	Retinal detachment
RH	---	Retinal haemorrhage
SOD	---	Superoxide Dismutase
SICS	---	Small incision cataract surgery
UGH	---	Uveitis glaucoma hyphaema syndrome
Uv	---	Ultraviolet
VA	---	Visual acuity

ABSTRACT

Background: Posterior capsular opacification is the most common delayed complication following cataract surgery . Nd-YAG laser capsulotomy is the gold standard treatment for PCO which is both simple and effective.Though being a non-invasive and simple outpatient procedure, it is not without complications. I hence undertake this study to evaluate the visual outcomes and complications following Nd-YAG laser capsulotomy in patients with PCO

Aims and Objectives:

1. To assess the visual acuity in patients undergoing Nd-Yag laser capsulotomy.
2. To document the complications following ND-YAG capsulotomy over a period of 3months.

Materials and Methods: 89 patients with PCO attending the ophthalmology OPD between Dec 2016 to May 2018 were selected for the study. Patients who met the inclusion/exclusion criteria were taken up for Nd-YAG laser and followed up on day 1, after 1week, 1month and 3 months for visual acuity, IOP and other complications.

Results: There is excellent and statistically significant($p<0.001$) improvement in visual acuity immediately after the procedure with 90.9% getting a VA of ≥6/18. Several complications are noted which are transient & self resolving. IOP rise is seen in 35.9% patients which is statistically significant ($p< 0.001$) but this rise was transient and subsided with topical betablockers. Iritis is noted in 23.6% but subsided within 1 week. IOL pitting was seen in 5.6 % , but did not affect the visual acuity, hence no much intervention was required. CME was noted in 5patients at the end of 1 month which subsided with topical NSAIDS in 3 patients by the end of third month visit. Certain complications like Corneal

haze & burns, Retinal detachment, Endophthalmitis and Secondary closure of capsulotomy were not seen in our study.

Conclusion: Nd-YAG laser capsulotomy is a safe and effective method of treating PCO. Excellent Visual improvement is noticed immediately after the procedure. Certain complications are noted among patients which can be minimised with minimal energy settings. Majority of the complications are transient and can be managed safely with a proper follow-up.

LIST OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

LIST OF PHOTOGRAPHS

INTRODUCTION

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

The number of people in the age group of >60yrs is shown to increase from 901 million to 1.4 billion in 2030 in the world. Thus the increasing life expectancy will add up the current scenario[2].

The surgical treatment for cataract dates back from sushruta to the present age through which it has undergone numerous improvements from the 'needling' technique to current 'Extra capsular cataract extraction' with PCIOL implantation being the standard of care.

In the modern era of cataract surgery, the lens capsule is left intact to preserve a site for the implantation of the intraocular lens which is the most practised mode of visual rehabilitation, but this posterior capsule may lead to the significant secondary visual loss due to the development of PCO, acting as a substrate for the proliferating remnant epithelial cells.

Due to its multi-factorial causation and pathogenesis, it is difficult to target one specific pathway to alter its development. Many techniques were advocated to reduce the incidence including surgical techniques, IOL biomaterial, IOL design and pharmacological methods.[3,4,5].The incidence of PCO ranged from 30%-50% in 1980's[6][7][8][9] ,it was found to be reduced later in 1990's as studied by **Schaumberg et al**[10] and further decrease is noted with the advent of different IOL materials and edge designs with YAG-capsulotomy rates of

upto 1.3%-14%[11] ,although nearly 100% opacification occurs in cases of children.The interval between surgery and opacification time ranges from 3months to 5years with an average opacification duration being 26months[12].

Inspite of many new advances in the IOL's implanted, the development of PCO still continues to be the most common delayed non-refractive cause of visual dissatisfaction among the patients.

PCO can be treated either with surgical (or) laser capsulotomy, either of them have their own pros & cons. Today Neodynium doped yttrium aluminium garnet (Nd-YAG) laser capsulotomy has become a gold standard approach for treating PCO, due to its ease and effectiveness to improve the dropped visual acuity.[13]

Though being a non-invasive and simple outpatient procedure, it is not without complications. Several complications have been listed in various studies, though majority of them are transient and treatable[14][15]. I hence undertake this study to evaluate the visual outcomes and complications following Nd-YAG laser capsulotomy in patients with PCO attending OPD in R.L.J Hospital, Tamaka, between December 2016 – April2018.

AIMS & OBJECTIVES

1. To assess the visual acuity in patients undergoing Nd-YAG laser capsulotomy.
2. To document the complications following ND-YAG capsulotomy over a period of 3months.

INCLUSION CRITERIA

- Patients with posterior capsular opacification following cataract extraction with IOL implantation with decreased best corrected visual acuity of two or more Snellens lines (≤6/12).

EXCLUSION CRITERIA

- Corneal scarring, dystrophies or degeneration.
- Decentered IOL.
- Thick membranous PCO not suitable for Nd –YAG capsulotomy.
- Patients with uveitis, glaucoma, trauma.
- Retinal and macular disorders.

REVIEW OF LITERATURE

HISTORY OF CATARACT SURGERY:

The term "cataract" was introduced by **Constantinus Africanus(ad 1018)** ,a monk and an Arabic oculist. He translated Arabic 'suffusion' into latin 'cataracta' meaning "the water fall". **(16)**

For more than 20 centuries, '**couching'** was the primary method for dislodging the cataract away from the pupil. The first written description of couching came from **Sushruta**, an ancient Indian surgeon (600 bc).

Figure: 1- surgeon performing couching. From Elliot RH:The Indian operation of couching for cataract. London: HK Lewis; 1917

Couching was performed by a surgeon who sat facing the patient. The patient sat with her or his face illuminated by the midday sun streaming in from a window. An assistant was

positioned behind the patient and further stabilized the patient's head. A pointed needle was plunged either through the sclera ~4 mm temporal to the limbus or through clear cornea. The needle was then passed through the conjunctiva and sclera in a blind approach behind the iris toward the lens. The surgeon would then use a blunted needle to push the white opacity downward (a maneuver called ‘depression’) or to push the superior pole of the opacity backward (a maneuver called ‘reclination’).

Rhazes (ad 865-925) of the Arabian school wrote about **Antyllos** (ad 150), who removed the cataract by means of a glass tube.

Ammar (ad 996-1020), an Iraqi oculist, wrote the Book of Selection of Eye Diseases and described the suction of the cataract through a hollow needle. In the twelfth and thirteenth centuries, Syrian surgeons tried Ammar's aspiration method.

Jacques Daviel (1696-1762), a Normandy-born French oculist, started a revolution of surgical innovation (which continues to the present time) by describing a new, planned method for extraction of the cataract from the eye. In 1753, he published details of this innovative surgery.

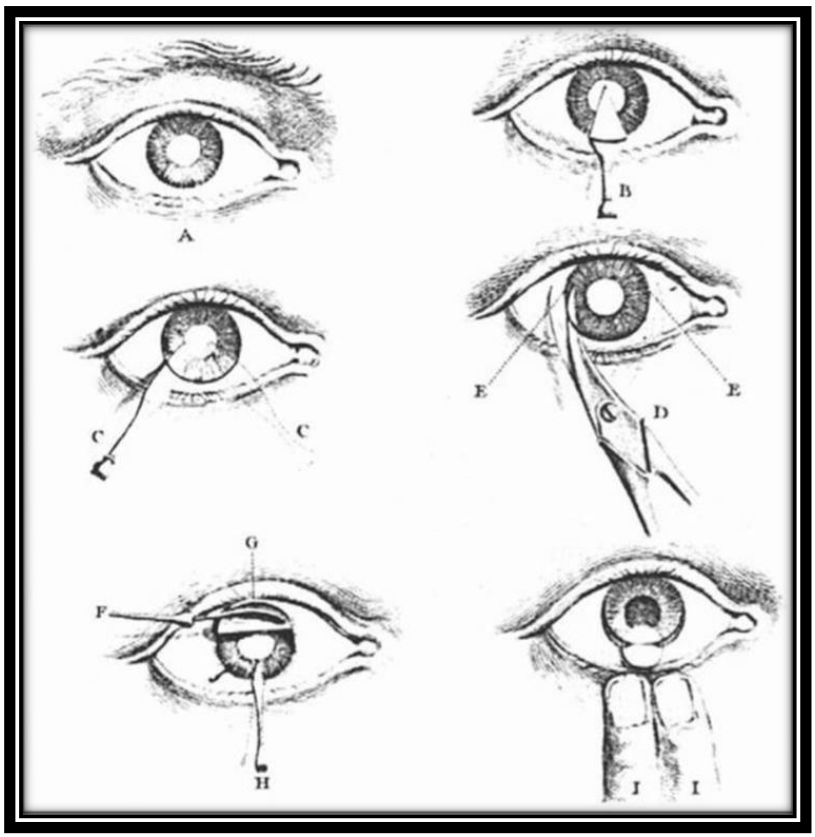


Figure 2: Daviels approach of cataract through an inferior incision and the cataract is expressed out with finger pressure.(Hubbell AA. Samuel Sharp, the first surgeon to make the corneal incision in cataract extraction with a single knife:A biographical and historical sketch. Med Library Historical J. 1904;242:1–16)

Pierre–Francois–Benezet shifted the surgical incision to the upper part of the eye. He had the patient lie on his or her back and operated from the head of the table.

Carl Himly, a German oculist, improved the surgeon's view by introducing pharmacologic mydriasis.

Samuel Sharp (1753) described surgery that introduced the subject of taking the entire lens out of the eye with the capsule intact.

Ignacio Barraquer (1917) performed **'phacoerysis**' with a pneumatic forceps. His son, Jose Barraquer, developed an electric vacuum pump machine with a special erysiphake handle for suction removal of the cataract.

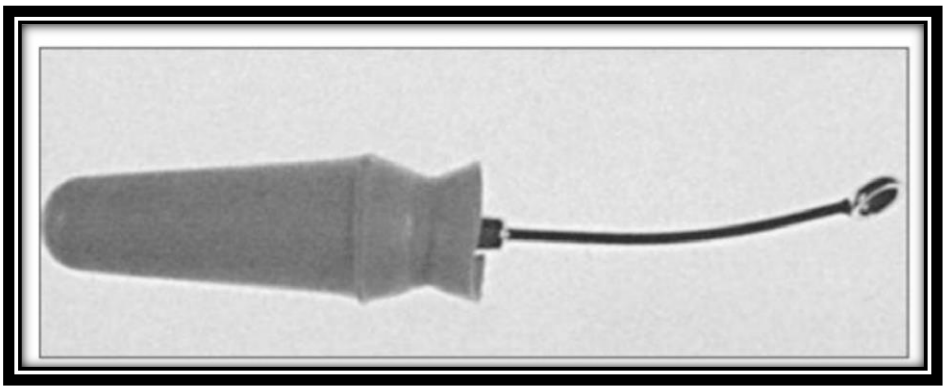


Figure 3: A miniature erysiphake with a rubber suction bulb attached to the probe end.

(Courtesy of the Abraham Pollen Archives and Rare Book Library, Massachusetts Eye and Ear Infirmary, Boston, MA.)

The next breakthrough came to ICCE surgery with the development of chemical zonulolysis. **Jose Barraquer** (1958) demonstrated the dramatic efficacy of chemical zonulolysis using an enzyme α-chymotrypsin.

Krawawicz in Poland (1961) introduced the Cryoextractor. A small, cold probe could be frozen to the surface of the lens forming an ice ball, fusing the lens capsule, cortex, and nucleus, lessening thus the risk of capsule rupture during extraction.

THE RETURN TO ECCE

Despite the encouraging results with ICCE, there remained a substantial rate of potentially blinding complications, including aphakic retinal detachment and cystoid macular edema, which could be reduced by keeping the posterior capsule intact. Moreover, smaller incisional cataract wounds were sought.

But the major concern was the optical rehabilitation of the aphakic patient with glasses.

Harold Ridley performed his first artificial lens implant at St Thomas‘ Hospital in London on Nov 29, 1949. The ICCE left nothing to support Ridley's posterior chamber lens.

Later, **Jaffe and co-workers** made a major contribution by pointing out that the Extracapsular procedure carried a lower incidence of complications. **Norman Jaffe, Henry Clayman, and Marc Jaffe** showed in a prospective study that angiography-proven cystoid macular edema was lower in uncomplicated ECCEs than in uncomplicated ICCEs.

Kelman introduced his phacoemulsifier in 1967**,** but the potential for complications concerned many intracapsular surgeons. The first innovative idea to advance the safety of phacoemulsification was a new capsulotomy. Simultaneously, **Gimbel** introduced his ‘continuous tear capsulotomy‘, and **Neuhann** described his ‘capsulorrhexis‘.

Gimbel and Neuhann recognized their important contribution and decided to co-publish a thorough description and to rename the procedure ‘continuous curvilinear capsulorrhexis’ (CCC).

Gimbel propelled a giant advancement to phacoemulsification by showing that the nucleus could be fractured within the bag by cracking the nucleus his **‘**Divide and conquer nucleofractis’.

Fine described his ‘chip and flip’, a method of flipping over and emulsifying the epinucleus.

Dillman and Maloney described a ‘crack and flip’**.**

Kunihiro **Nagahara** from Japan stunned the surgical world with his clever **‘phaco chop’**.

Bimanual phacoemulsification through a 0.90-mm clear corneal incision, a technique he called ‘Phakonit’ developed by **Amar Agarwal** from India.

HISTORY OF IOL

The development of modern cataract surgery with IOL implantation began after World War II (WWII) with the first implantation of an IOL by Sir Harold Ridley in the St Thomas

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

On 29 Nov 1949, **Ridley** carried out his first lens implantation on a 45-year-old woman.

The IOL material consisted of polymethylmethacrylate (PMMA). This is considered as one of the greatest milestones in the medical history. Ridleys invention has become an accepted option for optical correction of aphakia. Thereafter tremendous changes and improvements were noticed in this field of medicine which had led to the development of different biomaterials and designs.

Generation	**Types**	**Examples**	**Advantages**	**Disadvantages**
1	PCIOL (1949-1954)	RIDLEYS PMMA IOL	Revolutionised the visual rehabilitation of patients after cataract surgery	Subluxation
2	RIGID ACIOL (1952-1962)	-Strampelli tripod ACIOL -CHOYCE Mark1 -DannheimACIOL	No decenteration	UGH syndrome ,Corneal decompensation
3	Iris fixated lens (1953-1975)	-Binkorst iris loop lens -Fydorov "sputnik lens" -Jan worst iris claw lens	Can be used both in ICCE and ECCE	Iris chaffing and AC inflammation
4	Flexible and semi-flexible ACIOL (1963-1990)	Azar 91Z ACIOL ORC Inc stableflex Surgidev Kelman multiflex	Biocompatible materials Better fit and design	Corneal endothelial damage
5	Improved PCIOL (1975-1990)	All current 3-piece PMMA IOLs	-Cost effective -More stability -Less chances of decenteration	Need large incision Astigmatism due to wound

6	Foldable IOLs (1970s to present)	Silicone IOL	-Heat resistant. -Excellent tensile and tear strength.	-low refractive index -can be pitted
		Hydrophobic acrylic IOL	-Reduced rate of PCO -Thinner lens -good resistance to YAG laser	-photopsias & glistenings -susceptible to damage by forceps
		Hydrophilic acrylic IOL	-Thin lens	-Higher rate of PCO. -Susceptible to damage
7	Multifocal IOLs (1986 to present)	-Bulls eye type -Annulus type -Rezoom IOL	No need of a near addition required.	Glare,halos Undesirable visual aberrations
8	-Accomodative IOLs -Toric IOLs(1994 to present)	Staar surgical IOLs AcrySof IQ toric IOL AcriComfort646TLC	Astigmatism corrected	Rotation of IOL

Table 1: generations of IOLs

EMBRYOLOGY OF LENS

Development of the lens begins as surface ectodermal cells, overlying the optic vesicle which thicken to form the lens placode . This placode subsequently invaginates into the forming optic cup until it pinches off as the inverted lens vesicle. The cells approximating the retinal half of the vesicle are then induced to terminally differentiate and, as a consequence, are transformed from cuboidal cells into long fibers-like cells or simply fibers. As these first or 'primary' fibers elongate along the visual axis, the lumen of the vesicle is obliterated. At this point the lens consists of a ball of primary fibres, overlaid by a monolayer of the remaining undifferentiated vesicular cells.

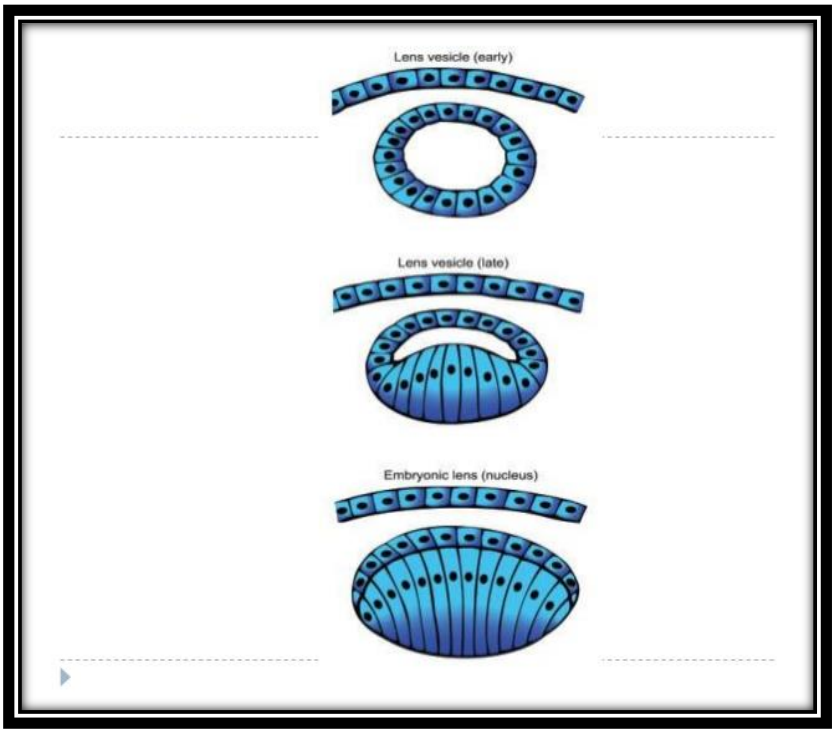


Figure 4: key structural events in the lens development.

Throughout life, the anterior monolayer, generally referred to as the "lens epithelium", serves as the germ cell layer of the lens, a stratified epithelial-like tissue. In lens, the stem cells are sequestered as a narrow latitudinal band within the lens epithelium, known as the "Germinative zone" (gz). The gz lies at the periphery of the lens epithelium just above the lens equator.[10] Some of the gz cells undergo mitotic division, and a number of the daughter

cells terminally differentiate to become additional fibers. Because these are the second fibers to develop, they are referred to as 'secondary' fibers.

As additional secondary fibers develop throughout life, their anterior ends are embedded beneath the apical membranes of the lens epithelium and above the anterior ends of previously formed fibers; while their posterior ends reach above the capsule and beneath the basal membranes of the same previously formed fibers. In this manner, fibers of every layer lie on the top of fibers of the previously formed layer and beneath the fibers of the subsequently formed layer.

In addition, the entire lens mass is enclosed in a basement membrane-like capsule, that is produced by the lens epithelial cells and elongating fibers.The lens has a permanent record of all of its fibers arranged in order of increasing age from its periphery to the interior of the lens. This growth of lens fibres occurs throughout the life, although at a much slower rate after the age of 3.[17]

In foetus, the lens grows rapidly because it is supplied by hyaloid artery, which forms a plexus on the posterior capsule. This vascular posterior capsule is formed from mesenchyme. The true posterior capsule is formed from the thickened basal lamina.[18]

LENS ANATOMY

The lens is a transparent crystalline structure with biconvex configuration and is placed between iris and the vitreous in a saucer shaped depression called 'patellar fossa', diameter being 9-10 mm and thickness varies with age from 3.5 mm (at birth) to 5 mm (at extreme of age). Its weight varies from 135 mg (0-9 years) to 255 mg (40-80 years of age).

The lens is suspended in position by the zonules of Zinn, which consist of delicate yet strong fibers that support and attach it to the Ciliary body.

The lens is composed of i) Lens capsule,

ii) Lens epithelium,

iii) Cortex, and

iv) Nucleus.

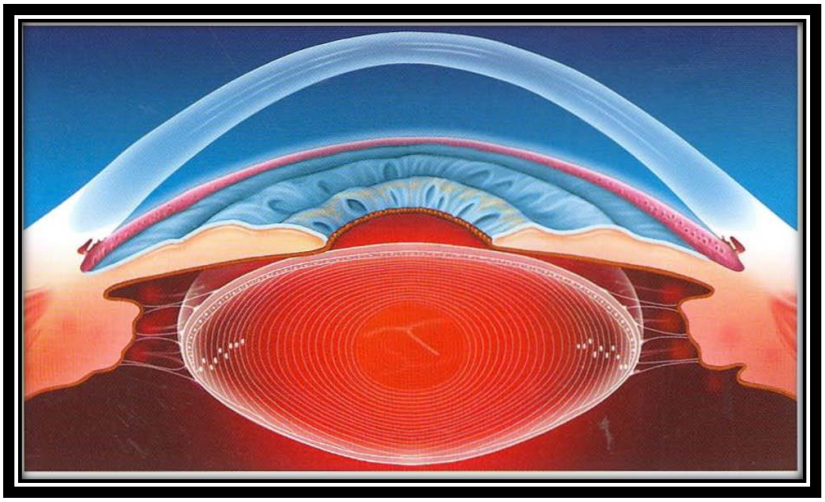


Figure 5: A highly simplified and schematic diagram showing Lens

LENS CAPSULE

The lens capsule is an elastic, transparent basement membrane composed of type IV collagen laid down by the epithelial cells. The capsule contains the lens substance and is capable of changing the shape during accommodative changes. The outer layer of the lens capsule, the zonular lamella, also serves as the point of attachment for the zonular fibers.

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

The anterior lens capsule is considerably thicker than the posterior capsule at birth and increases in thickness throughout life.

Under light microscope, the capsule appears Homogenous, Birefringent and transparent with lamellar structure with fibres arranged parallel to its surface.

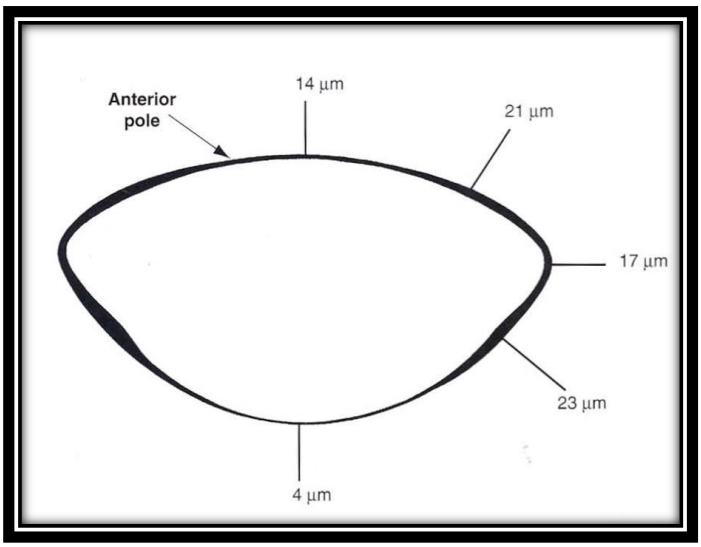


Figure 6: showing the thickness of lens capsule at different sites.

LENS EPITHELIUM

The lenticular epithelium, which lies below the capsule is found only on the anterior surface of the lens. It is made of cuboidal cells which become columnar as they approach the equator. There is no epithelium on the posterior surface, as these cells fill the central cavity of lens vesicle during development of lens and hence used up.

Electron microscopic observation shows that membrane of these polygonal epithelial cells are tortous with many interdigitations.(19)

LENS FIBRES

The epithelial cells elongate to form lens fibres. Mature lens fibres are cells which have lost their nuclei. As the lens fibres are formed throughout the life, these are arranged compactly as nucleus and cortex of the lens.

Nucleus- It is the central part containing the oldest fibres. It consists of different zones, which are laid down successively as the development proceeds. In the beam of slit-lamp these

are seen as zones of discontinuity. Depending upon the period of development, the different zones of nucleus in the lens include:

Embryonic nucleus- it is the innermost part of the nucleus,consisting of fibrs formed upto 3 months of gestation. It consists of the primary lens fibres which are formed by elongation of the cells of posterior wall of lens vesicle.

Foetal nucleus - It lies around the embryonic nucleus and corresponds to the lens from 3 months of gestation till birth. These fibres meet around sutures which are anteriorly Y-shaped and posteriorly inverted Y-shaped

Infantile nucleus –It corresponds to the lens from birth to puberty, and

Adult nucleus –This part corresponds to the lens fibres which are formed after puberty till the rest of the life.

CORTEX

It is the peripheral part of the lens which comprises of youngest lens fibres.

The recent layers are laid on top of the previous layers which are buried gradually as the lens ages.

The cortex of young adult lens is comprised of all the mature secondary fibres added after sexual maturation.

In aged lens, the cortex consists of all the mature secondary fibres added after the middle age.

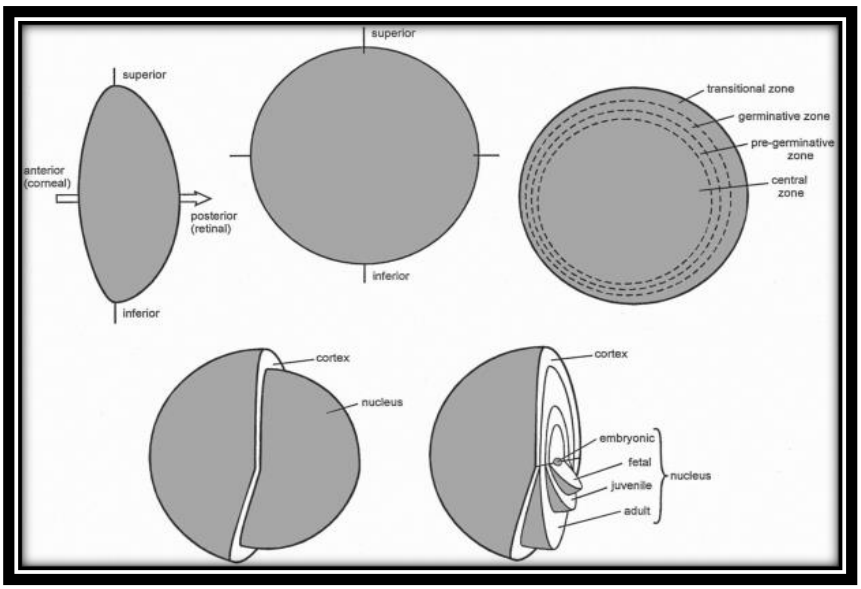


Figure7: Diagrammatic representations of the gross shape, anatomic orientation, and developmentally defined regions of a normal human adult lens.

PHYSIOLOGY & BIOCHEMISTRY OF LENS

The molecular composition of the lens is quite different, with two-thirds made of water, one-third made of protein and the other constituents accounting to less than 1%.[(20)]

Its high protein helps to maintain high refractive index.

The adult human lens contains approximately 65% water, out of which lens capsule constitutes 80% of water.

LENS PROTEINS-

Proteins account for 35% of the net weight of the lens. Based upon their solubility in water, there are 2 classes:-

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

ii) Water insoluble proteins- they consists of membrane proteins, cytoskeletal proteins and aggravated crystallins.

LENS CRYSTALLINS

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

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

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

WATER INSOLUBLE PROTEINS

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

2. Cytoskeletal proteins.

LENS LIPIDS :

It includes cholesterol, phospholipids and Glycosphingolipids.

About 50-60% of lens lipids is cholesterol. Major phospholipids are Sphingomyelins.

SODIUM & POTASSIUM:

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

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

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

CALCIUM:

The normal young lens has one of the lowest levels of calcium level of all tissues.

Value of 0.14mg/mg dry weight in human lenses.

ANIONS:

The main anions of the lens are chloride, bicarbonate, phosphate and sulphates. Phosphate is the predominant anion in the lens.

ORGANIC PHOSPHATES:

They also form a significant group of lens constituents.

It includes nucleotides of both adenosine and pyridine. Adenosine triphosphate (ATP) causes phosphorylation of glucose. Besides ATP, the presence of various other nucleotides such as the mono- and di- phosphates have been reported.

Pyridine nucleotides act as coenzymes to the dehydrogenases, involved in the oxidation-reduction processes.

GLUTATHIONE:

The content of glutathione in the lens varies from 3.5-5.5mm/g wet weight of the lens.

There is a relative drop in the levels of glutathione with age due to in the wet weight of the lens.

Glutathione is a tri-peptide with 3 aminoacids - glycine, cysteine and glutamic acid. The cysteine group is the most reactive constituent which enables glutathione in 2 forms i.e, oxidised form (GSSG) and reduced form (GSH).hence glutathione contributes to the so-called REDOX-SYSTEMS in the lens.

ASCORBIC ACID:

A wide variation of ascorbic acid levels is reported in the lens ranging from 5 to 48 mg/100 gm wet weight. In aqueous humour ascorbic acid is transported to a level 15times higher than that of plasma. Though the level of ascorbic acid is higher in lens compared to the aqueous, but it is neither synthesized nor actively transported into the lens.

LENS METABOLISMS:

GLUCOSE METABOLISM- Is the main source of energy. The lens requires a source of energy continuously for the active transport of ions, amino acids, maintenance of lens dehydration and lens transparency.

Glucose is of prime essence for the normal working of the lens. When deprived of glucose, the lens rapidly uses up endogenous energy reserves (ATP, glucose, sorbitol and fructose) and begins to gain water and lose transparency.

Glucose from aqueous and vitreous diffuses into the lens and is rapidly metabolised through 4 main pathways-

1) Anaerobic glycolysis
2) Krebs cycle
3) Hexose monophosphate shunt
4) Sorbitol pathway.

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

Free amino acids are transported actively from the aqueous with the ATP produced from carbohydrate metabolism. protein synthesis rate is different in different areas of the lens, slowest being the nucleus.

Protein breakdown in the lens is catalysed by the peptidases and proteases.

OXIDATION- REDUCTION PATHWAY- Free radicals are produced in the normal course of cellular metabolic activities. These free radicals which are highly reactive can damage lens fibres.

The lens is equipped with several enzymes that protect against free radical or oxidative damage- Super oxide dismutase (SOD), catalase, and glutathione peroxidase.

SOD acts as a catalyst for the destruction of superoxide anion, O_2^-, and produces hydrogen peroxide, which in turn is broken down by catalase.

Glutathione peroxidase catalyses a reaction resulting in the production of glutathione disulphide (GSSG), which is then reconverted to glutathione (GSH) by glutathione reductase, using NADPH as a reducing agent. NADPH is produced from HMP shunt.

Vitamin E and ascorbic acid are present in the lens, acts as a free radical scavenger.[21][22]

MORPHOLOGICAL CHANGES OF LENS WITH AGEING

Many morphological changes can be observed in the epithelial cells, lens fibers, and the capsule as the lens ages.

The proliferative capability of the epithelial cells decreases with age and the overall cell density is reduced.[23] Thus epithelial cells become thinner with flatter nuclei, acquire electron-dense bodies and vacuoles, and there is an increase in cytoskeletal components.

There is a marked increase in the density of the surface projections of the cells with age which results in an increase in plasma membrane surface area.

At the ultrastructural level, lens fibers show a total loss or partial derangement of certain plasma membrane and cytoskeletal proteins as the lens ages. The most significant is the degradation of major intrinsic protein 26 (MIP26) which in turn reduces cell–cell communication.

The cytoskeletal elements spectrin, vimentin, and actin are present in both the epithelial layer and the outer cortical fibers; however, they are degraded with ageing and become further internalized and their expression is restricted to the epithelial cells by 80 years of age.

The cholesterol: phospholipid ratio of the plasma membranes in the fibre cell increases throughout life, and as a result the membrane fluidity decreases and structural order increases. These changes occur from the second decade and are greatest in the nucleus

From the fourth decade onward, ruptures are found in the equatorial region of cortical fiber plasma membranes and reparation of these ruptures can prevent the formation of opacities.

The lens capsule thickens, loses its elasticity, shows loss of laminations and accumulates age-related cross-links in the matrix proteins. The young lens capsule is known to contain collagen type IV and the aged capsule collagen I,III,IV.

CHANGES IN LENS PHYSIOLOGY WITH AGEING

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

The major gap junction protein MIP26 looses some of its amino acids to form new variants.

The membrane potential of an isolated, human lens will have reduced to –20 mV at the age of 80 years compared to –50 mV at the age of 20 years.

The sodium (Na^+) content of the lens increases from 25 mmol/L at 20 years of age to 40 mmol/L by 70 years of age, while potassium (K^+) levels remain relatively constant throughout life at ~150 mmol/L.

The Na^+: K^+ permeability ratio increases six fold by age 80 and results in a proportionately greater rise in sodium content of the lens which correlates with the increase in optical density of the lens .[24]

Free Ca^{2+} increases from 10 μmol/L at the age of 20 years to ~15 μmol/L by the age of 60 years.

AGE-RELATED CHANGES OF LENS CHROMOPHORES.

With ageing there is a progressive accumulation of different types of chromophores absorbing blue initially and then visible light of longer wavelength. The accumulation of these chromophores leads to progressive yellowing of the lens, which, in advanced age, may appear brunescent.

AGE-RELATED METABOLIC CHANGES

The overall metabolic activity of the lens, as well as the activity of many glycolytic and oxidative enzymes decreases with increasing age.

Although overall metabolic activity decreases, the lens, for the most part, retains the capacity to synthesize proteins, fatty acids, and cholesterol at substantial rates. Decreased metabolic activity is not a significant limiting factor for the production of new lens fibers and may reflect an aging lens rather than pathology.

OXIDATIVE STATUS OF THE AGED LENS

In order to retain its main function – focusing visible light on the retina, the lens must retain appropriate refractive status and transparency throughout life under conditions of oxidative damage.

Firstly, the lens is continually exposed to UV light that causes oxidation reactions and generation of reactive oxygen species.

Secondly, there is an age-related increase in the concentration of other lenticular photosensitizers.

Glutathione peroxidase levels increase from birth until ~15 years of age and then slowly decrease throughout adulthood. It dereases by 50 – 60% in lenses older than 70years.[25]

CHANGES IN CRYSTALLINS

The ability to maintain a clear lens and to resist lens opacification (cataract) is dependent upon the structure, stability, and function of lens crystallins. Up to 80% of nuclear proteins of an aged lens may be insoluble including α-crystallin by the age of 45 years and this may contribute to the loss of lens transparency and the development of senile cataract.

The lens is exposed to the cumulative effects of radiation, oxidation and posttranslational modification throughout life. Posttranslational modifications of crystallins occurs with aging and this results in the unfolding and eventual aggregation of crystallins.

In the lens, premature aggregation of crystallins is avoided due to the presence of proteins, including α-crystallins, which belong to the family of Heat Shock Proteins (HSP).These proteins act like molecular chaperones by binding to partially denatured proteins, thereby preventing irreversible protein aggregation during aging.

CLASSIFICATION OF CATARACT:-

1) Congenital
2) Acquired – further classified as-
    a. Age-related
    b. Radiation induced

c. Toxic
d. Traumatic
e. Ocular diseases
f. Systemic diseases
g. Metabolic disorders.

MECHANISMS OF CONGENITAL CATARACT-

A number of different types of genetic mutations may result in different forms of defective crystallins, which are associated with formation of different types of congenital cataract.

Any insult to the nuclear or lenticular fibers can result in formation of cataract. Possible causes include intrauterine infections, metabolic disorders, and genetically transmitted syndromes.

Infectious causes of cataracts include-

Rubella (most common),

Cytomegalovirus

Herpes zoster,

Herpes simplex,

Syphilis

Epstein–barr virus,

Syphilis,

Toxoplasmosis and

Familial congenital cataracts- like Galactosemia, pierre-robin syndrome, Down syndrome, trisomy 13, Lowe syndrome.

SENILE CATARACT-

Three principal types of age-related cataract exist, depending on the area of the lens opacity, namely –

i)Nuclear,

ii)Cortical,

iii)Subcapsular cataract and

iv)Mixed cataract

NUCLEAR CATARACT-

1. As the lens ages, there is a reduction in the rate at which nutrients, water, and antioxidants can enter the cells of the lens nucleus via the epithelium.
2. Reduced lens epithelial cell density results in an alteration of lens fiber formation and homeostasis.
3. There is an increase in the ratio of insoluble to soluble proteins in the cataractous lens after the age of 50.
4. Oxidative factors cause biochemical damage and morphologic alterations in the human lens resulting in cataract formation. Glutathione (GSH) is the most important anti-oxidant in the lens along with ascorbic acid and vitamin C.

5. Cell membrane damage -The amino acids methionine and cysteine are most vulnerable to oxidation, particularly in the proteins associated with the cell membranes. Oxidation of membrane lipids is observed in cataractous lens. influence the function of membrane-associated enzymes, such as $Na^{+}K^{+}$-ATPase and Ca^{2+}-ATPase leading to epithelial cell death. This leads to osmotic shock, crystalline aggregation, and lens opacification.

CORTICAL CATARACT-

Several mechanisms may initiate the cortical cataract, which includes -

1. Damage to the plasma membrane of lens fibre.
2. Loss of protective molecules such as glutathione,
3. Excessive breakdown of proteins,
4. Disruption of electrolyte and water balance and
5. Damage to the system responsible for calcium homeostasis. These factors are interrelated in the initial formation of cortical cataracts. Loss of calcium homeostasis spreads opacification around the lens periphery and towards the nucleus. Calcium levels are elevated in damaged cells in cortical cataracts. Elevated calcium levels cause proteolysis, protein aggregation, and light scattering.

SUB-CAPSULAR CATARACT

Anterior subcapsular type of cataract lies directly under the anterior lens capsule and is associated with fibrous metaplasia of the lens epithelium. Posterior sub-capsular opacity develops due to swollen and migratory lens epithelial cells and causes opacity due to

breakage of the swollen lens fibre cells .Opacification occurs exactly in the pupillary axis and particularly has a profound effect on vision.

TOXIC CATARACT

There are several drugs which are believed to cause cataract like Steroids, miotics, chlorpromazine, allopurinol, chloroquine, and amiodarone, on the other hand there are other drugs that are proven to retard cataract formation like non-steroid anti-inflammatory drugs.([26]. Corticosteroid use has been reported to cause cataracts in 5%-60% of patients, particularly bilateral posterior subcapsular cataracts.[27]

RADIATION

Ionizing radiation like X-rays, gamma rays, beta rays, and neutrons cause cataract because of their effect on the germinal epithelium at the lens equator. This process may take 4-10 years depending upon the degree of exposure. The defective fibres migrate to the posterior pole, producing feathery and dust-like opacities.

TRAUMA

Lens can be damaged by both blunt and penetrating injuries. The blunt force causes antero-posterior shortening and equatorial expansion of the globe, which causes tearing apart of the lens fibers in the axial region of the cortex, leading to flower-shaped cataract.

METABOLIC DISEASES

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

CATARACT GRADING

Cataract classification is the designation of the type or severity of cataract, or both, according to a standardized method.

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

LOCS III system employs six nuclear standards for color and opalescence grading and five standards for cortical and posterior sub capsular grading. It is currently accepted and utilised for grading cataract worldwide.[28]

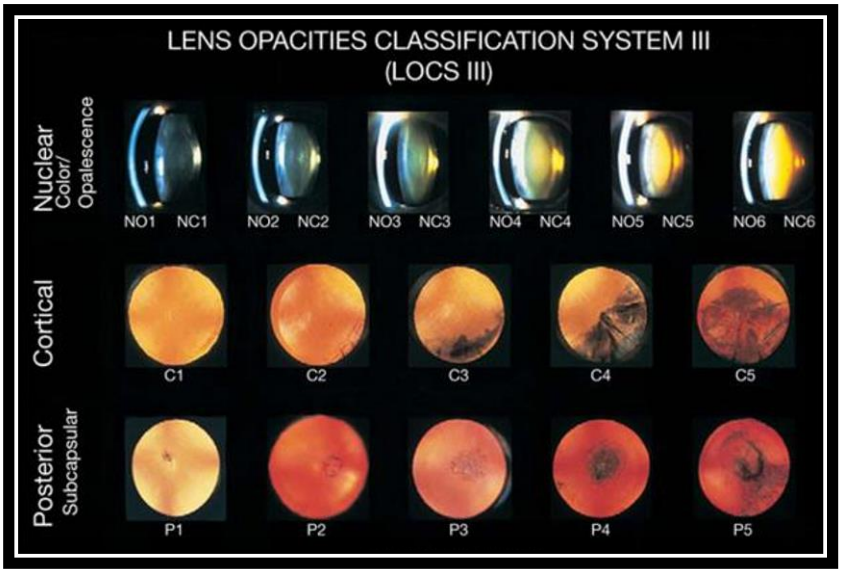


Figure 8 : LOCS III. Set of slides for grading standardized photographic images of opacity.

(Chylack LT Jr, Wolfe JK, Singer DM, et al: The lens opacities classification system III. The Longitudinal Study of Cataract Study Group. Arch Ophthalmol 1993; 111:831-836.)

Other invivo gradings were also described-

WILMER GRADING

The Wilmer system uses four nuclear standard photographs for grading nuclear opacity. The gradings are based on three criteria: visual acuity, density, and extent of the opacity. [29]

The Only opacities that could be seen in retro-illumination are graded by estimating the proportion of the total circumference of the lens occupied by the combined cortical opacities as if they are adjacent and posterior sub-capsular opacities are examined using retro illumination.

OXFORD GRADING

It is a comprehensive method of assessment which grades the intensity from a scale of zero to five.it includes several characteristics like anterior clear zone, anterior subcapsular cataract, posterior sub capsular cataract, cortical spoke opacities, water clefts, vacuoles, focal dots, retro dots, nuclear brunescence and white nuclear scatter[30].

Japanese-CCESG system-

This system also uses a set of standard photographs in grading cortical, nuclear, and subcapsular opacities.

These cataracts are graded as early, moderate, or advanced[31].

CATARACT SURGERY

The decision to operate mainly depends on several indications and on patients demand, which has been greatly expanded in the recent times, due to availability of high quality implants and modern cataract surgery. It has to be ensured that the visual impairment was due to cataract.

It is an essential part of patient preparation to counsel patients to anticipate the expected outcome, and yet to forewarn the patient that there are risks in any surgical procedure as well as their desired benefits

PRE-OPERATIVE EVALUATION IN CATARACT SURGERY

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

OCULAR EXAMNATION

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

Informed consent requires explanation to the patient of the nature of the whole process, the surgical timetable, explanation of what may be expected post-surgery and a complications review.

CATARACT SURGERY

It is the most common surgery performed in patients over 65yrs of age.

There are different tecniques of cataract surgery-

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

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

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

BASIC STEPS OF ECCE-

Manual SICS and phacoemulsification are commonly performed surgeries.

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

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

In phacoemulsification, a clear corneal tunnel of 2.5-3.2mm is made followed by anterior capsulotomy and hydro-dissection, the nucleus is emulsified with ultrasonic energy in the bag followed by implantation of foldable IOL after a thorough cortical wash.

COMPLICATIONS OF CATARACT SURGERY

(A) Preoperative complications

(B) Intraoperative complications

(C) Early postoperative complications

(D) Delayed (late) postoperative complications

(E) IOL-related complications

PRE-OPERATIVE COMPLICATIONS

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

6. Insufficient analgesia
7. Insufficient akinesia

COMPLICATIONS OF CATARACT SURGERY

Intra-operative complications

1. Superior rectus injury.
2. Incision related complications

-Button holing of anterior wall of tunnel,

-premature entry into AC

4. Tear of descemet Membrane.
5. Iris prolapse/ intra-operative floppy iris syndrome
6. Complications related to anterior capsulotomy

-Peripheral extension

-Small capsulotomy

7. Chamber collapse, Positive pressure, Thermal burns in phacoemulsification.
8. Posterior capsular tear
9. Improper placement of IOL
10. Zonular dialysis
11. Nucleus drop
12. Expulsive choroidal haemorrhage

Early post operative complications

1. Pain
2. Wound dehiscence ,wound leak, wound rupture

3. Corneal oedema

4. Secondary Glaucoma

5. Postoperative anterior uveitis

6. Bacterial endophthalmitis

Late post-operative complications

1. Cystoid macular edema

2. Pseudophakic bullous keratopathy

3. Retinal detachment

4. Epithelial ingrowth

5. Fibrous downgrowth

6. Secondary galucoma

POSTERIOR CAPSULAR OPACIFICATION/ AFTER CATARACT

It is one of the vision impairing complication which occur in upto 50% patients post-cataract surgery between 3months- 5years.

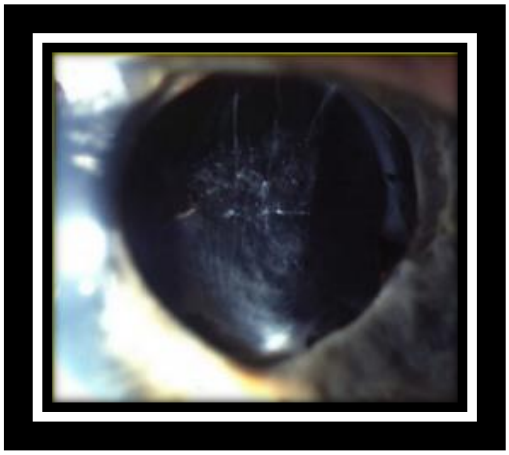
With the advent of new IOL designs and in-the-bag placement of the lens and techniques, the incidence has reduced but not yet eliminated.

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

Clinically they are mainly divided into 3 types-

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

FIBROUS TYPE OF PCO



Photograph 1: Fibrous type of PCO

The main cause of this type of PCI is the presence of residual lens epithelial cells attached to the undersurface the anterior capsule.

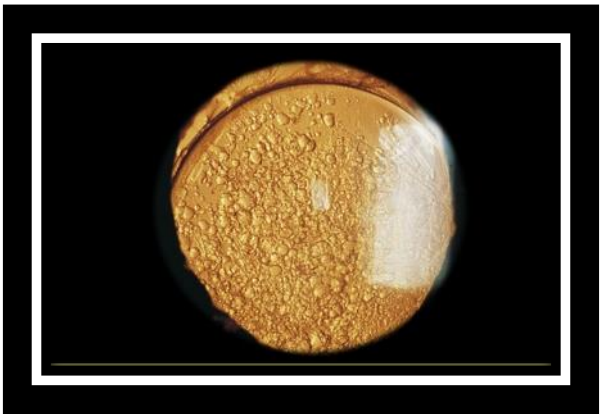
These residual epithelial cells differentiate into spindle cells, fibroblast like cells which have contractile function. These fibroblastic cells proliferate and migrate into the posterior capsule to form a cellular layer which secretes extra-cellular matrix components and basal-laminar like material.The contraction by this cellular layer results in the folds & wrinkling on the posterior capsule.

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

The basal laminar like material that is laid is composed of collagen type 4 and proteoglycans like heparin sulfate. This fibroblastic proliferation and cellular growth is further stimulated by

the presence of certain growth factors such as acidic/basic fibroblast growth factor, epidermal derived growth factor, platelet-derived growth factor and transforming growth factor beta.

ELSCHNIG'S PEARLS



Photograph 2 : Elschnigs pearl type of PCO.

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

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

Individual pearl is an epithelial cell that has attempted to differentiate into a new lens fibre which possess the characteristics of both epithelial cells and fibres. They affect the vision when the pearls accumulate in the centre of the posterior capsule.

SOMMERING'S RING

Residual cortical fibres and epithelial cells are trapped within the area between the anterior capsule and posterior capsule and sealed within this structure.

The equatorial cells still retain the capacity to proliferate and differentiate into lens fibres. This results in the formation of ring which is formed in the periphery, hence does not affect the vision.

PATHOGENESIS OF PCO

Anatomically there are two types of cells involved in the development of PCO.

They are mainly of 2 types- 'A-cells' and 'E-cells'.

McDonell et.al have shown that lens epithelial cells undergo hyperplasia & differentiation into myofibroblast like cells within few days after surgery.

'E-cell' proliferation and migration onto posterior capsule forms balloon-like bladder cells (Wedl cells) of Elschnigs pearls.

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

Both autocrine and paracrine mechanisms were found to influence the formation of PCO. These mechanisms were found to be influenced by several cytokines.

Nishi O.et.al found that cytokines such as interleukin IL-1 and fibroblastic growth factors increased lens epithelial cells mitosis and collagen synthesis.

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

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

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

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

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

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

METHODS OF ASSESMENT OF PCO-

1) Subjective tests
2) Objective tests

Subjective assessmentof PCO is mainly based on the visual function tests.

Hayashi.et.al,2003[13], Aslam & patton[32],2004 and **Meacock[42]** et.al 2003 have shown that visual acuity and contrast sensitivity are the 2 main visual functions that are affected by PCO.

They have linked the development of glare disability and forward light scatter also to PCO development.

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

Kruger et.al[33] graded the PCO of central 3mm as-

0=absent

1=very mild

2=moderate

3=dense white

Sellman[34] and lindstorm graded PCO on a fourpoint scale which uses diagrams to illustrate the various grades of both fibrous as well as pearl type of PCO.

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

2=mild PCO reducing the red reflex / Elschnigs pearls on the IOL edge

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

4=severe fibrosis / pearls covering the visual axis reducing the red-reflex.

Legler.et.al [35]used the visibility of posterior segment structures including optic nerve head, retinal blood vessels, retinal nerve fibre layer with indirect ophthalmoscope.

Grade 1= RNFL &blood vessels seen clearly

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

Grade 3= optic nerve head hazy.

Sangeeta.et.al[36] used the visibility of posterior segment for grading the PCO.

Mild= Fundus seen clearly with direct ophthalmoscope.

Moderate= Fundus seen clearly with indirect ophthalmoscopy.

Severe= No view of fundus (or) details hazy.

We have used the same technique to grade the PCO as in Sangeeta.et.al.

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

Imaging systems

1) Scheimpflug system- it uses are densitometry that measures the scattering ligt intensity.It is highly reproducible but IOL material significantly affects the scattered light density measurement.
2) Digital photography- there are several group of computer based systems, each of which adopts a specific principle to quantify it.

   Brightness based analysis sytem which depends on image pixel grey value to grade PCO as per wang.et.al.

   Heidelberg imaging, which uses retro-illumination photos to score PCO (tetz et al)

   Density map system by friedman.et.el
3) TRACEE, an image analysis computer program that is enhanced with modues for the grading of PCO.

RISK FACTORS FOR THE DEVELOPMENT OF PCO

1. Young age

The rate of LEC proliferation is age –dependant and the the rate in patients <40yrs is 3times faster than that of rate in >60yrs.

2. Surgical procedure-

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

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

Though contradicted by some studies[37] phacoemulsification with cortical irrigation & aspiration lead to less residual LEC concentration than with ECCE with manual nuclear expression[38] and meticulous vacuuming of the capsule using ultrasound endocapsular cataract extraction greatly reduced the need for laser capsulotomy[39].

3. IOL related factors- Many IOL related factors are taken into account which are thought to influence the incidence of pco.

The bio-compatibility of the material that is used is a factor that significantly influences the incidence. It has been proved that the hydrophilic lenses are associated with more incidence of PCO than hydrophobic lenses.[40]

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

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

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

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

4.**Pharmacological alterations-**

Immunotoxin (MDX-A) is an important pharmacological factor that is found to be associated with reduced PCO rates.[41,42]

Use of heparin is also advocated to reduce the incidence of the PCO either in the form of irrigating solution (or) with the usage of IOL coated with heparin (or) as topical eye drops.[43]

TECHNIQUES TO PREVENT PCO FORMATION ARE AS FOLLOWS:

Surgical strategies/ modifications-

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

Intra-ocular lens related changes-

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

MANAGEMENT OF POSTERIOR CAPSULAR OPACIFICATION-

1. Surgical management

2. Laser management.

SURGICAL MANAGEMENT

In remote areas where there is no laser infrastructure or if the thickness of the PCO is not amenable to laser, surgical approach can be adopted , however the results are inferior compared to laser[(44)].

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

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

1. Primary posterior capsulotomy.

   Congenital cataracts which are expected to show almost a 100% incidence of posterior capsular opacification within a 2year duration can be managed in the same

sitting by a planning a posterior capsulorhexis followed by anterior vitrectomy with either a in-the-bag placement (or) by an IOL-optic capture.

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

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

2. Secondary surgical capsulotomy

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

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

Complications associated with surgical treatment of PCO-

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

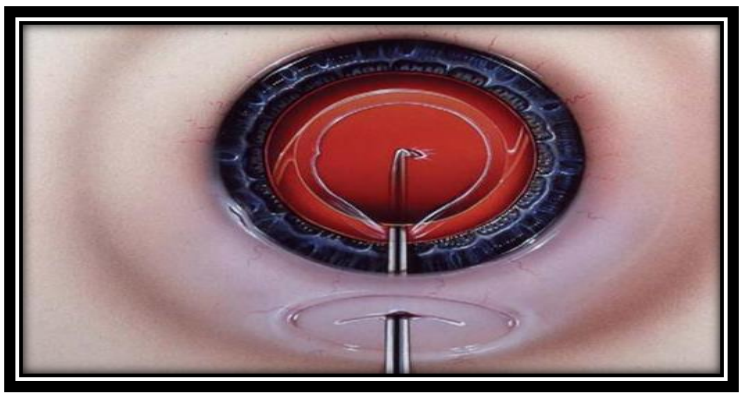


Figure 9: Posterior capsulotomy. The posterior capsule is punctured using a cystotome or a finely barbed disposable 27-gauge needle.

LASER MANAGEMENT

NEODYMIUM YTTRIUM ALUMINIUM GARNET LASER(ND-YAG)

CAPSULOTOMY

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

Albert Einstein in 1917 was the first to introduce the concept of laser. However it was **Maiman** in 1960, who first produced pulsed ruby laser which is utilised with a direct ophthalmoscopy.

Later in 1961 **Zare**t and others began to use ruby laser for photocoagulation both in animal studies and later followed by human studies.

Lasers were a breakthrough in the field of ophthalmology due to the ocular tissue consistency and visibility which made the penetration better and non-invasive.

Bell laboratories were the first to produce Nd-YAG laser in 1964,but their usage in clinical practice was observed many years later.

Pulsed Nd-YAG laser was introduced by **Frankhauser & others** which were used to clear the transparent eye tissues like posterior capsular opacification / vitreous strands.

Later the development of ultra-short pulsed laser had led to further improvement in the treatment of PCO with extremely short duration of laser pulses.

There are several clinical applications of laser in ophthalmology for which a basic understanding of the light-tissue interactions are needed.They are –

a) Photocoagulation
b) Photo ablation
c) Photo disruption.

In **photocoagulation,** the target ocular tissue/ adjacent tissue absorb the laser light & generates heat which denatures the proteins. This laser- tissue interaction applies to the Pan-retinal photo coagulation, peripheral iridectomy, argon laser trabeculopasty, and coagulation of choroidal- neovascular membrane. This photocoagulation can be achieved by different types of lasers such as argon blue green laser, argon green laser, Red ruby laser, diode laser, kypton red laser and Nd-YAG laser(frequency doubled).

In **photoablation** , absorption of photons breaks the chemical bond which binds the tissue together by vapourising the tissue. This is the cause of precision without any damage to the adjacent tissue. This mechanism is used in the photo-refractive keratectomy with Argon fluoride excimer laser.

Photodisruption works principally on the mechanical effect. Nd-YAG laser used for posterior capsular opacification to create a laser capsulotomy is an example of this and discussed further in detail.

Principle of Nd-yag laser- It works on the principle of photodisruption.

A infrared beam of 1064nm is produced which concentrates sufficient energy required to produce a ‘plasma effect’ at a focal point of 11microns which disrupts the tissues adjacent to it acoustically.

History of photodisruption:-the lasers that were built initially had very low power which were not successful in achieving optical tissue breakdown. Later due to the development of

Q- switched mode by **Hellwarth** in 1962, it was possible to achieve giant ruby pulses which are very brief in the order of 10-50nano seconds with the power range of megawatts.

Krasnov demonstrated that clinically desirable disruption of tissues can be achieved by high peak power pulses. He described and performed a technique called 'phakopuncture' in which he successfully disrupted the anterior lens capsule in rabbits.

Gasterland successfully caused photodisruption with a laser system that he built i.e, 'Q-switched rule' laser system. However,the energy was much greater and the output was bought to a focal spot of 175microns which was required in membranectomy.

Frankhauser and associates worked on the Q-switched Nd-glass and later on with Q-switched Nd-yag laser.

Aron rosa worked with mode-locked Nd-yag and began her clinical trials in 1978.

CHARACTERISTICS OF LASER LIGHT:- **(45,46,47,48,49)**

There are several properties of laser which make it useful for clinical application in medicine.

1. MONOCHROMATIC-

   The light emitted is of only one wavelength .it can also produce a combination of wavelengths which can be easily separated. This property of monochromaticity allows the light to be focussed as a smaller spot compared to white light which is diffuse.

2. DIRECTIONAL-

   This property allows the light to focus on a small spot by collecting it with a simple condensing lens. The narrow beam that travels between the 2 mirrors are only

amplified by laser. Beam divergence is a measure of directionality of laser light beam and expressed in milli-radians.

3. COHERENCE-

   It is the measure of regularity and predictiblity of electro-magnetic field with tissue and space.This property is useful in focussing of the beam.

4. POLARIZATION-

   It is the property of orientation of the light. This property is incorporated into the system for maximal transmission in a particular direction without any loss due to reflection.

ELEMENTS OF LASER

There are 3 basic components in laser, namely-

1. Active medium
2. Energy input through pumping
3. Optical feedback

Active medium has an atomic medium which enables large number of atoms to reach above the ground state energy level and allows stimulated emission. The active medium can either be a gas (or) liquid (or) a solid (just as in Nd-YAG where the active component neodymium is supported by a crystal i.e, yttrium aluminium garnet. In Nd-YAG, the active element is neodymium which is doped into YAG crystal which is merely a carrier material.

The second component of laser system involves the energy input that is required to activate the active element from ground state to higher energy states. This process is

called as “population- inversion”, which is exactly reverse of the normal condition where the atoms occupy the lowest possible energy state.

The energy used to produce this condition of inversion is called “pumping” and is differently done in different state of lasers. In Nd-YAG, the solid crystal is pumped with incoherent light like xenon arc flash. Gas lasers are pumped electrically with the electrodes placed in the gas.

The final element is the laser is the optical feedback which is necessary for stimulated emission & supressing the spontaneous emission. This optical feedback is created within the laser cavity which is an optical resonator.

With the placement of mirrors at the either ends of the light path, any reflected rays are collected and passed through the active medium, which again maintains a population inversion by pumping. Through this process, each time the light wave resonates in the acoustic medium and increases the energy through stimulated emission. This kind of energy amplification does not occur in spontaneous emission, because the emission occurs in several random directions which do not hit the mirror ,hence not amplified.

This radiant energy when absorbed by the matter, the temperature rises with increase in the irradiance, and the matter transforms into gaseous state. With further rise in temperature, this gas partly gets ionized and releases free electrons.Thus within the space of focal volume, there are electrons, ions and atoms which move and collide with each other. This collision results in production of electromagnetic radiations in the form of photons (or) electrons. The ‘plasma’ which is produced has different properties from that of normal gas.

This plasma once produced helps to attain 'optic breakdown threshold', and with further increase in energy, the plasma formed increases causing stronger and larger acoustic waves. With increase in the energy, the focus of the beam should be shifted posteriorly to avoid inadvertent damage to the IOL.

The following are the mechanisms involved in the photodisruption-

1. Acoustic wave
2. Ionization
3. Electric field stress
4. High temperatures
5. Cavitation.

In Nd-YAG laser, Neodynium is the active element, trivalent ion, called as 'dopant'. This dopant is incorporated into a Yttrium aluminium garnet (YAG) $Y_3AL_5O_2$ in the ratio of 1:100 and this process is called 'doping'. This YAG crystal is a stable hard crystal which can withstand high laser energies due to its excellent optical and mechanical properties. This doped material has an electron cloud which surrounds the active element i.e, Neodynium & emits light in the wavelength of 1064nm.

The laser delivery rod is a cylindrical structure and the umping system is maintained close to the laser rod for good pumping effect.**(50,51)**

MODES OF LASER DELIVERY-

1) Continuos mode : 0.1millisec to 1.0milliseconds
2) Pulsed mode : a) Q- switched mode: 5 – 20 nanoseconds
   b) Mode locked: 30 – 100 picoseconds.

In **Q-switched mode**, laser light emission is blocked by one of the mirrors till a large population inversion occurs. The shutter is then opened to release bursts of energy in brief bursts of about nanoseconds. The size of the spot produced will be around 8microns. It has a duration of few nanoseconds and produces energy of 10-20mj.

This method aims at obtaining short duration pulses at high peak power which is achieved by controlling the loop gain within the optical cavity. This high peak is achieved through very fast shutter that is located between the HR mirror and the active medium. The shutter system facilitaes in acheiving the energy in the laser cavity rod & further amplification on continuos pumping.

The total energy that is stored in the laser cavity is converted into light wave once the shutter is opened and this is called as 'Q-Switching'.

An ideal Q-switch should cause zero loop gain when it is closed and cause no loss in the cavity when it is opened.

We have two types of shutters to achieve this- 1) pocket cell polarizer assembly-good synchronization.

3) Bleachable dye- no synchronization.

Pocket cell polarizer is an optical assembly that is electrically switched which is transparent in one state and opaque in the other state to the light energy passing.

Bleachable dye is an organic dye interposed at shutter in the form of plastic film. The bleaching of the dye at high intensities makes it transparent and allowing the light energy to pass through it.

In mode-locking, the light is emitted in extremely short pulses due to an optical element which synchronizes all the modes. It has 3 pre-requisites-

i. The laser medium- more number of oscillating axial modes which require a broad transition.
ii. Mode separation should be small for which the laser cavity should be very long.
iii. Synchronization of phase relationship.

This synchronization is achieved through shutters. There are two types of shutters: Active mode locking and Passive mode locking.

Here in ophthalmology we use passive mode locking which uses the property of a saturable dye. The dye bleaches after absorbing the light pulses and becomes transparent. The dye must have the capability to bleach and recover within very short period of picoseconds.

It is difficult to maintain since it is very expensive, hence Q-locking mode is more extensively used.

Monomode & Multimode- we can compare multimode to a "shot gun" and mono-mode to a "rifle". In mono-mode, there is an aperture in the laser cavity which allows only a part of laser light energy to pass through it. This produces a fine spot with a narrow beam which is crucial in application where adjacent structures are to be safe-guarded.

Multi-mode has no aperture in the laser cavity, hence the energy produced is higher. The beam is wide with a more diffusely concentrated focus.

The YAG laser delivery system is mounted on a stereoscopic binocular slit lamp microscope that uses Helium-Neon co-axial laser aiming beam.

This laser helium-neon was introduced by **Javan & associates** in 1961.

There are 2 separate focussing beams for pulsed YAG laser, because-

In pulsed delivery, there is no delivery of laser in-between the pulses.

a) Helium neon laser is a visible red light of 632nm.
b) Nd-YAG is invisible as it has 1064nm wavelength.
c) The helium-neon pointer beam has continuos emission, though of a sub-threshold level.

This dual beam system is widely used since then. The 2 beams of Helium-neon superimpose to form a single sharp beam on the tissue. Hence, the focus is identified.

FOCUS OF THE AIMING BEAM-

1. The focus beam must be round and sharp

2. The beam should not flicker

3. Spot size should be as small as possible.

Abraham contact lens can be used to stabilise the eye for better focus. This lens increases the convergence angle from 16degrees to 24degrees and decreases the area of laser shot on the posterior capsule from 21microns to 14microns and increases the beam area on both retina and cornea preventing their damage.[52]

Photograph 3: Abraham lens

It is not always necessary that Nd-YAG laser beam and the helium-neon beam pointer coincide. In some delivery systems the Nd-YAG focus is 0.3mm behind the helium-neon laser focus. This difference is referred to as "off-set".It is done to minimize the damage to the IOL and the optical breakdown in such lasers occur in the anterior part of vitreous and produces a shock-wave anteriorly which propagates forward and ruptures the posterior capsule.

CRITICAL FOCUS-

1. Optical breakdown predictability is affected by media opacity.
2. Contact lens enhances the focus.
3. Predictability of optical breakdown is affected by induced astigmatism.
4. Chromatic aberrations result in laser beam being focussed posterior to the Helium neon pointer beam.
5. The focal point of helium-neon should be well defined.
6. Optical interfaces have less threshold for breakdown than uniform media.
7. Optical breakdown is non-linear.
8. By increasing the energy, the breakdown zone moves anteriorly.

9. Minimal energy is always advisable, as it helps in keeping the Helium-neon & YAG beam in appropriate positions.

Each pulse energy should be in the range of 0.8-2mj. Energy delivery should start with low energy and then gradually increased upto 2mj.

PATTERNS OF CAPSULOTOMIES-

a) Cruciate pattern
b) Christmas tree pattern
c) Hexagonal

CRUCIATE CAPSULOTOMY- it is most commonly done. The laser shots are started from 12'o clock position and gradually shot towards 6'o clock. From the 3'oclock proceeded towards 9'o clock. This way cruciate pattern is achieved. Any fragments of capsule hanging in the centre should be fired with laser shots.

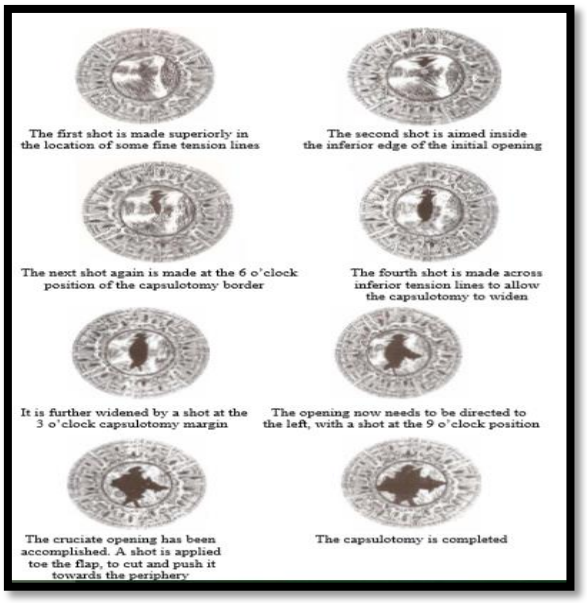


Figure10 : cruciate pattern of capsulotomy.

CHRISTMAS TREE PATTERN- This pattern is usually used when the IOL damage is inticipated (or) when the gap between the opacified posterior capsule and the IOL is too less. In this method, the laser is initially started at 12'o clock and proceeded towards 5'o clock and 7'o clock avoiding the central area. Any large overhanging chunks should be avoided.

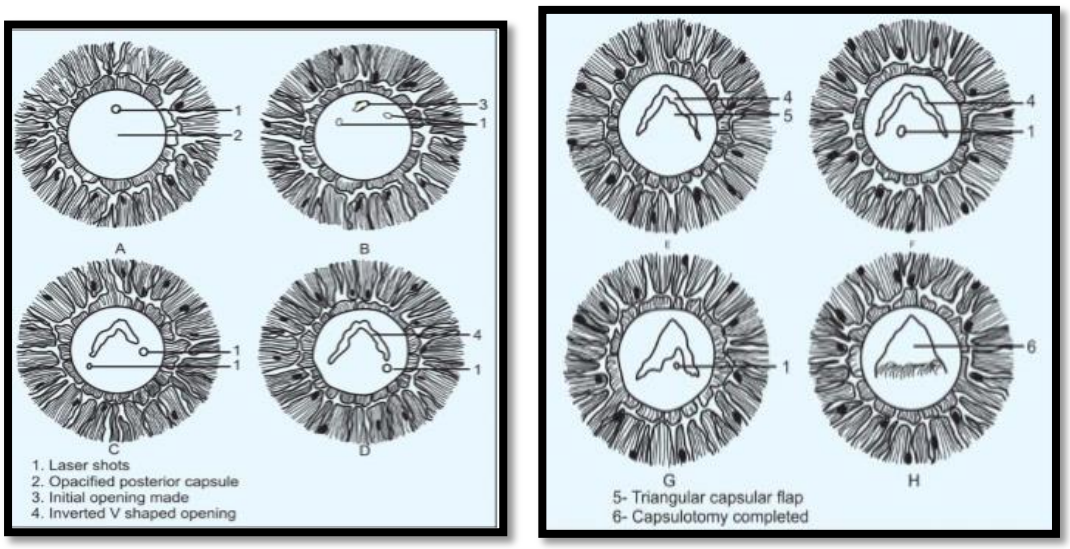


Figure11 &12: Christmas tree pattern of capsulotomy

Revised HEXAGONAL PATTERN OF I.HOWARD FINE: In this a symmetrical hexagonal capsulotomy with posteriorly scrolled edges are made. A sufficient area of capsulotomy is achieved by using high energy settings which push the margins posteriorly and away from the optic.

PRE-OPERATIVE ASSESSMENT

Ocular examination includes slit lamp evaluation and proper grading of the PCO, fundus examination with a direct & indirect ophthalmoscope to rule out any macular lesions which might contribute to the visual loss and intra-ocular pressure measurement. Any topical medications (or) systemic medications usage should be looked for.

It is tricky to judge the contribution of posterior capsular opacification to the visual loss. Other factors like cystoid macular oedema which are easily missed out due to the hazy media may pose difficulty post- laser and should be documented to avoid unnecessary laser. Any retinal breaks or holes might be missed due to poor visibility in dense PCO, but in mild/moderate PCO they have to be looked for.

Potential acuity meter and laser interferometer can penetrate mild to moderate opacities, but they are inaccurate in assessing cystoid macular oedema patients due to false positives.

PATIENT PREPARATION & PROCEDURE:

The patient is informed regarding the need for the procedure and proper **consent** is taken in the patients understandable language.

The procedure is explained in detail. The duration of the procedure is only a few minutes and is painless and simple. When the patient hears multiple small clicks during which he/she should remain steady and should maintain fixation.

The position and size of the pupil in day light illumination should be noted to avoid eccentric capsulotomy. A drop of topical 0.5% or 1% tropicamide (or) 2.5% phenylephrine is instilled . After adequate mydraisis a drop of topical anaesthetic is instilled into the conjunctival sac.

The patient is made to sit in a comfortable position by proper adjustment of the stool height, chin rest height and slit lamp height. The head is supported by fastening the strap behind the head. The procedure is performed in a dimly lit room.

The patient is advised to fixate on a target. Abraham contact lens is used to facilitate focussing. This lens have to be used with great care as it is a modified posterior pole lens and improper aiming may result in retinal damage.

The laser energy delivered is started with the minimum value of 0.8mj and gradually increased upto 2mj. The capsulotomy would be completed with 15-20 laser shots. A central marker laser shot is initially fired to avoid eccentric opening. The wrinkling of the capsule denotes the tension lines. A shot on the tension lines tends to stretch to become wider. Cruciate pattern of opening of 3-5mm is made. A wider capsulotomy allows adequate viewing of the retinal periphery.

POST-OPERATIVE CARE

- Topial antibiotic and steroid combination of eye drops are used sixth hourly for a week.
- Topical application of 0.5% Timolol (or) 1% Apraclonidine is advised for a week duration to avoid post-laser pressure rise.

COMPLICATIONS OF LASER CAPSULOTOMY

IOP elevation- It is a major post-YAG complication. It was estimated that early rise in IOP occurs during the first 3-4hr following laser. Studies proved that IOP returns to its pre-treatment level in 89% of cases within 1 week. An IOP rise of upto 30mmHg was seen in majority of cases[53].A persistent IOP rise was reported in 1% of cases.[54

Several thoughts were put **forward** by certain studies showing that Nd-YAG could cause persistently elevated IOP.**(55,56,57)**

There are several factors which contribute to the IOP rise which include pre-existing glaucoma, total laser energy used, capsulotomy size, placement of intra-ocular lens.(54)

The inflammatory cells, Capsular debris and the liquid vitreous are known to cause clogging of the trabecular meshwork.

An IOL fixated in the capsular bag and a small size of the capsulotomy opening acts as a mechanical barrier to the vitreous from entering the AC.

The high energy used for capsulotomy could cause damage to the trabecular meshwork leading to the outflow obstruction and consequently high IOP.

IOL related compllications-These include pitting, cracks and decentration of the IOL. It is the result of improper focussing or aiming if the patient is uncooperative. It causes significant trouble like glare and diminished vision if in the visual axis. This can be minimised with stabilizing the eyes with Abrahams contact lens, usage of minimal energy and avoiding multiple bursts. Deep focussing technique is advised to focus the beam in the anterior vitreous so that the shock wave radiates in the forward direction propelling towards the opacity.

Cystoid macular oedema-. It is estimated to occur in patients from 0.5% - 2.5%, because of disruption of anterior hyaloid face, & vitreous which is caused by the laser capsulotomy. An inflammatory response is produced due to the release of capsular debris which releases prostaglandins and leukotrienes.**(58,59)**

Retinal detachment ,holes and tears-it mainly occurs due to direct burn to the retina and due to disruption of the hyaloid face which may cause the vitreous to prolapse anteriorly and may exert traction on the vitreous base. There are several risk factors associated with the development of retinal detachment , which include- RD of other eye, axial length of greater than 26mm, age > 65yrs and lattice degeneration.

One potential mechanism leading to increased occurrence of retinal complications after capsulotomy was reported by Osterlin in 1971.Opening made to the capsule by any means allows hyaluronic acid to diffuse anteriorly out of vitreous causing vitreous instability which causes posterior vitreous detachment and its complications like retinal tear, retinal detachment and macular hole**.**[(60,61)]

Iritis- it is seen up to 6months following the capsulotomy and it occurs due to the inflammatory reaction that is elicited due to the laser energy. Any laser shot inadvertently shot on the iris tissue results in the anterior chamber inflammation and release of pigments into the chamber.

Hyphaema-any inadvertent shot onto iris surface (or) any pre-existing rubeosis of the iris may lead to hyphaema.

Endophthalmitis-Though rare, it may occur due to reactivation of priopionobacterium acnes which was quiescent in the capsular bag.

Secondary closure of capsulotomy- opacification after the initial laser capsulotomy is usually seen due to hyper-proliferation of the lens epithelial cells around capsulotomy opening an is seen in less than 1% patients.a repeat capsulotomy has to be performed in these patients.

CONTRAINDICATIONS FOR ND-YAG LASER CAPSULOTOMY

A. Absolute

a. Corneal leucoma/macular opacity.

b. Corneal surface disorders.

c. Corneal edema.

In above mentioned situations it is very difficult to visualise the target properly and if Nd: YAG capsulotomy is carried out results in unreliable optical breakdown.

d. Inability to fixate eye steadily.

e. Uncooperative or unwilling patients

B. Relative.

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

b. Diagnosed Cystoid Macular Edema (CME)

c. Eyes with very active inflammation

d. High risk group of patients for rhegmatogenous retinal detachment

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

– Treated prophylactically.

– Myopia.

– Having peripheral retinal degenerations, silent holes, etc

In the above situations, minimum possible laser energy per pulse and least number of laser shots should be fired to create a capsular window/opening adequate for visualisation of peripheral retina.

MATERIALS AND METHODS

TITLE OF THE STUDY:

"VISUAL OUTCMES AND COMPLICATIONS AFTER NEODYNIUM DOPED YTTRIUM ALUMINUM GARNET LASER CAPSULOTOMY IN POSTERIOR CAPSULAR OPACIFICATION"

SOURCE OF DATA:

89 eyes of paients with posterior capsular opacification fulfilling the inclusion criteria, attending the routine ophthalmology outpatient department, R.L.JALAPPA HOSPITAL AND RESEARCH CENTRE attached to SRI DEVERAJ URS MEDICAL COLLEGE , TAMAKA, KOLAR between December 2016 and May 2018.

SAMPLE SIZE:

A total number of 89 patients with posterior capsular opacification were evaluated for ocular and systemic co-morbidities.

INCLUSION CRITERIA:

Patients with posterior capsular opacification following cataract extraction with IOL implantation with decreased best corrected visual acuity of two or more Snellens lines. (Less than (or) equal to 6/12)

EXCLUSION CRITERIA:

1. Corneal scarring, dystrophies or degeneration.
2. Decentred IOL.
3. Thick membranous PCO not suitable for Nd –YAG capsulotomy.
4. Patients with uveitis, glaucoma, trauma.
5. Retinal and macular disorders.

After fulfilment of the criteria, informed and written consent is taken from the patient and they are further subjected to complete ocular and systemic evaluation.

PRE LASER EVALUATION-

1. Detailed history of any previous ocular diseases.
2. Surgical history obtained from the patients medical records (or) verbal history is noted which includes the duration of symptoms from the time of cataract surgery and history of cataract surgery.
3. Visual acuity recording by Snellen's or kannada charts & best corrected visual acuity.
4. Slit lamp examination.
5. Intra-ocular pressure recorded with Goldman applanation tonometry.
6. Fundus examination with both Direct and Indirect ophthalmoscopy.
7. Ultrasound B-scan was done in patients with hazy media.
8. Written informed consent was taken from all the patients.

PREPARATION OF THE PATIENT & PROCEDURE OF CAPSULOTOMY:

1. The procedure is explained to the patient and informed consent is obtained before dilating the patient.
2. A topical preparation of tropicamide 0.8% with phenylephrine hydrochloride 5% (chlorbutol 0.5 as preservative) is used for achieving mydriasis.
3. After adequate dilation, topical preparation of proparacaine 0.5% is instilled into the conjunctival sac.
4. The patient is made to sit comfortably by adjusting the stool height and chin rest height.
5. The head band is fastened to achieve stability to prevent gross head movements.
6. The patient is explained that he hears a click sound and a bright flash with each shot of laser with which he/she might feel a slight stinging sensation. He/she is told not to move and to maintain the gaze towards the fixation target.
7. An Abraham lens is placed gently for achieving better stability and focus.
8. VISULAS YAG III laser machine manufactured by ZEISS is used for the procedure. Initially the laser beam is properly focussed onto the posterior capsule or slightly behind it in the anterior vitreous and to start with, a minimal laser energy is used i.e 0.8mj- 1mj depending on the thickness of the PCO. The energy can be increased upto 2mj.
9. The initial shots are made along the tension lines and a cruciate pattern of opening is made with shots starting in the peripheral part at 12'o clock position gradually moving towards 6'o clock position. Horizontally from 3'o clock towards 9'o clock.
10. Any residual tags were shot to avoid freely floating fragments.
11. Adequate size of the opening is made, keeping a note on the cumulative energy used. A 3mm opening would open almost 50% of pupillary area and a 4mm opening opens almost 100%.

12. After the procedure, visual acuity and intraocular pressure was checked and noted after 1 hour.

16. The patient is advised to use:

a) 0.5%Timolol eye drops twice daily for a week, and

b) Topical antibiotic & steroid combination medication QID for a week.

c) Reviewed after 1 week, 1month and 3months to looked carefully for any signs of inflammation like Aqueous flare and cells, Hyphaema, Intraocular pressure rise, Cystoid macular oedema and Retinal detachment.

17. The patient was asked tovisit again after 1 day, 1week, 1month & 3months.

Photograph 4: performing Nd-YAG capsulotomy.

RESULTS

STATISTICAL ANALYSIS AND RESULTS[62-65]

Statistical analysis:

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Continuous data was represented as mean and standard deviation. **Paired t test** is the test of significance for paired data such as before and after surgery for quantitative data.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram, Pie diagram.

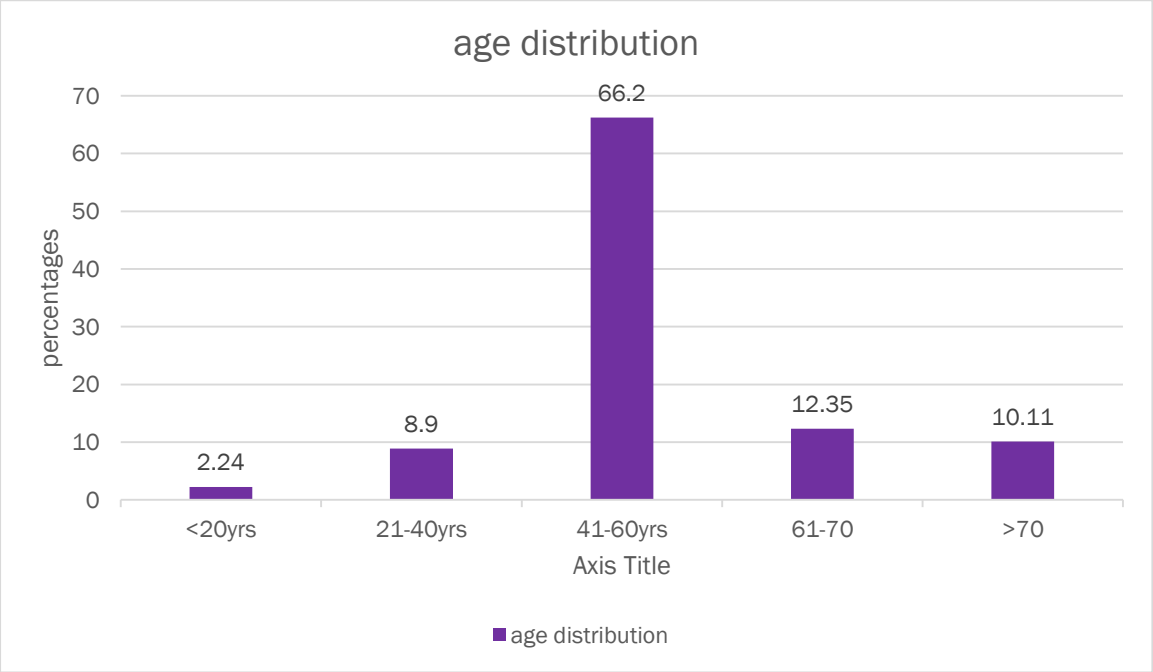
p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

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

Table 2: Age distribution of subjects in the study

AGE GROUP	NUMBER OF PATIENTS	%
<20 years	2	2.24%
21 to 40years	8	8.9%
41to 50 years	20	22.4%
51 to 60 years	39	43.8%
61 to 70 years	11	12.35%
>70 yrs	9	10.11%
Total	89	100.0%

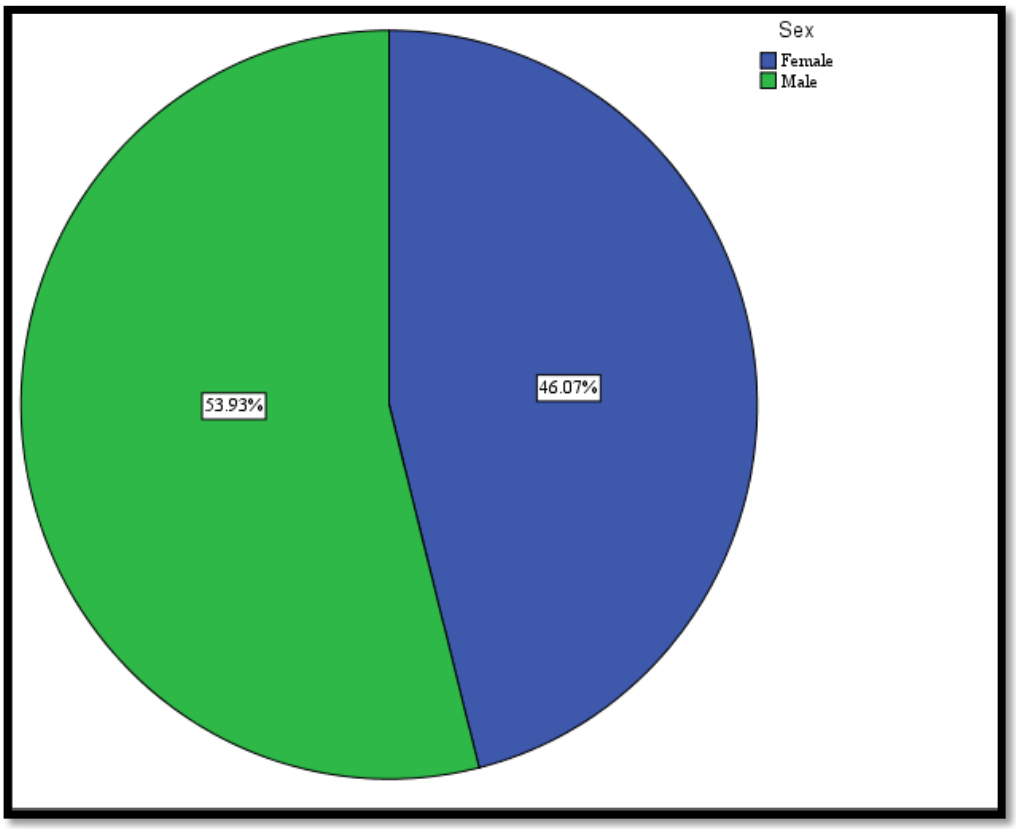
In our study 2.24% of patients were <20yrs,8.9% patients were in the age range of 20-40yrs,22.4% patients were in the range of 41-50yrs, 43.8% patients in 51-60yrs range ,12.35% patients were in the age range of 61-70yrs and 10.11% are over 70yrs.



Graph 1: Bar diagram showing Age distribution of subjects in the study

		Count	%
Sex	**Female**	41	46.1%
	Male	48	53.9%
	Total	89	100.0%

Table 3: sex distribution



Graph 2: Pie diagram showing Sex distribution of subjects

In our study of 89 patients enrolled in the study, 53.93% patients were males and 46.07% were females

		Count	%
Laterality	**Left Eye**	36	40.4%
	Right Eye	53	59.6%
	Total	89	100.0%

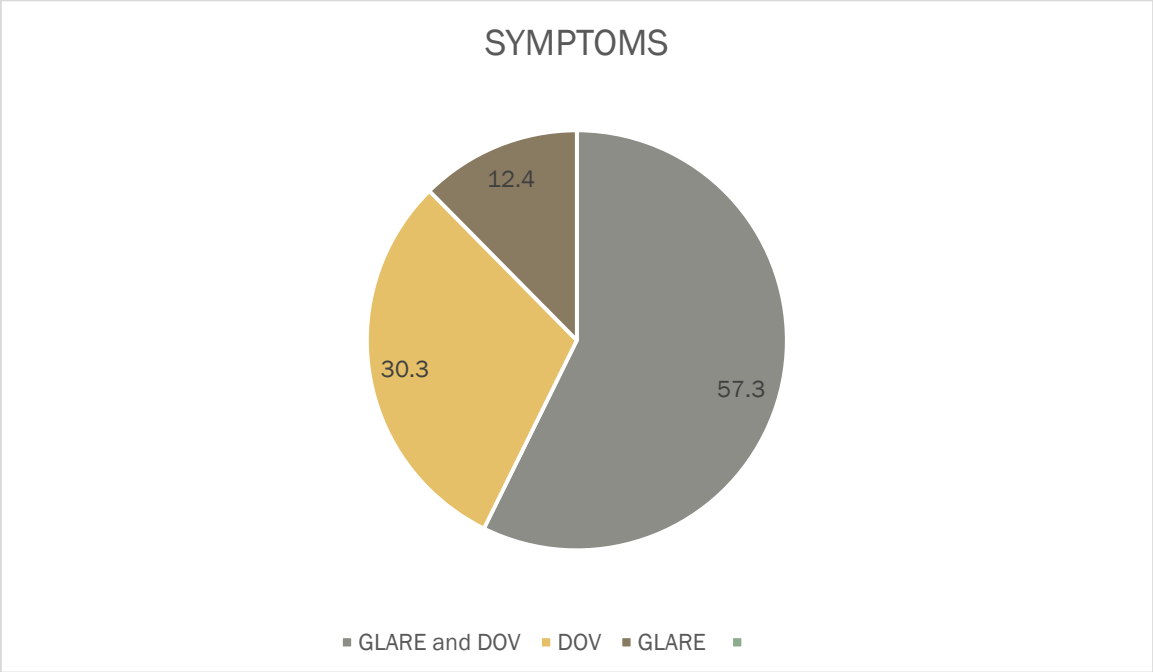
Table 4 : laterality

In the study 59.6% were right eye and 40.4% were left eye.

		Count	%
Symptoms	**DOV**	27	30.3%
	GLARE & DOV	51	57.3%
	GLARE	11	12.4%
	Total	89	100.0%

Table 5: Symptoms among subjects

In the study 57.3% presented with Glare and Diminution of Vision, 30.3% had Diminution of Vision, 12.4% presented with Glare

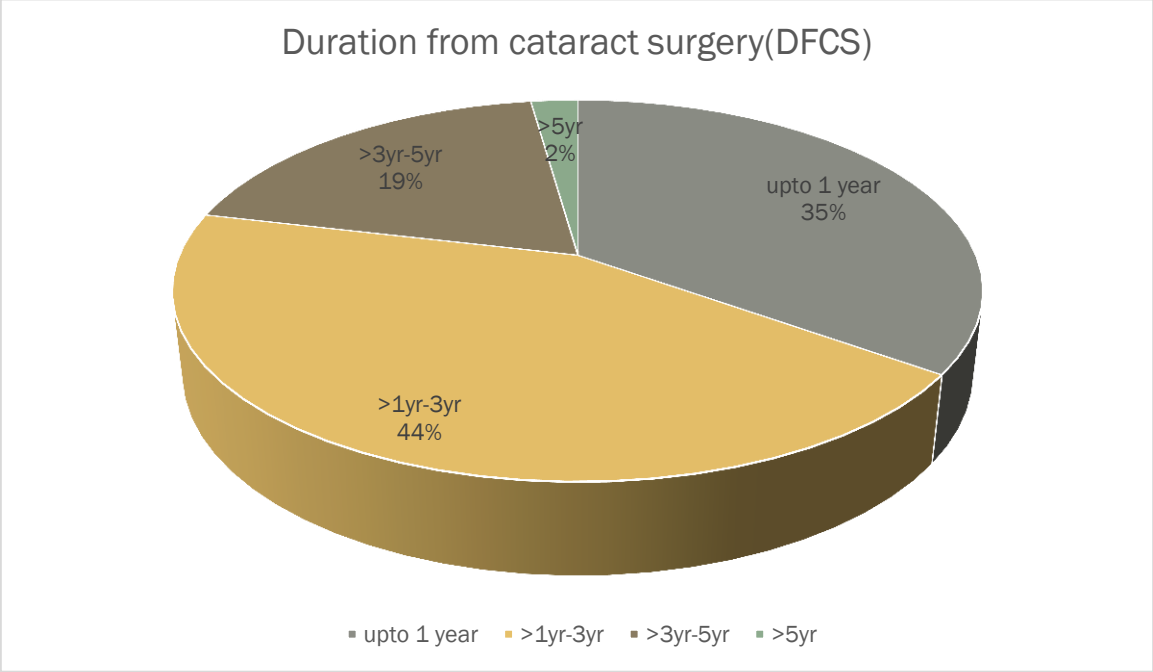


Graph 3 : Pie -diagram showing Symptoms among subjects

		Count	%
DFCS	**<12months**	31	34.83%
	12months to 36months	39	43.82%
	36months to 60months	17	19.10%
	>5years	2	2.24%
	Total	89	100.0%

Table 6: Duration from cataract surgery (DFCS) among subjects

In the study 34.8% had DFCS upto 1 year, 43.8% had DFCS from >12months to 3years, 19% had DFCS of>3yrs upto 5years and in 2.24% it is >5yrs.



Graph4: Pie diagram showing duration of DFCS among subjects

		Count	%
Type of PCO	EP	53	59.6%
	EP+FIB	11	12.4%
	FIB	25	28.1%

Table 7: Type of PCO among subjects

In our study type of PCO in 59.6% was EP, in 12.4% was EP + FIB and in 28.1% was FIB.

		Count	%
Grading of PCO	Mild	23	25.8%
	Moderate	33	37.1%
	Severe	33	37.1%

Table 8: Grading of PCO among subjects

In our study 25.8% had mild PCO, 37.1% had moderate PCO and 37.1% had severe PCO.

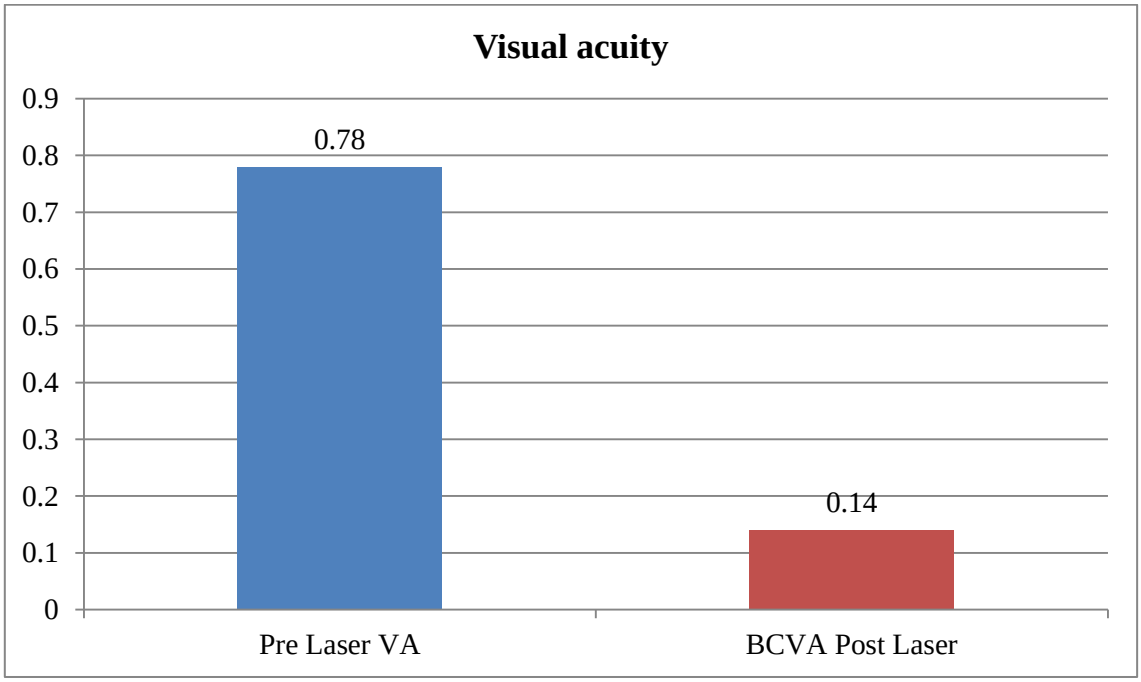
Visual acuity	Visual acuity pre laser.n(%)	VA immediately After laser,n(%)	VA 1 day post-laser.n(%)	VA 1 week Post-laser.n(%)	VA 1month Post-laser.n(%)	VA 3months post-laser.n(%)
6/6-6/9	0	70(78.6)	70 (78.6)	72(80.8)	73 (82)	75(84.2)
6/12-6/18	25 (28)	11(12.3)	11(12.3)	9(10.1)	9 (10.1)	10(11.2)
6/24-6/36	36 (40.4)	8.9(8.9)	8 (8.9)	8(8.9)	7 (7.86)	4(4.49)
6/60	20 (22.47)	0	0	0	0	0
<6/60	8 (8.9)	0	0	0	0	0

n = number of cases,%=percentage. **Table 9**: showing Visual acuity comparison between Pre Laser and Post Laser

	Mean	SD	P value
Pre Laser VA	0.78	0.36	
BCVA Post Laser	0.14	0.24	<0.001*

Table 10: Visual acuity comparison between Pre Laser and Post Laser in logMAR.

Pre Laser VA in Log MAR was 0.78 ± 0.36 which changed to 0.14 ± 0.24 at Post Laser. There was significant improvement in visual acuity post laser in the study.



Graph 5: Bar diagram showing Visual acuity comparison between Pre Laser and Post Laser visual acuity in logMAR after 4weeks following laser.

Spherical error(diopters)	number of patients,n=	Percentage %
+2.25 to +3.00	6	6.7
+1.25 to +2.00	24	26.9
+0.25 to +1.00	14	15.7
-0.25 to -1.00	22	24.7
-1.25 to -2.00	2	2.24
-2.25 to -3.00	4	4.49

Table11: Spherical error of the patients at the presentation.n= number of patients

At the time of presentation 67.3% are in the range of +2.00D to -1.00D.

26.9% patients showed spherical error of +1.25 to +2.00 and 15.7% patients showed a range of +0.25 to +1.00D. The total number of patients with hyperopic spherical error are 44 in number.

24.7% patients are in the range of -0.25 to -1.00D and 6.9% are in the range of -1.25 to -3.00D range. The total number of patients with myopic spherical error are 28 in number.

Spherical error(diopters)	No of patients,	Percentage
+2.25 to +3.00	4	4.49
+1.25 to +2.00	14	15.7
+0.25 to +1.00	30	33.6
-0.25 to -1.00	15	16.8
-1.25 to -2.00	5	5.6
-2.25 to -3.00	4	4.49

Table12 : spherical error shift seen in patients after Nd YAG laser capsulotomy

After capsulotomy 66.1% patients are in the range of +2.00 to -1.0D.

33.6% patients showed a range of +0.25 to +1.00D and 15.7% showed a range of +1.25 to +2.00D. the total number of patients with hyperopic spherical error after capsulotmy are 48 in number.

16.8% patients are in the range of -0.25 to -1.00D and 10% are in the range of -1.25 to -3.00D.the total number of patiets with myopic spherical error after the procedure are 24 in number.

Cylindrical error @ 180 $\pm 30^0$	Number of patients	Percentage
-0.25 to -1.00	15	16.8
-1.25 to -2.00	29	32.5
-2.25 to -3.00	7	7.8
-3.25 to -4.00	5	5.6
-4.25 to -5.00	0	0

Table13 : cylindrical error before the procedure

There are 56 patients who showed a cylindrical error. Majority of the patients(32.5%) are in the range of -1.25 to -2.00D followed by -2.25 to -3.00D(7.8%). 16.28% patients are in the -0.25 to -1.00D range.

There are 5 patients in the range of -3.25D to -4.00D.

Cylindrical error @ 180 $\pm 30^0$	No. of patients	Percentage
-0.25 to -1.00	15	16.8
-1.25 to -2.00	34	38.2
-2.25 to -3.00	7	7.8
-3.25 to -4.00	2	2.24
-4.25 to -5.00	0	0

Table14 : cylindrical error of the cases after laser

Majority of the patients are in the range of -1.25 to -2.00D followed by -2.25 to -3.00D range. There are 16.8% patients in the -0.25 to -1.00D range and 2 patients are in the -3.25 to -5.00D range.

Table 15: IOP comparison between Pre laser and Post Laser at 1 hr, 1 Week, 1 Month and 3 months

IOP	Mean	SD	P value
Pre Laser	14.38	2.31	
1hour	15.08	2.91	0.001*
1day	17.10	4.70	<0.001*
1 Week	14.54	2.25	0.052
1 Month	14.43	2.32	0.158
3 Months	14.43	2.32	0.158

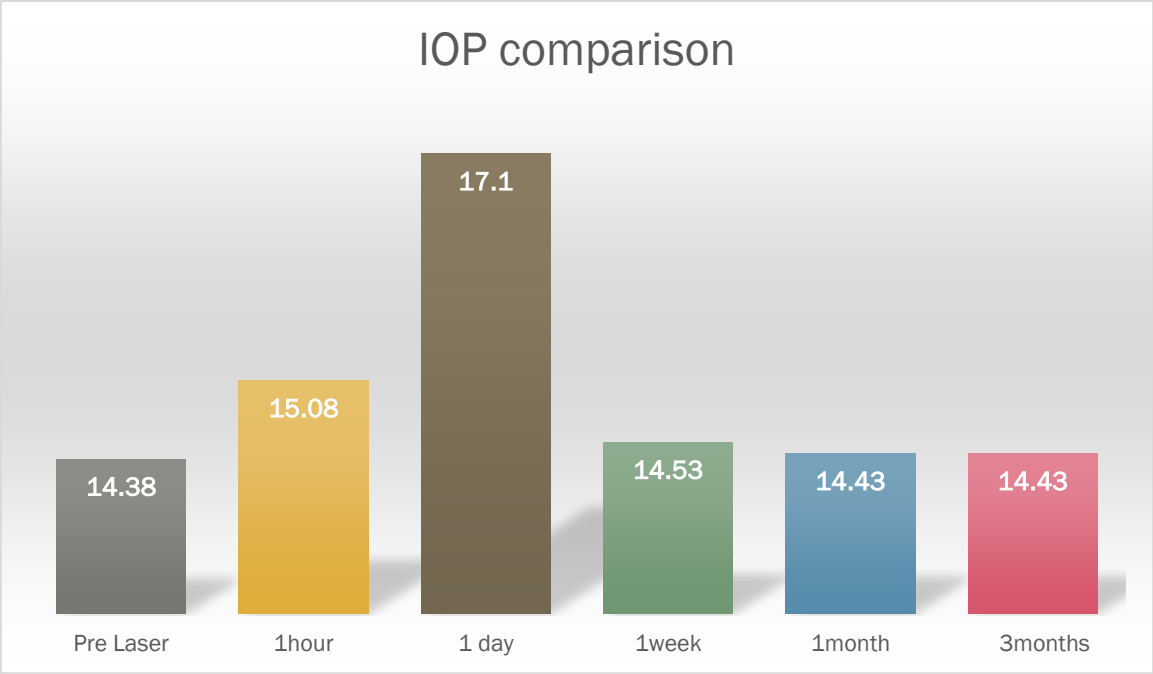
In the study Pre Laser IOP was 14.38 ± 2.31 mmHg, IOP following I hour after laser was 15.08 ± 2.91mm of Hg, 1day IOP was 17.10 ± 4.70 mmHg, 1 week was 14.54 ± 2.25 mmHg, 1 month was 14.43 ± 2.32 mmHg and 3 Months was 14.43 ± 2.32 mmHg.

There was significant increase in IOP at 1 hour and 1 day compared to Pre laser value. At 1 week, 1 month and 3 months there was no significant difference in IOP compared to pre laser value.

	Rise in IOP	<5mm of Hg rise n (%)	5-10mm of Hg rise n (%)	>10mm of Hg rise n(%)	Total N(%)
Total number of patients.	After 1hour	6(6.7)	9(10.1)	2(2.24)	17(19.1)
	After 1day	**6(6.70)**	**16(17.9)**	**10(11.1)**	32(35.9)
	1 week	5(5.6)	1(1.1)	0	6(6.7)
	1month	0	1(1.1)	0	1(1.1)
	3months	0	1(1.1)	0	1(1.1)

Table 16: IOP rise in mm of HG

Rise in IOP is usually transient and subsided within a week in majority of the patients. Persistent elevation in IOP is noted only in 1 patient over a period of 3months.



Graph 6: Bar diagram showing IOP comparison between Pre laser and Post Laser after 1hour,1day, 1 Week, 1 Month and 3 months.

Total energy used.(milli joules)	energy	Number of patients	Average IOP in mm of Hg.
Less than 50mj	<30	8	16.9
	30-50	43	
Greater than 50mj	50-70	27	17.27
	>70	11	

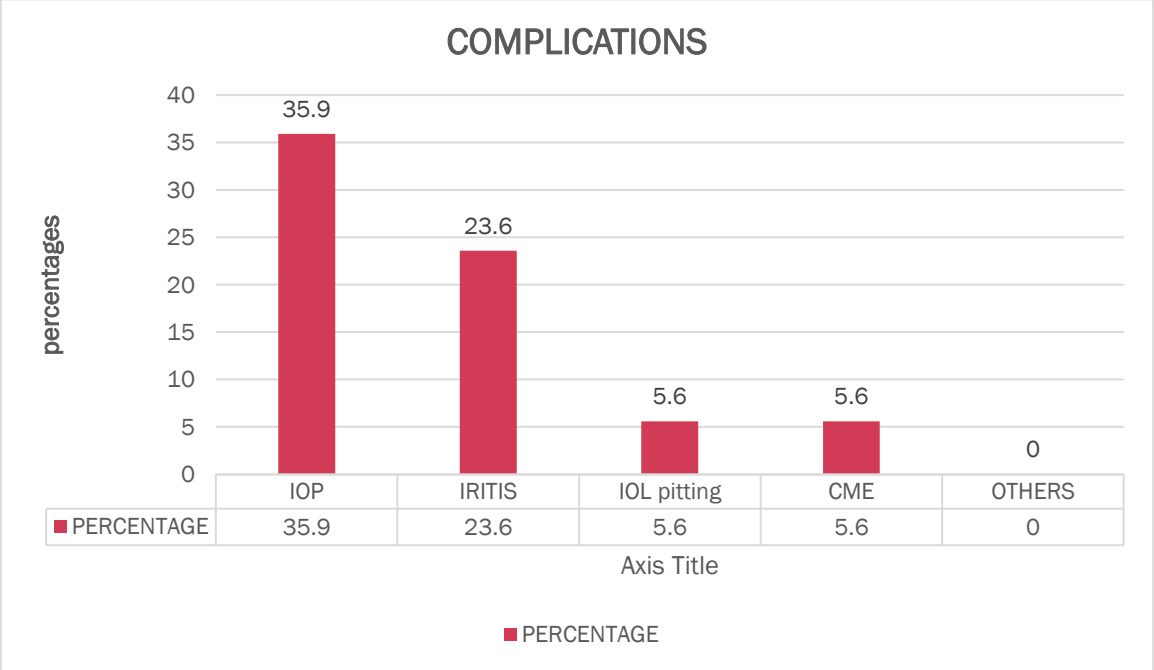
Table 17: Total energy used for laser.

According to the total energy used for effectively creating a capsulotomy,<50mj is used in 51 patients and >50mj is used in 38patients.The average IOP of the patients with <50mj and >50mj energy used was 16.9mmHg and 17.27mmHg respectively.

	Negative		Positive	
	Count	%	Count	%
IOP rise	57	64.1	32	35.9%
Iritis	68	76.4	21	23.6%
CME	84	94.4	5	5.6%
IOL Pitting	84	94.4	5	5.6%
IOL Movement	87	97.8	2	2.2%
Corneal Burns	89	100	0	0%
RD	89	100	0	0.0%
RH	89	100	0	0.0%
MH	89	100	0	0.0%
Endophthalmitis	89	100	0	0.0%
Sec Closure	89	100	0	0.0%

Table 18: complications

In the study35.9% patients had IOP rise, 23.6% had iritis, 5.6% had CME, 5.6% had IOL Pitting and none of them had corneal complications, RD, RH, MH, Endophthalmitis and Secondary closure.



Graph 7: Bar diagram showing Complications among subjects

DISCUSSION

In the modern era, with the advances in cataract surgery it has now become a refractive surgery. PCO is a major late post-operative complication of cataract surgery which impedes with the result of a successful surgery. With the advent of Nd YAG laser capsulotomy which is a simple, non-invasive outpatient procedure, the treatment of PCO is simplified.

In our study, 89 subjects with PCO were included and were treated with Nd-YAG laser capsulotomy in our institute. Our main aim is to note the improvement in visual acuity and study the complications over a 3month duration.

In the present study 53.9% of the patients who underwent laser capsulotomy were males and 46.1% were females which was in accordance to several other studies like Gopinath.et.al and Pankaj S.et.al. But it might not be a true reflection of high incidence among males due to relatively high outdoor activity among males which would have practically caused more discomfort to them than females. Several other studies like **SpaltonD.**et.al, **Aminollan**,et.al., and **Srinivas.**et.al showed that there is no gender predilection.(66)

The incidence of PCO gradually decreases as the age advances. In our study only 2 patients of age <20yrs were included. Out of 89 patients included in the study majority of the patients were in the age range of 51- 60 years contributing to 43.8% (39patients) followed by patients in the age range of 41-50years contributing to 22.4%(20 patients).

The average age group of the patients is 57yrs, because of more cataract surgeries being performed in that age group. This is in accordance with the study by **Emery.et.al.,** in which the average age of the patients developing PCO was 55years.**(67)**

Maximum number of patients needed laser capsulotomy within 36months of cataract surgery , accounting to 78.6% (70patients) . Majority of presented between the duration of 12months to 36months from cataract surgery.The history of cataract surgery was obtained either from the medical records submitted by the patients (or) simply with oral statement. This is in favour of study done by **Jagat Ram.et.al.** in which the mean time elapsed for the development of PCO after cataract surgery was 30months. **(68)**

Dangel.et.al, have reported that the average time as 27.6months following cataract extraction.**(69)**

Aminollah have found that most opacification had occurred between the period of 3months-18months with a mean of 16.3months. The longer is the duration of follow-up after cataract extraction, the higher is the incidence. **(70)**

In our study 59.5% patients had Elschnig pearl type of PCO, Fibrous type which was seen in 28.1% patients followed by combined type of PCO which accounted to 12.4%. A study by **Moreno Montanes** et.al**(71)** has shown 60.6% of pearl type and 39.4% of fibrosis type of PCO. Another study by **Ronald Holweger.et.al,** showed that 61.38% patients in his study had pearl type of PCO followed by fibrous type seen in 28.71% & mixed type seen in the remaining patients**(72).**Our study has similar distribution of PCO types as the above mentioned studies.

Though these 2 clinically distinguished types of PCO have different pathways of development both are associated with visual loss.

Based on the visibility of fundus, PCO is graded as mild, moderate and severe.

37.1% had severe PCO, 37.1% had moderate PCO and 25.8% had mild grade of PCO.

In the present study, 57.3% had both glare and diminished vision, 30.3% had complaints of only diminished vision and 12.4% had only glare as their symptoms.

A major parameter evaluated in our study is visual outcome after capsulotomy.

Pre Laser VA was 0.78 ± 0.36 which changed to 0.14 ± 0.24 after 1month of laser. There was a significant improvement in visual acuity post laser in the study. Visual acuity improved to 6/6 in 57, 6/9 in 18patients by the end of 3 months.

The best corrected visual acuity is better than 6/18 in 92.1% patients. This is in agreement with other studies.Visual acuity ≤ 6/24 and is seen in 7 patients i.e, 7.86%, but this has improved and visual acuity ≤6/24 is noted only in 4 patients(4.49%) by the end of 3months. This improvement is seen in patients with cystoid macular oedema due to the usage of topical non-steroidal anti-inflammatory drugs in only 2 patients.by the end of 3months there are 2 patients with unresolved CME and 2 patients with ARMD changes which were undetected pre-laser due to hazy media.

Not all patients gain a visual acuity of 6/6 due to the presence of undetected pre-existing ARMD changes which were subtle to be determined preoperatively, amblyopia and cystoid macular oedema. Visual improvement achieved in our study is in agreement with other studies.

Serial number	Study	Percentage of patients with VA >/= 6/18
1.	Our study	92.1
2.	Lias Georgalas	91.7
3.	Tariq M Aslam	95
4.	Davis durham	89.9
5.	Srinivas G S	92
6.	Paul V, MC Graw	90
7.	Hassain MT	90
8.	Jafar Dawood	93.3
9.	Wasserman.et.al	87.5

Table19: Showing visual acuity improvement of >/=6/18 as seen in various studies after 6weeks.([73-78])

In our study maximum patients i.e.24 (26.9%) were in the +0.25 to +1.00D spherical error range, whereas after laser procedure 33.6% were in that range. Patients with myopic spherical error reduced from 28 to 24 after the procedure. After laser maximum patients(50.4%) were in the -1.00DS to +1.00DS. This slight hyperopic shift can be attributed to the slight posterior movement of the IOL with the laser shots. A study by **Zaidi.et.al** has demonstrated a hyperopic shift in their patients[(79).] Another study by **Thronval.**et.al, did not observe any change in the refractive error in their patients.[(80)]

In our study there is no significant change noted in the astigmatism component after the procedure which is in con-cordance with **VioletteV, Jan Willem.et.al**.[(81)]

Elevated IOP is an anticipated complication following any anterior segment laser surgery. Though it is the most common complication following Nd-YAG, it is relatively transient.

Immediate rise in IOP following Nd-YAG capsulotomy has been shown in many studies. This is attributed to the clogging of the trabecular meshwork mechanically by the fragments formed due to laser photo-disruption of the posterior capsule.

In our study IOP elevation was seen in 19.1% patients 1hr after the procedure, 35.9% had raise in the IOP after 1 day. These numbers gradually declined over the next visits to 6.75% at the end of 1week & 1.1% at the end of 1 month and 3months.

In the study Pre Laser IOP was 14.38 ± 2.31 mmHg, IOP after 1hour was 15.08±2.91, 1 day IOP was 17.10 ± 4.70 mmHg, after 1 week it was 14.54 ± 2.25 mmHg, 1 month was 14.43 ± 2.32 mmHg and 3 Months was 14.43 ± 2.32 mmHg. With the usage of post laser topical beta blockers like Timolol 0.5% significantly brought down the IOP to the lower values within 1week duration.

There was significant increase in IOP at 1 hr and 24hrs compared to Pre laser value. At 1 week, 1 month and 3 months there was no significant difference in IOP compared to pre laser value. The rise in the intra-ocular pressure starts immediately after the procedure and remains elevated through the initial 1-2days and returns to baseline within 1 week.

The average rise in IOP was 5.4mmHg after 1hour of procedure and 7.6mmHg after 1day. Our results are in concordance with other studies like **Hassan.et.al** and **Kraff.et.al** who reported an average rise in IOP of 6mmHg and 3.5mmHg respectively.**(82)**

In our study, we have used Timolol 0.5% eydrops every 12th hourly for 1 week until the next visit. A study by **SinghM** has shown that patients receiving placebo had significant increase in IOP as compared to patients receiving topical hypotensives**(83)**

According to a study by **Steiner RF**.et.al., the mean intraocular pressure rise after ECCE was 5.4mm of Hg, which was statistically significant (p=0.018).

Richter.et.al, has shown a decrease in the aqueous outflow from 0.18microlit/min/mmHg before laser capsulotomy to 0.08microlit/min/mmHg at 4hrs after Nd-YAG.**(84)**

Akansha.et.al, should in her study that there is significant elevation of IOP in patients following laser capsulotomy. The baseline IOP was 14.16(S.D=3.15) which has risen to 15.52 within 24hrs.

Stark.et.al, reported an elevation in IOP of >5mmof Hg in 39% of their patients, a rise in IOP to >30mm of Hg is seen in 28% patients. Although the rise was transient and subsided in majority of the patients, it was persistently elevated in 2-3% of thepatients. IOP >30mmHg was found to be associated with 1 (or) more of following conditions as per Stark.[(54)]

1) Preoperative IOP was >20mm of Hg.
2) Glaucoma patients
3) Use of high total energy for Nd YAG laser.
4) Use of cycloplegics
5) Multiple Nd-YAG laser procedures.
6) Aphakia.

Aphakics, Glaucoma patients and ocular hypertensives are exluded from the study.It is a wel known fact that presence of IOL is an effective barrier to prevent the cortical matter from reaching the trabecular meshwork. Hence IOP elevation will be more in aphakics as compared to peudophakics.[82]

Intraocular pressure rise is an anticipated complication following Nd-YAG and is necessary to closely follow-up these cases.[(85-89)]

Relationship between IOP elevation and total energy used was well established in literature. ([90,91,92)] .In our study the average energy used was 50.04mj. Patients receiving <50mj were about 57.3% and those receiving >50mj were up to 42.7%. The average IOP in patients receiving total energy of less than 50mj was 16.9mmof Hg and the patients receiving >50mj

was 17.27mm of Hg, 24hours after laser. There is no significant correlation of energy used with the IOP spike in our study. However the IOP rise was within the normal range, hence they were observed without any further intervention.

However, there are certain studies which were against our observation which have postulated that healthy pseudophakic eyes do not show rise in IOP following Nd-YAG laser capsulotomy.[93]

In our study, Cystoid macular oedema was noted in 5 patients (5.6%) during the 1 month follow up which was clearly appreciated with a 90D examination. Patients were then started on topical NSAID drops for 1month.

During the 3month follow up visit, it was found that cystoid macular edema persisted only in 2 patients (2.25%). The relatively higher incidence of cystoid macular oedema in our study as compared to other studies like **Bath.et.al** and **Keates.et.al.,** might be associated with cataract surgery rather than Nd YAG laser itself as pre-laser FFA was not done in our study to exclude this possibility. [94]

There is substantial evidence that opening the posterior capsule results in Cystoid macular oedema . [95-98]

Development of CME can be attributed to the vitreous instability secondary to the hyaluronic acid and prostaglandin diffusion through the deficit in posterior capsule.

Another hypothesis by **Jampol** says that UV-A light generates free radicals which facilitates the production of prostaglandins, thus inducing inflammation.[99]

Lewis et al. has shown a low rate of CME when Nd YAG was delayed for >6 months from the initial cataract surgery with an IOL implant. In our study 2 of the 5 patients who had CME had underwent cataract surgery within 1 year,2 of them underwent within 1.5years and 1 patient had cataract surgery 5years back. [100,101]

A study by **Ari.et.al** showed that macular thickness was as significantly higher in patients receiving high energy for capsulotomy. However no such conclusion can be drawn from our study due to the non-availability of necessary equipment for evaluating the macular thickness.[102]

SERIAL NUMBER	STUDY	PERCENTAGE
1.	present study	5.6
2.	Gopinath et al	2
3.	Shankar Ganvit.et.al	3
4.	Keates.et.al	2.3
5.	Maqsood.A.B, Ather.M.T	9
6.	Bath PE	2.5
7.	Gore.et.al	4

Table20: showing comparision of incidence of CME in various studies.[94,103 - 107]

A study by Maqsood.et.al, resulted in cystoid macular oedema in 9% of study population which was higher than ours and they have concluded that Nd: YAG laser could have exacerbated the pre-existing cystoid macular oedema which could have occurred post-cataract surgery.

There are several studies which have demonstrated a relationship between Nd-YAG capsulotomy and incidence of retinal detachment.

S.NO	STUDIES	PERCENTAGE (%)
1	PRESENT STUDY	0
2	Steinert.et.al	0.89
3	Dardenne M.U et.al	1.6
4	Raza et.al	2
5	Jahn CE.et.al,	0.5
6	Powell SK	0.8
7	Ranta.P	2

TABLE 21- Comparision of several studies for incidence of retinal detachment([55,107-112])

In our study there are no cases of retinal detachment seen. Retinal detachment is usually seen within a duration of year. Our study has a follow-up period of 3months which might be a limiting factor in determining R.D in our patients.

Pitting of the IOL is noted in 5 patients (5.6%) which however did not lead to any significant visual deterioration in our study. IOL pitting is seen due to the uncooperativeness of the patient. Proper focussing onto the posterior capsule will prevent this complication.

Serial number	Studies	Percentage
1.	Present study	5.6
2.	VimalJV.et.al	8
3.	Ajithe K.O.et.al	2.2
2.	WassermanEL.et.al	9.5
3.	Gopinath et.al	20
4.	Maqsood.et.al	19.2

TABLE22: showing incidence of IOL related complications in various studies[113,114]

The incidence of IOL pitting was less in our study compared to many other studies, however it was higher than AjitheKO.et.al which showed a pitting of 2.2%.

In our study 23.6% had iritis after Nd:YAG laser capsulotomy which manifested as cells and flare in the anterior chamber on slit lamp examination. But the iritis subsided in majority of the cases within 1 week as our patients were routinely started on topical antibiotic & steroid medications QID for a week.

Gore et al. reported that 33.5% of patients had iritis in his study(**[107].**

Other complications like Hyphaema, Corneal damage, Retinal hole, Retinal haemorrhage and Endophthalmitis are not detected in our study, but other studies have reported their incidence.**[115]**

CONCLUSION

Cataract surgery has evolved into a refractive surgery achieving excellent results and any hindrance to the post-operative vision causes a major dissatisfaction among the patients. PCO is a major delayed post-operative complication following cataract surgery.

Nd-YAG laser capsulotomy is the gold standard treatment for posterior capsular opacification.

There is excellent improvement in visual acuity in majority of the patients. Care should be taken to identify the cause of low vision before capsulotomy . Inspite of proper and uneventful laser capsulotomy, few patients might not achieve a 6/6 visual acuity due to other causes like amblyopia & undetected subtle macular lesions which might be missed before the procedure

IOP monitoring is mandatory both before and after Nd-YAG laser capsulotomy. In majority of the patients, the IOP spikes are transient but in cases with prolonged rise of IOP, an additional medication might be needed and they are to be monitored for other signs of glaucoma.

Iritis which is seen in many patients is usually self limiting and transient.

Certain complications like retinal detachment and posterior capsular reopacification may occur after a long time.

A careful follow-up of the patients would give us an advantage of treating the complications immediately.

On the whole Nd-YAG is a safe and effective modality of treatment for posterior capsular opacification.

SUMMARY

In our study, a total of 89 patients are enrolled from the outpatient department of R.L.Jalappa attached to Sri Devaraj Urs Medical College from December 2016- april2018.

After a thorough pre-operative assessment, informed consent is obtained from the patient. Nd YAG laser capsultomy is performed and they are evaluated 1 hour after the procedure and then followed up for 3months with 4 subsequent visits at 1 day, 1week, 1 month and 3 months.

In our study 53.9% were males and 46.1% were females.

Visual acuity has improved to 6/6 -6/9 in 73 patients, 6/12 - 6/18 in 9 patients, 6/24 - 6/36 in 7 patients by the end of 1 month. Visual improvement is excellent and comparable to other studies.

Visual acuity of 6/24 – 6/36 is seen in 4 patients at the end of 3 months due to underlying ARMD changes in 2 patients which were missed during the preoperative evaluation due to hazy media and cystoid macular oedema in 2 patients.

Cystoid macular oedema was a contraindication for laser, as it would exacerbate the condition. However, CME was seen in 5 patients at the end of 1 month which resolved in 3 patients by the end of 3 months with the usage of topical NSAIDS QID. 2 patients had CME even at the end of 3months and their visual acuity was in the range of 6/24 – 6/36. This CME in our study was slightly higher when compared to other studies. FFA was not performed in the patients before laser, hence Nd-YAG might not be the sole cause, it might be associated with the cataract surgery.

Immediate and 1 day rise in IOP is noted in 32 patients which was transient and subsided during the subsequent visits. All the patients were routinely started on topical Timolol 0.5% irrespective of pre-laser IOP. High IOP patients at the end of 1 week were continued on

Betablockers for 1 more week. Persistent elevation of IOP was noted only in 1 patient who had 20mmHg at the end of 3months compared to pre-laser value of 14mmHg. This patient had no glaucomatous changes and the IOP was within normal range, hence no further intervention was needed for him.

IOL pitting was seen in 5 patients due to patient uncooperation during the procedure. However, the vision was close to normal in almost all the 5 patients. Hence no further intervention was needed.

Though transient and self-limiting iritis was noted in 21patients. As all the patients were routinely started on topical antibiotic & steroid combination drops, it resolved within the subsequent visits.

In our study we have not seen any cases of Retinal detachment, Corneal stromal haze/ burns, Retinal haemorrhage, Retinal holes, Endophthalmitis and cases of Secondary capsular opacification. The duration of follow-up might be a limiting factor in studying the incidence of retinal detachment and Re-opacification as they usually occur over 1 year after the procedure.

To conclude, Nd-YAG is a safe and effective procedure and it very important to look for any complications in the subsequent visits for timely intervention.

BIBLIOGRAPHY

1. Khairallah M, Kahloun R, Bourne R, Limburg H, Flaxman SR, Jonas JB, *et al*. Number of people blind or visually impaired by cataract worldwide and in world regions, 1990 to 2010. Invest Ophthalmol Vis Sci 2015;56:6762-9.

2. United Nations, Department of Economic and Social Affairs, Population Division. World Population Ageing; 2015

3. Norman S Jaffe, Mark S Jaffe, Gary F Jaffe, Cataract surgery and it's complications, 6th edition, Published by C. V. Mosby company Missouri, USA, 1997; 410.

4. Auffarth GU, Golescu A, Becker KA, et al. Quantification of posterior capsule opacification with round and sharp edge intraocular lenses. Ophthalmology. 2003;110: 772–780.

5. Auffarth GU, Peng Q. Posterior capsule opacification: Pathology, clinical evaluation and current means of prevention. Ophthalmic Practice. 2000;18(4):172–182)

6. Apple DJ, Peng Q, Visessook N, et al. Surgical prevention of posterior capsule opacification. Part 1: Progress in eliminating this complication of cataract surgery. J Cataract Refract Surg 2000;26:180–7

7.Apple DJ, Mamalis N, Lotfield K, et al. Complications of intraocular lenses. A historical and histopathological review. Surv Ophthalmol 1984;29;1–54.

8.Leiseberg TJ, Bourne WM, Ilstrup DM. Secondary surgical and neodymium-Yag laser discissions. Am J Ophthalmol 1985;100:510–5.

9.Terry AC, Stark WJ, Maumene AE, Fagadau W. Neodymium-Yag laser for posterior capsulotomy. Am J Ophthalmol 1983;92:716–20.

10. Schaumberg DA, Dana MR, Christen WG, Glynn RJ. A systemic overview of the incidence of posterior capsule opacification. Ophthalmology 1998;105:1213–21

11. Apple DJ, Peng Q, Visessook N, et al. Eradication of posterior capsule opacification: documentation of a marked decrease in Nd:YAG laser posterior capsulotomy rates noted in an analysis of 5416 pseudophakic human eyes obtained postmortem. Ophthalmology 2001;108:505–18.

12. Pankaj Soni, Akash Srivastava, Deepti Yadav.Nd-YAG laser posterior capsulotomy and visual outcomes. Indian Journal of Clinical and Experimental Ophthalmology, July-September,2016;2(3): 271-277.

13. Hayashi K, Hayashi H, Nakado F, Hayashi F, Correlation between posterior capsule opacification and visual function before and after Nd: YAG laser posterior capsulotomy. Am J Ophthalmol. 2003; 136(4):720-6.

14. Channell MM, Beckman H, Intraocular pressure changes after Nd: YAG laser posterior capsulotomy. Arch Ophthalmol 1984; 102:1024-26.

15. G S Gopinath1, K Satish1, Nirati Srivastava2, Savita Patil2, Raheela Afshan2.Visual Outcome and Complications of YAG Laser Therapy for Posterior Capsular Opacification Following Cataract Surgery. International Journal of Scientific Study | June 2015 | Vol 3 | Issue 3.

16. Peyman G.A., Sanders D.R., Goldberg M.F. (1987) Opacification of the posterior capsule, Principles and practice of ophthalmology Jaypee., 1st edition, 2, 617-618.

17. Gholam A, Paymen , Donald R, Sanders, Mortton F, Goldberg: Principles and practice of ophthalmology, Vol.1;1987:3-47.

18. Sneel R.S., Lemp M.A.Clinical anatomy of eye, second edition, Boston: Blackwell Sceince, Inc, 1998: 197-204.

19.Bron A.J, Tripathi.R.C, Tripathi.R.J. Wolff's anatomy of the eye and orbit 8th edition, London,Chapman and Hall;1997;411-422.

20. Patterson C.A, Delamere.N.A. The lens in Hart .W. M. Edn. Adlers physiology of the lens. 9th ed. St.Louis Moshy, 1992; 348-383.

21. Beebe DC. The lens. In: Kaufman PL, Alm A, eds. Adlers physiology of the eye: Clinical application. 11th ed. St Louis: Mosby; 2009:131-163.

22. Boulton ME, Rozanowska M, Wride M. Biophysics and age changes of the crystalline lens. In: Albert DM, jakobiec FA,eds. principles and practice of ophthalmology. 3rd ed. Philadelphia: saunders ; 2008:vol1, chapter 106.

23. J.R. Kuszak, John I. Clark, Kim E. Cooper, James L. Rae. Biology of lens. Jackobie.ch 104.

24. Kobayashi.S, Roy D, Spector A : sodium/potassium ATPase in normal and cataractous human lenses. Curr Eye Res 2:327, 1983.

25. Harding JJ : Free and protein bound glutathione in normal and cataractous human lenses. Biochem J.117:957, 1970.

26. Harding JJ, Heyningen RV. Drugs, including alcohol, that act as risk factors for cataract, and possible protection against cataract by aspirin-like analgesics and cyclopenthiazide. Br J Ophthalmol 1988;72:809-14.

27. Brocklebank JT, Harcourt RB, Meadow SR. Corticosteroid-induced cataracts in idiopathic nephrotic syndrome. Arch Dis Child 1982;53:30-4

28. Chylack LT Jr, Wolfe JK, Singer DM, et al: The lens opacities classification system III. The Longitudinal Study of Cataract Study Group. Arch Ophthalmol 1993; 111:831–836.

29. Taylor HR, West SK: A simple system for the clinical grading of lens opacities. Lens Res 1988; 5:175, by courtesy of Marcel Dekker, Inc.

30. Sparrow JM, Bron AJ, Brown NAP, et al: The Oxford clinical cataract classification and grading system. Int Ophthalmol 1986; 9:207.

31. Sasaki K, Shibata T, Obazawa H, et al: Classification system for cataracts: application by the Japanese Cooperative Cataract Epidemiology Study Group. Ophthalmic Res 1990; 22(Suppl 1):46.

32. AslamTMD,TaguriB,WadoodA. Quantifying posterior capsule opacification. ESCRS winter meeting 2002.

33. Kruger AJ, Schauersberger J,AbelaC,et al. Two year results: sharp versus rounded optic edgesbon silicone lenses. JCRS 2000;26:566-70.

34. Sellman TR, Lindstrom RL. Effect of a plano-convex posterior chamber lens on capsular opacification from Elschnigs pearl formation. JCRS.1988;14:68-72.

35. Legler UF, Apple DJ, Assia EI, et al. Inhibition of posterior capsule opacification: the effect of colchicine in a sustained drug delivery system. JCRS.1993;19:462-70.

36. IdrusS, Sangeeta.N, Satyabhama.M, Suchitra D. A clinical profile of visual outcome after Nd-YAG laser posterior capsulotomy in pseudophakic posterior capsular opacity in RIMS. JMS - Journal of Medical Society 26(1):3-6 · January 2012

37. C. Wertheimer, T. C. Kreutzer, M. Dirisamer et al., "Effect of femtosecond laser-assisted lens surgery on posterior capsule opacification in the human capsular bag in vitro," Acta Ophthalmologica, vol. 95, no. 2, pp. e85–e88, 2017.

38. N.Mamalis,A.S.Crandall,E.Linebarger,W.K.Sheffield,and M. J. Leidenix, "Effect of intraocular lens size on posterior capsule opacification after phacoemulsification," Journal of Cataract&RefractiveSurgery,vol.21,no.1,pp.99–102,1995.

39. O. Nishi and K. Nishi, "Intercapsular cataract surgery with lens epithelial cell removal. Part III: long-term follow-up of posterior capsular opacification," Journal of Cataract & Refractive Surgery, vol. 17, no. 2, pp. 218–220, 1991.

40. R. S. Joshi, "Postoperative posterior capsular striae and the posterior capsular opacification in patients implanted with two types of intraocular lens material," Indian Journal of Ophthalmology, vol. 65, no. 6, pp. 466–471, 2017.

41. Clark DS, Emery JM, Munsell MF. Inhibition of posterior capsule opacification with an immunotoxin specific for lens epithelial cells: 24 month clinical results. *Journal of Cataract & Refractive Surgery* 1998;24(12):1614-20

42. Meacock WR, Spalton DJ, Hollick EJ, Boyce JF, Barman S, Sanguinetti G. Double-masked prospective ocular safety study of a lens epithelial cell antibody to prevent posterior capsule opacification. *Journal of Cataract & Refractive Surgery* 2000;26(5):716-21.)

43. Mastropasqua L, Lobefalo L, Ciancaglini M, Ballone E, Gallenga PE. Heparin eyedrops to prevent posterior capsule opacification. *Journal of Cataract & Refractive Surgery* 1997;23(3):440-6.

44. I. M. Wormstone, C. S. Liu, J. M. Rakic, J. M. Marcantonio, G. F. Vrensen, and G. Duncan, "Human lens epithelial cell proliferation in a protein-free medium," Investigative Opthamology & Visual Science, vol. 38, no. 2, pp. 396–404, 1997.

45. Fankhauser F, RousselP,SteffenJ.Clinical studies on the efficacy of high power laser radiation upon some structures of the anterior segment of the eye. Int Ophthalmol.1981.May;3(3):129-39.

46. Francis L' Esperence (1983) Neodynium-YAG laser, Ophthalmic lasers. Mosby, 2nd edition, 79-83.

47. Spansrov A.F. (1981) ND:YAG Laser in ophthalmology, Laser and their applications, 129-132.

48. Thomos D. Duane. An introduction to basic laser physics. Clinical ophthalmology. Harper and raw publisher, clinical volume -1 chapter-69, 390-98.

49. Marshall J. (1982) Properties of laser light, Laser in ophthalmology Basic principal, 20-26.

50. Francis L' Esperence (1983) Neodynium-YAG laser, Ophthalmic lasers. Mosby, 2nd edition, 79-83.

51. Spansrov A.F. (1981) ND:YAG Laser in ophthalmology, Laser and their applications, 129-132.

52.Albert D.M., Jackobiec F.A. (1994) Use of contact lens for laser delivery, Principle and practice of Ophthalmology, 3, 1600.)

53. Terry, A. C., Stark, W. J., Maumenee, A. E., & Fagadau, W. (1983). Neodymium-Yag Laser for Posterior Capsulotomy. American Journal of Ophthalmology, 96(6), 716–720).

54. Stark W J , Worthen D, Holladay JT, Murray G,.Neodynium YAG lasers:an FDA report.

55. Steinert.R.F , Puliafito, C. A., Kumar, S. R., Dudak, S. D., & Patel, S. (1991). Cystoid Macular Edema, Retinal Detachment, and Glaucoma after Nd:YAG Laser Posterior Capsulotomy. American Journal of Ophthalmology, 112(4), 373–380)

56. .Jahn, C. E., & Emke, M. (1996). Long-Term Elevation of Intraocular Pressure after Neodymium: YAG Laser Posterior Capsulotomy. Ophthalmologica, 210(2), 85–89.)

57.Kurata, F., Krupin, T., Sinclair, S., & Karp, L. (1984). Progressive Glaucomatous Visual Field Loss after Neodymium-Yag Laser Capsulotomy. American Journal of Ophthalmology, 98(5), 632–634).

58. Liesegang, T. J., Bourne, W. M., & Ilstrup, D. M. (1985). Secondary Surgical and Neodymium-YAG Laser Discissions. American Journal of Ophthalmology, 100(4), 510–519.

59. Murill et al,1995). (Jampol, L. M. (1989). Cystoid Macular Edema Following Cataract Surgery-Reply. Archives of Ophthalmology, 107(2), 166.

60. Winslow, R. L., & Taylor, B. C. (1985). Retinal Complications Following YAG Laser Capsulotomy. Ophthalmology, 92(6), 785–789.

61. Blacharski, P. A., & Newsome, D. A. (1988). Bilateral Macular Holes after Nd:YAG Laser Posterior Capsulotomy. American Journal of Ophthalmology, 105(4), 417–418.

62. Gaddis, ML, Gaddis, GM. Introduction to biostatistics: Part 4, Statistical inference techniques in hypothesis testing. *Ann Emerg Med*. 1990;19:820–825.

63. Patra P. Sample size in clinical research, the number we need. Int J Med Sci Public Health. 2012;1:5–9.

64. Sunder Rao P S S , Richard J (2006) : An Introduction to Biostatistics, A manual for students in health sciences , New Delhi: Prentice hall of India. 4th edition, 86-160.

65. Elenbaas, RM, Elenbaas, JK, Cuddy, PG. Evaluating the medical literature, part II: Statistical analysis.*Ann Emerg Med*. 1983;12:610–6.

66.SpaltonDJ. Posterior capsular opacification after cataract surgery.Eye(London).1999 july, 13(pt 3b);489-92. Review.

67. JM.Emery, K A.Wilhelmus,Stanley Rosenberg,complications of phacoemulsification.feb 1978.volume85,issue 2, Pages 141–150.

68. Jagat Ram, Sunil k,JaspreetS ,sourabhS, Nd:YAG laser capsulotomy rates following implantation of square-edged intraocular lenses: polymethyl methacrylate versus silicone versus acrylic. April 2009,Volume 44, Issue 2, Pages 160–164.

69. DangelME,KirkhamSM,PhippsMJ. Posterior capsule opacification in extra capsular cataract extraction and the triple procedure:a comparative study.Ophth sur.1994;104:727-39.

70. Aminollah.N. Effect of two capsulotomy methods on posterior capsule opacification after cataract surgery

71.MorenoJM,AuroraA,Miguel.J.M.objective quantification of PCO after cataract surgery with OCT.Inv Oph & visual science.Nov 2005, vol 46, 3999- 4006.

72. Ronald R Holweger, Babak Marefat. Intraocular pressure changes after neodymium: YAG capsulotomy. JCRS, Feb 1997.Vol 23, Issue 1, p115-121.

73.Georgalas I, Petrou P, Kalantzis G, Papaconstantinou D, Koutsandrea C, Ladas I. Nd: YAG capsulotomy for posterior capsule opacification after combined clear corneal

phacoemulsification and vitrectomy.Ther Clin Risk Manag. 2009 Feb;5(1):133-7. Epub 2009 Mar 26.)

74. Tariq M Aslam, HilaryD, BaljeanD. Use of Nd YAG laser capsulotomy. Survey of Ophthalmology,Dec 2003 Vol. 48, Issue 6, p594–612.

75. Durham DG and Gills JP. Three thousand YAG lasers in posterior capsulotomies: an analysis of complications and comparison to polishing and surgical discussions. Transactions of the American Ophthalmological Society 1985;83:218-235.

76. Paul V. McGraw. Donal Brosnahan,Barry Winn and David Whitaker. Assessment of Retinal-Neural Function Before Neodymium:YAG Laser Capsulotomy. Invest Ophthalmol Vis Sci. 1995;36:1155-1.

77. Hasan KS, Adhi MI, Aziz M, et al. Nd:YAG Laser Posterior Capsulotomy. Pak J Ophthalmol. 1996;12: 3-7.

78. Wasserman EL, Axt JC, Sheets JH. Neodymium:YAG laser posterior capsulotomy. J Am Intraocul Implant Soc. 1985 May;11(3):245-8.

79. Zaidi M, Askari NS. Effect of Nd:YAG laser posterior capsulotomy on anterior chamber depth, Intraocular pressure, and refractive status. J Cataract Refract Surg. 2000;26(8):1183–89.)

80. Thornval P, Naeser K. Refraction and anterior chamber depth before and after neodymium:YAG laser treatment for posterior capsule opacification in pseudophakic eyes: A prospective study. Journal of Cataract & Refractive Surgery. 1995;21(4):457–60.

81. VioletteV, van der Linden J, Nieuwendaal C, van der Meulen I, Mourits M, Lapid-Gortzak R. Effect of Nd:YAG laser capsulotomy on refraction in multifocal apodized diffractive pseudophakia. Journal of Refractive Surgery. 2012;28(8):545–51.

82. Kraff CM et.al. Intraocular pressure and the corneal endothelium after Nd: YAG laser posterior capsulotomy, Relative effects of aphakia and pseudophakia. Arch Ophthal 1985; 103: 511-514 .

83. Singh M. Anterior Segment Nd:YAG Laser Procedures: to Study intraocular pressure spikes and their prevention. Delhi Journal of Ophthalmology. 2015;26(2):2454–784.

84. Richter CU, Arzeno G, Pappas HR, Steinert RF, Puliafito C, Epstein DL. Intraocular pressure elevation following Nd:YAG laser posterior capsulotomy. Ophthalmology. 1985;92(5):636–40 .

85. Channel MM, Beckman H. Intraocular pressure changes after neodymium-YAG laser posterior capsulotomy. Arch Ophthalmol. 1984;102:1024-1026.

86. Schubert HD, Morris WJ, Troken SL, Balazs EA. The role of the vitreous in the intraocular pressure rise after neodymium-YAG laser capsulotomy. Arch Ophthalmol. 1985;103:1538-1542.

87. Slomovic AR, Parrish RK. Acute elevations of intraocular pressure following Nd:YAG laser posterior capsulotomy. Ophthalmology. 1985; 92:973-976.

88. Flohr MJ, Robin AL, KelleyJS. Earlycomplications following Q-switched neodymium-YAG laser posterior capsulotomy. Ophthalmology. 1985;92:360-363.

89. Pollack IP, Brown RH, Crandall AS, Robin AL, Stewart RH, White GL. Prevention ofthe rise in intraocular pressure following neodymium-YAG posterior capsulotomy using topical 1% apraclonidine. Arch Ophthalmol. 1988;106:754-757.)

90. Waseem M, Khan H. Association of raised intraocular pressure and its correlation to energy used with raised versus normal intraocular pressure following nd yag laser

posterior capsulotomy in pseudophakes. Journal of the College of Physicians and Surgeons Pakistan. 2010;20(8):524–27.

91. NiharikaKS, SriyaS. Study of variation in intraocular pressure spike following Nd-YAG laser capsulotomy. J Clin Diag Res. 2016 Dec; 10(12): NC09–NC12.

92.Ari S, Cingü AK, Sahin A, Çinar Y, Çaça I. The effects of Nd: YAG laser posterior capsulotomy on macular thickness, intraocular pressure, and visual acuity. Ophthalmic Surg Lasers Imaging. 2012;43:395–400.

93. Shani L, David R, Tessler Z, Rosen S, Schneck M, Yassur Y. Intraocular pressure after neodymium : YAG laser treatments in the anterior segment. Journal of Cataract and Refractive Surgery. 1994;20(4):455–58.

94. Bath, P. E., & Fankhauser, F. (1986). Long-term results of Nd:YAG laser posterior capsulotomy with the Swiss laser. Journal of Cataract & Refractive Surgery, 12(2), 150–153.

95. Kratz RP. Results of phacoemulsification by Kelman and Girard techniques. In: Douvas NG, Hartstein J, Jaffe MS, eds. Symposium on Cataracts: Transaction of the New Orleans Academy of Ophthalmology. StLouis: CV Mosby Co., 1979; 196-200.

96. Sorr EM, Everett WB, Hurite FG: Incidence of fluorescein angiegraphic subclinical macular edema following phacoemulsification of senile cataracts. Ophthalmology 1979; 86:2013-8.

97. Percival P. Clinical factors relating to cystoid macular edema after lens implantation. Am lntraocullmplant Soc J 1981; 7:43-5.

98. Kraft MC, Sanders DR, Jampol LM, Lieberman H. Effect of primary capsulotomy with extracapsular surgery on the incidence of pseudophakic cystoid macular edema. Am J Ophthalmol1984; 98:166-70.

99. JampolM, Goldberg.MF,JednockN. Retinal damage from a Q-switched YAG laser. Am J Ophthalmol.1983; 96:326-9.

100. Lewis H, Singer TR, Hanscom TA, Straatsma BR. A prospective study of cystoid macular edema after neodymium: YAG laser posterior capsulotomy. Ophthalmology. 1987 May;94(5):478–82.

101. Giocanti-Aure´gan A, Tilleul J, Rohart C, et al. OCT measurement of the impact of Nd:YAG laser capsulotomy on foveal thickness. J Fr Ophtalmol. 2011 Nov;34(9):641–6. Doi: 10.1016/j.jfo.2011.02.020.

102. Ari S, Cingu AK, Sahin A, inar YC, Caca I. The effects of Nd:YAG laser posterior capsulotomy on macular thickness, intraocular pressure, and visual acuity. Ophthalmic Surg Lasers Imaging. 2012 Sep-Oct;43(5):395–40.

103.Gopinath GS, Satish K, Srivastava N, Patil S, Afshan R. Visual Outcome and Complications of YAG Laser Therapy for Posterior Capsular Opacification Following Cataract Surgery. Int J Sci Stud 2015;3(3):65-68.

104. ShankarG, Meera S , UtkarshP, Aanal S, Priya J,Niyant P.Visual outcome and complications following Nd:YAG laser posterior capsulotomy.J Int MedRes.2015; 2(2):31-35.

105. Keates RH, Steinert RF, Puliafito CA, Maxwell SK. Long-term follow-up of Nd:YAG laser posterior capsulotomy.J Am Intraocul Implant Soc 1984;10:164-8.

106. Maqsood A Burq, Ather M Taqui. Frequency of Retinal Detachment and Other Complications after Neodymium: Yag Laser Capsulotomy. Vol. 58, No. 10, October 2008.

107. Gore.VS. The study of complications of Nd:YAG laser capsulotomy. Klin Monbl Augenheilkd. 1994 May;204(5):286–7.

108. Dardenne, M.-U., Gerten, G.-J., Kokkas, K., & Kermani, O. (1989). Retrospective study of retinal detachment following neodymium:YAG laser posterior capsulotomy. Journal of Cataract & Refractive Surgery, 15(6), 676–680.

109. Raza A. Complications after Nd:Yag posterior capsulotomy. Journal of Rawalpindi Medical College. 2007;11:27–29.

110. Jahn CE, Richter J, Jahn AH, Kremer G, Kron M. Pseudophakic retinal detachment after uneventful phacoemulsification and subsequent neodymium: YAG capsulotomy for capsule opacification. J Cataract Refract Surg 2003; 29:925-9.

111. Powell SK, Olson RJ. Incidence of retinal detachment after cataract surgery and neodymium: YAG laser capsulotomy. J Cataract Refract Surg 1995; 21:132-5.

112. Ranta P, Tommila P, Kivela T. Retinal breaks and detachment after neodymium: YAG laser posterior capsulotomy: five-year incidence in a prospective cohort. J Cataract Refract Surg 2004; 30:58-66.

113. VimalJV, LaxmiD, BholeshR.To Study the Visual Outcome After ND:YAG Laser Posterior Capsulotomy. IJSR. Oct2016. Vol : 5. Issue:10. pg 55-7.

114. Ajite K.O., Ajayi I.A., Omotoye O.J and FadamiroC.O.,Visual outcome of patients with posterior capsular opacification Treated with Nd:YAG Laser Journal of medicine and medical research vol 1(4)december 2013:23-27.

115. Carlson AW, Koch DD. Endophthalmitis following Nd: YAG laser posterior capsulotomy. Ophthalmic Surg 1988;19: 168-70.

ANNEXURE I

STUDY PROFORMA

Case no: Date:

Name: IP/OP no:

Age: DOA:

Sex: DOS:

Occupation:

Address:

Brief history:

Past history:

Family history:

Personal history:

GPE:

Vital signs:

Pulse:- RR:-

BP:- Temp:-

Systemic examination:

a) CVS

b) CNS

c)

c) Respiratory

d) GIT

OCULAR EXAMINATION		
TESTS	OD	OS
1.HEAD POSTURE 2.OCULAR POSTURE 3.FACIAL SYMMETRY		
4.EXTRAOCULAR MOVEMENTS a) Ductions b) versions		
5.VISUAL ACUITY a) Distant b) Near		
6.ANTERIOR SEGMENT a. lids and adnexa b. Conjunctiva c. Cornea d. Anterior chamber e. Iris f. Pupil g. IOL- h. PCO type		
6.FUNDUS a. Direct ophthalmoscopy b. Indirect ophthalmoscopy		
8.INTRAOCULAR PRESSURE		

9.B-SCAN ULTRASONOGRAPHY		
10.INTRAOCULAR LENS TYPE & POWER		
11.TIME PERIOD BETWEEN CATARACT SURGERY AND PCO		
12.Energy used for capsulotomy		

POST ND-YAG LASER CAPSULOTOMY RESULTS

VISUAL ACUITY

VISUAL ACUITY	IMMEDIATELY	1ST DAY	1WEEK	1MONTH	3MONTHS
BCVA(distant)					
BCVA(Near)					
Refraction					

POST ND-YAG CAPSULOTOMY COMPLICATIONS

S.NO	COMPLICATIONS	1Hr	1day	1week	1month	3months
1	Corneal injury					
2	IOL movement & refractive change					
3	IOL pitting					
4	Iritis/ Uveitis					
5	Raise in IOP					

6	Cystoid macular oedema					
7	Retinal tear & detachment					
8	Retinal hemorrhage					
9	Macular hole					
10	Endocapsular Endophthalmitis					
11	Secondary closure of capsulotomy aperture					

ANNEXURE II

INFORMED CONSENT FORM

"Visual outcomes and complications after Neodynium Doped Yttrium Aluminum Garnet laser capsulotomy in posterior capsular opacification".

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

I understand the purpose of this study, the risks and benefits of Nd-YAG capsulotomy in PCO patients and the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

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

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

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

Subject name & signature/thumb impression

Date:

Name & signature of witness

Date:

Name & signature of person obtaining consent:

Date:

ANNEXURE III

IMAGES

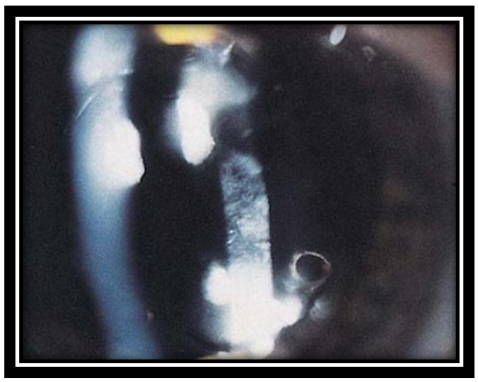


Image1: Fibrous PCO before laser capsulotomy.

Image 2: fibrous PCO after Nd-YAG laser capsulotomy.

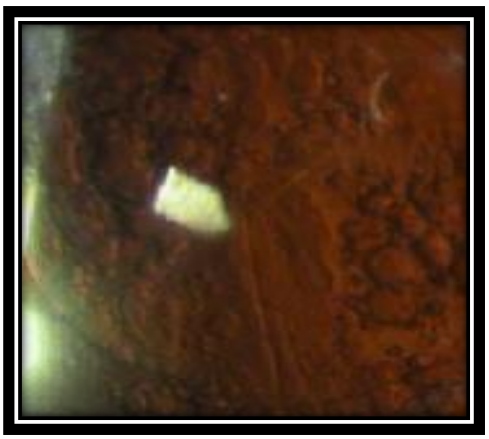


Image3: showing Elschnig's pearl type of PCO before laser.

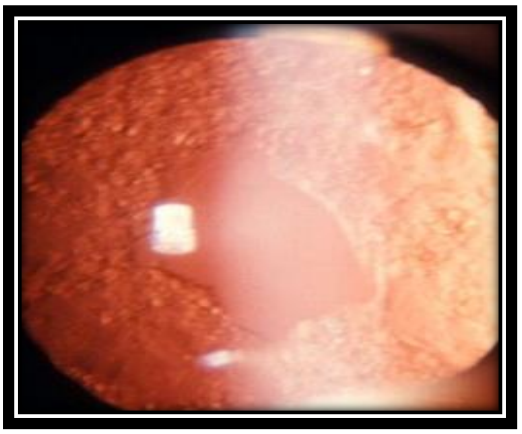


Image4: showing posterior capsular opening post-laser

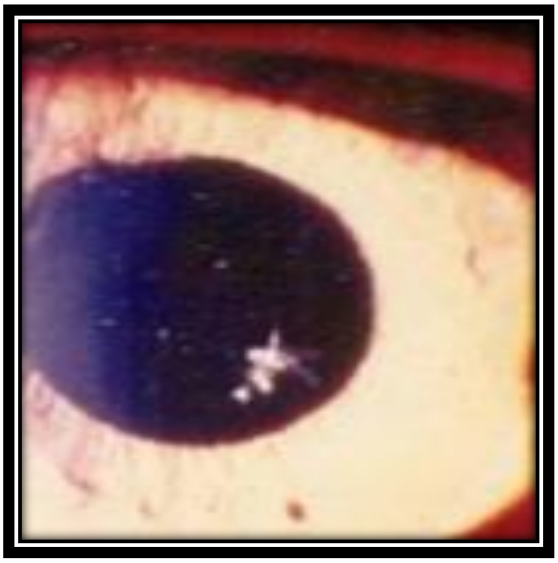


Image5 : showing IOL pitting post-laser

ANNEXURE IV

KEY TO THE MASTER CHART

Sr.no- Serial number

IP/OP- inpatient number/ out patient number

M – Male

F -- Female

RE --Right eye

LE -- Left eye

DOV—Diminution of vision

DFCS—Duration from cataract surgery

Yr—Year

Mon—Month

Pre-laserVA—Pre laser visual acuity

Pre-laser IOP—pre laser intraocular pressure

EP—Elschnigs pearl

FIB—Fibrous type of PCO

Mod—moderate

Sev—severe

Tot.ene—total energy

BCVA—Best corrected visual acuity

CME—Cystoid macular edema

R.D—Retinal detachment

R.H—Retinal haemorrhage

M.H—Macular hole

Endoph—endophthalmitis

Sec.clo—Secondary closure

ARMD--Age-related macular degeneration.

Sr No	IP/OP	AGE	SEX	LATERALITY	SYMPTOMS	DFCS	PRE-LASER VA	pre-refrac	PRE .L.IOP	TYPE OF PCO	GRADING OF PCO	post laser REF	TOT.EN(mj)	BCVA day 1	BCVA POST.LASER (1 week)	BCVA POST. LASER(1month)	BCVA 3months	IOP after 1hr	IOP day 1	IOP 1WK	IOP 1M	IOP 3M	CORNEAL BURNS	IOL MOVT	IOL PITTING	IRITIS 1day	IRITIS 1wk	CME(1mon)	CME 3mon	R.D	R.H	MH	ENDOPH	SEC.CLO	OTHERS
1	377526	60	M	RE	GLARE,DOV	2yr	0.3	+1.25DS	14	EP	mild	-0.25DS	30	0	0	0	0	14	18	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
2	377516	52	M	LE	DOV	2yr	0.47	+2.25DS	12	FIB	mod	+2.25DS	48	0	0	0	0	12	14	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
3	380378	60	F	RE	DOV,GLARE	11mon	0.47	-0.50DS	14	EP	mod	-0.5DS,-0.25DC@170	60	0.176	0.176	0.176	0.176	14	16	14	14	14	-	-	+	+	-	-	-	-	-	-	-	-	-
4	380900	58	M	LE	GLARE	4yr	0.30	+2.50	16	EP+FIB	mild	+0.25DS.	44	0	0	0	0	16	18	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
5	381392	65	M	LE	GLARE,DOV	1.5yr	0.6	+1.50	12	EP	mod	+1.75DS	72	0	0	0	0	12	18	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
6	388885	45	M	LE	GLARE	1yr	0.77	-1.25DS	14	EP	sev	+2.25DS	88	0.6	0.6	0.6	0.3	16	16	16	16	16	-	-	-	+	+	+	-	-	-	-	-	-	-
7	392869	52	M	LE	DOV	2yr	0.47	+2.5DS	18	EP	sev	"+0.25DS _0.50 @170	48	0.176	0.176	0.176	0.176	20	24	18	18	18	-	-	-	-	-	-	-	-	-	-	-	-	-
8	392864	65	F	RE	DOV	2yr	0.30	-0.50DS	18	EP	mild	+1.75DS,-1.50DC @ 150	24	0	0	0	0	18	18	18	18	18	-	-	-	-	-	-	-	-	-	-	-	-	-
9	392857	65	F	RE	DOV,GLARE	1yr	0.77	+1.50DS	14	EP	mild	-0.50DC@160	38	0	0	0	0	16	20	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
10	394669	30	F	RE	GLARE	4yr	0.6	-0.75DS	10	EP	mod	+0.25DS, -1.75DC @170	48	0	0	0	0	10	10	10	10	10	-	-	-	-	-	-	-	-	-	-	-	-	-
11	394661	70	F	LE	DOV	9mon	0.47	+0.75DS	14	EP	sev	+0.25DS,-2.00DC @170	72	0	0	0	0	20	22	14	14	14	-	-	-	+	-	-	-	-	-	-	-	-	-
12	397440	60	F	RE	DOV,GLARE	1yr	0.30	-0.75DS	12	EP	mild	+0.25DS, -0.50DC @ 20	44	0	0	0	0	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
13	399957	88	M	RE	DOV	2yr	0.77	-0.25DS	16	EP	mod	+1.75DS	38	0	0	0	0	20	24	18	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
14	402501	67	M	LE	GLARE	1.5yr	0.77	+2.25DS	12	EP+FIB	mod	+2.75DS	36	0	0	0	0	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
15	402504	60	F	RE	GLARE	1.5yr	1.0		14	EP	sev	+1.0DS,-1.25DC@10	66	0.176	0.176	0.176	0.176	14	20	14	14	14	-	-	-	++	-	-	-	-	-	-	-	-	-
16	402520	51	F	RE	GLARE	9mon	0.77	+1.25DS-2.25DC@150	16	EP+FIB	sev	+1.75DS	72	0.176	0.176	0.176	0.176	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
17	405045	38	F	RE	GLARE,DOV	2yr	1.0	+0.50DS,-1.25DC@160	12	EP	sev	+0.25DS-0.5DC@20	48	0	0	0	0	18	24	14	12	12	-	-	-	+	-	-	-	-	-	-	-	-	-
18	405015	60	F	RE	GLARE	2yr	0.6	+2.75DS	14	EP	mod	+2.0DS	38	0.176	0.176 .	0.176.	0.176 .	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
19	407674	65	F	RE	GLARE,DOV	1yr	0.77	-0.50DS,-0.75DC@20	12	EP	mod	+0.25DS,-2.00DC@10	28	0.77	0.77	0.77	0.77	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	ARMD
20	410559	39	M	RE	DOV	1yr	0.47	+1.25DS	14	EP	mild	+2.00DS	32	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
21	410574	65	M	RE	GLARE	2yr	0.47	-2.25DC@180	14	EP+FIB	mild	-2.50DC	44	0	0	0	0	14	22	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
22	410291	65	F	LE	DOV	9mon	1.0	-2.25DC@170	16	FIB	sev	-2.50DC	34	0.3	0.30	0.30	0.30	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
23	413243	63	M	LE	GLARE,DOV	1.5yr	1.0	+1.50DS	14	EP	sev	+2.0DS,-1.25DC@10	30	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
24	418480	62	M	LE	GLARE	1.5yr	1.7	+0.50DS	12	EP	sev	+1.0DS,-1.25DC@10	32	0.77	0.77	0.77	0.77	12	12	12	12	12	-	-	-	++	+	+	+	-	-	-	-	-	-
25	418464	60	M	RE	DOV	5yr	0.6	+0.50DS	12	EP	mod	+1.00DS	42	0	0	0	0	14	24	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
26	426545	52	M	RE	DOV,GLARE	1.5yr	0.6	-2.5DC@150	16	EP	mod	-2.25DC@30	28	0	0	0	0	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
27	434579	61	F	LE	DOV,GLARE	4yr	0.77	-0.75DS,-0.25DC@150	12	EP	mod	-0.25DS,-0.75DC@20	46	0	0	0	0	18	22	14	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
28	434553	74	F	RE	DOV	2yr	1.7	+2.50DS,-1.25DC@170	16	FIB	sev	+2.75DC@30	38	0.77	0.77	0.77	0.77	16	22	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	ARMD
29	434600	49	M	RE	DOV,GLARE	2yr	0.47	-0.50DS,-0.75DC@20	16	FIB	mod	-0.25DS,-0.75DC@20	36	0	0	0	0	16	24	16	16	16	-	-	-	+	-	-	-	-	-	-	-	-	-
30	437283	35	M	LE	DOV	2yr	0.77	+1.5DS,-1.25DC@20	14	EP	sev	+2.00DS,-1.00DC@30	44	0.76	0.176	0.176	0.176	14	14	14	14	14	-	-	-	+	-	-	-	-	-	-	-	-	-
31	442700	60	M	RE	DOV	4yr	1.7	-2.50DC@10	16	EP	sev	-2.25DC@13	45	1	0	0	0	16	24	16	16	16	-	-	-	+	-	-	-	-	-	-	-	-	-
32	441726	55	F	LE	DOV	1yr	1.0		18	EP + FIB	sev		66	0.3	0.30	0.30	0.30	18	18	18	18	18	-	-	-	-	-	-	-	-	-	-	-	-	-
33	445307	38	M	RE	GLARE,DOV	8mon	0.6	-0.25DS,-0.50DC@10	16	FIB	mod	-0.5DS,-0.75DC@10	42	0.176	0.176	0.176	0.176	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
34	445837	65	M	LE	DOV	1.5yr	1.0	+0.50DS,-0.5DC@160	14	EP	sev	+0.25DS,-0.75DC@150	38	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
35	451728	63	F	RE	GLARE,DOV	2yr	0.77	+2.25DS	14	EP+FIB	sev	-2.00DS	42	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
36	454526	70	M	RE	GLARE,DOV	3yr	0.77	+1.50DS	12	FIB	sev	+1.00DS	56	0	0	0	0	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
37	461166	79	M	RE	GLARE	4yr	0.47	-0.50DS,-0.75DC@10	10	EP	mod	+0.25,-0.5DC@23	71	0	0	0	0	10	10	10	10	10	-	-	-	-	-	-	-	-	-	-	-	-	-
38	461193	73	M	RE	GLARE	6mon	0.47	+.1.50DS	12	EP	mild	-0.25DS,-0.5DC@20	52	0	0	0	0	24	28	16	12	12	-	-	-	+	-	-	-	-	-	-	-	-	-
39	464585	35	M	LE	DOV	5yr	0.6	-0.75DS,-0.25DC@30	14	EP	mod	-0.50DS,-0.75DC@170	38	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
40	468050	71	F	RE	GLARE,DOV	2yr	0.6	-2.25DC	18	FIB	mild	-2.00DC@20	25	0	0	0	0	18	26	18	18	18	-	-	-	+	-	-	-	-	-	-	-	-	-
41	471288	62	F	RE	GLARE,DOV	4yrs	0.6	+1.75DS,-1.25DC@23	14	EP	mild	-0.75DC@170	28	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
42	477469	65	F	LE	GLARE,DOV	8mon	0.77	2.50DC@13	12	EP	sev		62	0.3	0.3	0.3	0.3	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
43	483493	59	M	LE	DOV	2yrs	1.0	+0.25DS,-0.25DC@5	14	FIB	mod	-0.75DC@23	48	0.47	0.176	0.176	0.176	14	14	14	14	14	-	-	-	+	-	-	-	-	-	-	-	-	-
44	484021	62	M	LE	GLARE,DOV	4yrs	0.77	-0.50DS,-0.5DC@15	12	FIB	mod	-0.75DS	52	0.176	0.176	0.176	0.176	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
45	486824	63	F	RE	GLARE,DOV	10mon	1.0	+1.50DS,-0.5DC@30	14	EP	sev	+1.00,-1.25DC@20	48	0.47	0.176	0.176	0.176	20	26	16	14	14	-	-	-	+	-	-	-	-	-	-	-	-	-
46	490224	35	M	RE	GLARE,DOV	5yrs	0.6	-2.25DS,-3.25DC@23	12	EP + FIB	mod	2.75DS,-3.25DC@20	32	0.176	0.176	0.176	0.176	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
47	493363	60	F	LE	GLARE,DOV	6mon	0.77	-0.50DS,-0.75DC@10	14	EP	sev	-0.25DS,-0.75DC@13	29	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
48	499220	60	M	LE	GLARE,DOV	4yrs	0.47	+1.75DS,-1.25DC@170	12	EP	mild	-1.25DC@163	37	0	0	0	0	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
49	502196	65	F	LE	GLARE,DOV	3yrs	0.47	-3.25DC@170	14	EP	mod	-3.50DC@165	57	0	0	0	0	14	20	14	14	14	-	-	-	+	-	-	-	-	-	-	-	-	-
50	507932	57	F	RE	DOV	3yrs	1.0	+0.25DS,-0.75DC@163	16	EP+FIB	sev	+0.75DS	80	0.3	0.30	0.30	0.30	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
51	510378	57	F	LE	DOV	3mon	1.0	-0.50DS,-0.75DC@20	16	EP	mild	-0.75DS,-1.5DC@23	42	0	0	0	0	16	26	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
52	510365	62	M	RE	GLARE,DOV	5yrs	1.7		12	EP	sev		88	0	0	0	0	14	20	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
53	299818	51	M	LE	GLARE,DOV	10mon	0.6	+1.50DS,-1.25DC@20	14	FIB	sev	+1.75DS,-1.25DC@23	91	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
54	344468	61	M	LE	GLARE,DOV	1yr	0.6	-0.25DS,-1.5DC@10	16	EP	mod	-0.75DS,-1.5.0DC@13	55	0.176	0.176	0.176	0.176	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
55	378607	59	M	RE	DOV	1.5yr	0.77	+0.25DS	16	EP+FIB	mod	-0.75DS	43	0	0	0	0	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-
56	376788	48	M	LE	DOV	1yr	1.7		12	EP	sev		85	0.6	0.6	0.6	0.6	12	26	12	12	12	-	-	-	+	+	+	+	-	-	-	-	-	-
57	551766	61	F	RE	GLARE,DOV	2yrs	0.47	-0.75DS,-1.25DC@10	14	EP	mod	+0.25,-1.50DC@10	51	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
58	551798	48	M	RE	GLARE,DOV	9mon	0.77	+1.75DS	14	EP	mod	+2.00DS	62	0	0	0	0	14	20	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
59	346341	39	F	RE	DOV	5yrs	1.7		16	EP+FIB	sev		33	0.77	0.77	0.77	0.176	16	16	16	16	16	-	-	-	++	+	+	-	-	-	-	-	-	-
60	336030	60	M	LE	GLARE,DOV	4yrs	1.0	-0.50DS,-1.25DC@180	18	FIB	mild	-1.00DS,-1.50DC@173	29	0.176	0.176	0.176	0.176	18	18	18	18	18	-	-	+	-	-	-	-	-	-	-	-	-	-
61	268265	70	M	RE	GLARE,DOV	3yrs	0.6	+1.50DS,-1.5DC@10	20	EP	mod	-0.25DS,-1.75DC@13	61	0	0	0	0	20	20	20	20	20	-	-	-	-	-	-	-	-	-	-	-	-	-
62	369930	65	M	RE	GLARE,DOV	5yrs	0.47	-3.25DC@15	20	EP	mod	-3.00DC@10	32	0	0	0	0	20	20	20	20	20	-	-	-	-	-	-	-	-	-	-	-	-	-
63	555988	59	M	RE	DOV	4yrs	1.0	+1.50DS,-1.50DC@20	14	EP+FIB	mild	+1.00DS,-1.75DC@13	27	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-
64	556059	60	F	RE	GLARE,DOV	5mon	0.77	+1.50DS,-1.5DC@10	16	FIB	sev	+2.00DS,-1.75DC@15	69	0.776	0.776	0.176	0.176	16	18	16	16	16	-	-	-	++	+	-	-	-	-	-	-	-	-
65	327915	64	M	RE	GLARE,DOV	4yrs	0.77	-2.50DS	12	FIB	mod	+2.00DS	48	0.176	0.176	0.176	0.176	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-
66	533012	51	F	LE	GLARE,DOV	3yrs	0.47	+0.25DS	14	EP	sev		68	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-

67	366572	44	F	RE	GLARE,DOV	2yrs	0.47	-0.50DS	16	FIB	sev	+0.25DS,+1.25DC@23	67	0	0	0	0	22	26	16	16	16	-	-	-	+	-	-	-	-	-	-	-	-	-	-	
68	354+N4:N90	45	F	LE	GLARE,DOV	15mon	0.6	-2.50DS,-1.5DC@10	18	EP	sev	-2.50DS,-1.00DC@20	58	0	0	0	0	18	18	18	18	18	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
69	566457	52	M	RE	GLARE,DOV	1yr	1.7	+1.50DS,-1.5DC@10	12	FIB	sev	+1.00DS,-2.00DC@20	68	0	0	0	0	12	20	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
70	362171	62	M	LE	DOV	16mon	1.7		18	FIB	mild		82	0	0	0	0	18	18	18	18	18	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
71	582955	60	F	RE	GLARE,DOV	1yr	1.0	+0.50DS,-1.50DC@10	20	FIB	sev	+0.75DS,-1.75DC@20	65	0.3	0.30	0.30	0.30	20	20	20	20	20	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
72	589180	52	F	RE	GLARE,DOV	3mon	0.47	-0.25DS,-1.25DC@30	18	EP	mod	+0.25DS	40	0	0	0	0	18	18	18	18	18	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
73	342272	48	F	RE	GLARE,DOV	15mon	0.30	+0.50DS	14	EP	mod	+1.25DS	33	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
74	427383	50	F	LE	GLARE,DOV	15mon	1.0	+1.75DS	16	FIB	sev	+1.50DS	39	0.3	0.30	0.30	0.30	22	28	18	16	16	-	-	-	++	-	-	-	-	-	-	-	-	-	-	
75	572721	45	M	RE	GLARE,DOV	1.5yr	0.77	-2.25DS	12	FIB	mod	-2.25DS	49	0	0	0	0	12	12	12	12	12	-	-	+	-	-	-	-	-	-	-	-	-	-	-	
76	455173	15	M	RE	GLARE,DOV	10mon	1.0	-0.25DS	10	FIB	mod		59	0	0	0	0	10	10	10	10	10	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
77	455165	18	M	LE	DOV	1.5yr	1.0	+0.50DS	18	FIB	sev	+1.25DS	62	0.77	0.77	0.77	0.176	18	26	18	18	18	-	-	-	++	+	+	-	-	-	-	-	-	-	-	
78	602387	52	M	RE	GLARE,DOV	6yrs	1.0		14	EP	sev		39	0.3	0.30	0.30	0.30	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
79	613305	81	F	LE	DOV	6yrs	1.0	-0.25DS,-1.25DC@30	18	EP	mod	+0.5DS,-1.75DC@20	76	0.176	0.176	0.176	0.176	18	18	18	18	18	-	-	+	-	-	-	-	-	-	-	-	-	-	-	
80	623482	65	F	RE	GLARE,DOV	2.5yrs	0.47	+1.25DS,-1.25DC@13	12	EP	mild	+2.00DS,-1.5DC@10	61	0	0	0	0	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
81	624320	44	F	LE	GLARE,DOV	1yr	0.6		14	EP	mod		58	0	0	0	0	14	20	14	14	14	-	-	-	+	-	-	-	-	-	-	-	-	-	-	
82	628227	71	M	RE	DOV	9mon	1.0	+0.50DS,-1.25D@23	16	FIB	mod	+1.25DS,-2.00DC@20	55	0.3	0.30	0.30	0.30	16	16	16	16	16	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
83	385493	63	M	LE	GLARE,DOV	10mon	0.30	-2.50DS	12	EP	mild	-2.25DS	58	0	0	0	0	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
84	644795	45	M	RE	GLARE,DOV	1yr	0.47	+1.25DS,-1.25DC@170	14	FIB	mild	+1.75DS,-1.75DC@160	62	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
85	398550	67	F	RE	DOV	1.5yr	0.77		12	EP	mild		43	0	0	0	0	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
86	633482	65	F	RE	GLARE,DOV	1yr	0.77	-0.25DS,-1.25DC@10	14	FIB	mild	+0.50,-1.50DC@20	38	0	0	0	0	14	14	14	14	14	-	-	+	-	-	-	-	-	-	-	-	-	-	-	
87	644208	50	F	LE	DOV	1.5yr	1.0	+1.50DS,-1.25DC@12	12	EP	sev	+2.00DS,-1.75DC@15	49	0.3	0.30	0.30	0.30	12	12	12	12	12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
88	366837	49	F	RE	GLARE,DOV	2yr	0.3	-1.0DS,-1.50DC@30	14	EP	mild	-0.5DS,-1.25DC@20	52	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
89	336030	60	M	RE	GLARE,DOV	9mon	0.6	+0.25DS,-1.5DC@10	14	FIB	mild	+0.75DS,-2.00DC@15	61	0	0	0	0	14	14	14	14	14	-	-	-	-	-	-	-	-	-	-	-	-	-	-	
90																																				