POST OPERATIVE COMPLICATIONS IN HOSPITAL BASED CAMP PATIENTS UNDERGOING CATARACT SURGERY .

By

Dr. SUSHMA.A.HOSAMANI

Dissertation submitted to the

B.L.D.E. UNIVERSITY BIJAPUR, KARNATAKA

In partial fulfillment of the requirements for the degree of

MASTERS OF SURGERY

In

OPHTHALMOLOGY

Under the guidance of

Dr. VALLABHA.K M.S,DOMS.

PROFESSOR AND HEAD

DEPARTMENT OF OPHTHALMOLOGY

B.L.D.E.U'S SHRI B. M. PATIL MEDICAL COLLEGE

HOSPITAL & RESEARCH CENTRE, BIJAPUR

KARNATAKA

2014

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, BIJAPUR

DECLARATION BY THE CANDIDATE

I, **Dr. SUSHMA.A.HOSAMANI.,** hereby declare that this dissertation entitled **"POST OPERATIVE COMPLICATIONS IN HOSPITAL BASED CAMP PATIENTS UNDERGOING CATARACT SURGERY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.VALLABHA.K**., Professor & head, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date:

Place: Bijapur

Dr. SUSHMA.A.HOSAMANI.

Post Graduate Student,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College,

Hospital & Research Centre, Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, BIJAPUR

CERTIFICATE BY THE GUIDE

This to certify that the dissertation entitled **"POST OPERATIVE COMPLICATIONS IN HOSPITAL BASED CAMP PATIENTS UNDERGOING CATARACT SURGERY"** is a bonafide research work done by **Dr. SUSHMA.A.HOSAMANI.**, under my overall supervision and guidance, in partial fulfillment of the requirements for the degree of M. S. in Ophthalmology.

Date:

Place: Bijapur

Dr. VALLABHA.K **M.S,DOMS.**

Professor & Head,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College,

Hospital & Research Centre, Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, BIJAPUR

ENDORSEMENT BY THE HEAD OF DEPARTMENT

This to certify that the dissertation entitled **"POST OPERATIVE COMPLICATIONS IN HOSPITAL BASED CAMP PATIENTS UNDERGOING CATARACT SURGERY"** is a bonafide research work done by **Dr. SUSHMA.A.HOSAMANI.** under the guidance of **Dr. VALLABHA.K** M.S,DOMS., Professor & Head, Department of Ophthalmology at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date:

Place: Bijapur

DR. VALLABHA.K **M.S,DOMS.**
Professor and Head,
Department of Ophthalmology,
B.L.D.E.U's Shri B. M. Patil Medical College,
Hospital & Research Centre, Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, BIJAPUR

ENDORSEMENT BY THE PRINCIPAL

This to certify that the dissertation entitled **"POST OPERATIVE COMPLICATIONS IN HOSPITAL BASED CAMP PATIENTS UNDERGOING CATARACT SURGERY"** is a bonafide research work done by **Dr.SUSHMA.A.HOSAMANI.** under the guidance of **Dr. VALLABHA.K** M.S,DOMS. Professor & Head, Department of Ophthalmology at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date**:**

Place: Bijapur.

DR. M. S. BIRADAR **M.D.**
Principal,
B.L.D.E.U's Shri B. M. Patil Medical College,
Hospital & Research Centre, Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, BIJAPUR

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the B.L.D.E. UNIVERSITY, BIJAPUR, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purposes.

Date:

Place: Bijapur

Dr. SUSHMA.A.HOSAMANI.

Post Graduate Student,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College,

Hospital & Research Centre, Bijapur.

ACKNOWLEDGEMENT

This piece of work has been accomplished with the grace of almighty God. It gives me immense pleasure to express my heartfelt gratitude to all. I dedicate this page to each and everyone who have helped me to explore the expanses of knowledge.

I express my profound gratitude and sincere thanks to my guide, ***Dr. Vallabha.K*** ***M.S, DOMS*** *., Professor & Head, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, for his constant and unfailing support, professional insight, valuable suggestions, motivation and exemplary guidance to carry out and complete this dissertation. I am deeply grateful to him for providing me necessary facilities and excellent supervision to complete this work.*

I offer my sincere thanks to ***Dr. M. S. Biradar*** ***M.D.,*** *Principal, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, for his support and inspiration.*

My thanks to our Medical Superintentend ***Dr. Shivakumar.Hiremath*** *for this support and inspiration.*

I am deeply indebted and grateful to my professors, ***Dr. M. H. Patil*** ***M.S.,*** *and* ***Dr. Sunil. G. Biradar*** ***M.S.,*** *Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, who with their valuable suggestions and constant guidance supported me throughout the preparation of this dissertation work.*

My thanks to ***Dr. Shadakshari.S.Math*** ***M.S.,*** *Associate Professor and* ***Dr. Raghavendra.Ijeri*** ***M.S.,*** *Assistant professor, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, for their valuable suggestions and encouragement which have definitely helped me improve my research work.*

I sincerely acknowledge, ***Dr. Ravi.Jadhav, Dr. Cauvery, Dr. Renuka. Kattimani, Dr.Varunkumar****, for their help and encouragement which has definitely helped to improve my research work.*

I acknowledge my gratitude to ***Dr. Akshatha and Dr.Radhika,*** *Postgraduate colleagues, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, for his support, advice and help in data collection.*

I also thank all my Post-graduate junior colleagues ***Dr.Darshan, Dr.Shravan, , Dr.Shilpa*** *and friends in the department for their co-operation during the preparation of this dissertation.*

I thank ***Mrs. Vijaya Soraganvi****, Statistician for her masterly guidance and statistical analysis. I sincerely acknowledge the support and kindness shown towards me by all the staff of Central Library, Shri B. M. Patil Medical College, Bijapur, at all times.*

I am thankful to all the Technical and non-teaching Staff of the Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur for their co-operation.

It is with great pleasure I express my sincere gratitude to my parents for their constant encouragement, inspiration and sacrifices. My heartly thanks to my family, my husband ***Dr.Vijaykumar.G.Warad*** *, my daughter* ***Anushree*** *and my son* ***Anoop*** *for their encouragement, support and sacrifies .*

I am grateful to Preeti internet cafe, Bijapur for their timely and fantastic printing work.

Last but not the least, my sincere thanks to all the participants of this study for their cooperation without which this study would not have been possible.

Date- *Dr.Sushma.A.Hosamani*

Place :

LIST OF ABBREVIATIONS:-

AC.....	Anterior chamber
ACIOL……	Anterior chamber intraocular lens
ARMD.....	Age related macular degeneration
ATP.....	Adenosine triphosphate
ATR……	Against the rule
BCVA	Best corrected visual acuity
BK.....	Bullous keratopathy
CCC.....	Continuous curvilinear capsulorrhexis
CE	Corneal edema
CF.....	Counting finger
CME.....	Cystoid macular edema
CTF……	Counting fingers close to face
D…….	Diopters
DMD….....	Descemet detachment
DPN, DPNH.....	Diphosphopyridine
ECCE	Extracapsular cataract extraction
Endo	endophthalmitis
EVS.....	Endophthalmitis vitrectomy study
HMP.....	Hexose monophosphate pathway
HM…….	Hand movements
HMC……	Hypermature cataract
Hyp.....	Hyphema
ICCE	Intracapsular cataract extraction
ID……	Iridodialysis
IO	Intraoperative
IOL.....	Intraocular lens
IOP	Intraocular pressure
IO compln……	Intraoperative complications
Inj……..	Injection

Ir In.....	Iris incarceration
Ir.....	Iritis
IrP.....	Iris prolapse
Kg.....	Kilogram
LE.....	Left eye
LEC.....	Lens epithelial cell
LM.....	Lens matter
LS.....	Loose sutures
mEq.....	Milli equivalent
mg	Milligram
mM.....	Millimoles
M……	Meter
NA……	No astigmatism
Nd-YAG.....	Neodymium: Yttrium aluminum garnet
NS……..	Nuclear sclerosis
OC……..	Optic capture
PCIOL.....	Posterior chamber intraocular lens
PE……..	Premature entry
PPCO…….	Pre exsisting posterior capsular opacificatiom
PCO.....	Posterior capsule opacification
PCR.....	Posterior capsule rupture
PL.....	Perception of light
PMMA	Polymethyl methoacrylate
PO	Postoperative
Pr Uv	Persistent uveitis
PR.....	Projection of rays
PSC……...	Posterior subcapsular cataract
PXF	Pseudoexfoliation
RE	Right eye
RD……	Retinal detachment

SAC.....	Shallow anterior chamber
SCI	Sealed capsular irrigation
SG……..	Secondary glaucoma
SHMC.....	Senile hypermature cataract
SICS	Small incision cataract surgery
SIMC.....	Senile immature cataract
SK	Striate keratitis
SMC.....	Senile mature cataract
TPN, TPH	Triphosphopyridine
Tab……..	Tablet
U/ml	Units/ milliliter
UCVA.....	Uncorrected visual acuity
UGH.....	Uveitis glaucoma hyphema
VL	Vitreous loss
WTR……	With the rule
WG.....	Wound gape

LIST OF CONTENTS:-

LIST OF TABLES:-

LIST OF GRAPHS:-

ABSTRACT

Background : Cataract is a significant and increasing global problem with vast economic and social implications. It is the principal cause of blindness in India accounting for 62.6% . The principal solution to the backlog of cataract blind is performing cataract operations on a large scale. This may be carried out by cataract camps, comprehensive eye care camps and base hospital approach with screening camps.

Aim and objevtives : To study the profile of post operative complications in hospital based camp patients undergoing cataract surgery and to suggest the measures to overcome complications.

Materials and methods: Cataract surgery was carried out in 313 patients after screening at community based screening camps. All patients underwent manual small incision cataract extraction with posterior chamber intraocular lens implantation under local anesthesia after evaluation.

Results: Intraoperative complications occurred in 25 patients (7.99%). Posterior capsule rupture without vitreous loss (3.20%) was common followed by vitreous leak(1.60%). Others included Descemet's membrane detachment (0.96%), premature entry (0.95%) , iridodialysis (0.64%), and iris trauma(0.64%).

The most frequently occurring complication during the immediate postoperative period in decreasing order were corneal edema(12.46%),striate keratitis (8.95%), persistent uveitis(7.04%), secondary glaucoma(3.52%), followed by residual lens matter (3.52%), shallow anterior chamber (0.31%), hyphema (0.31%), iris incarceration (0.31%) and iris prolapse (0.64%).

Endophthalmitis(sterile) was seen in 0.31%. Postoperative complications at the end of first week include persistent uveitis (3.84%), corneal edema (3.20%), striate keratitis(1.93%) ,secondary glaucoma(1.60%) and one case each (0.31%) of displaced IOL, residual cortex & optic capture. The most common late postoperative complication was posterior capsule opacification (2.88%), cystoid macular edema(0.31%) and optic capture(0.31%) at the end of 6^{th} week. Posterior capsular opacification was present in 2.55% at the end of 3^{rd} month and 2 cases (0.64%) of retinal detachment at the end of 6^{th} month were noted.

Conclusion: The overall complications of cataract extraction in base camps were very low. Well organized cataract screening camps combined with efficient base hospital surgery, which provides early and good visual outcome can be a strategy to reduce the backlog of cataract blindness in rural communities in developing countries.

Key Words: Small incision cataract surgery, Base camps , post operative complications, vision 2020 .

INTRODUCTION:

Any opacity in the lens or its capsule ,whether developmental or acquired, is called cataract. The disease is caused by degeneration and opacification of the lens fibres already formed, the formation of aberrant lens fibres or deposition of other material in their place . As a general rule, developmental opacities are partial and stationary, acquired opacities progress until the entire lens is involved ; exceptions are well known in both the types [1].

Cataract has been documented to be the most significant cause of bilateral blindness in India where vision < 20/200 in the better eye on presentation is defined as blindness. In India cataract has been reported to be responsible for 50-80% of the bilaterally blind in the country. The most recent estimates from WHO reveal that 47.8% of global blindness is due to cataract and in South Asia region which includes India, 51% of blindness is due to cataract[2] . Cataract is a significant and increasing global problem with vast economic and social implications[3]. It is the principal cause of blindness in India accounting for 62.6% [4].

The prevalence of blinding cataract will only increase as people live longer, so cataract will continue to be, by far, the most important treatable cause of blindness. It is estimated that the present number of 20 million of cataract blind will double by the year 2020 [5]. In addition to the backlog, an additional 3.8 million become blind each year because of cataract[6].

An estimated 4 million people become blind because of cataract every year, which is added to a backlog of 10 million operable cataracts in India, whereas only 5

million cataract surgeries are performed annually in the country[7]. In 2000, 3.5 million cataract operations were performed, but this remains insufficient to treat the backlog and the newly blind[8].

The answer to the backlog of cataract blindness in India has many possible solutions that may differ in various locations. The principal solution to the backlog of cataract blind is performing cataract operations on a large scale. This may be carried out by cataract camps, comprehensive eye care camps and base hospital approach with screening camps. More effort needs to be directed to improve the quality of cataract surgery, through improvement in areas such as case selection and postoperative care, rather than just concentrating on surgical technique and volume. It is only through an integrated approach that the challenge of creating widespread areas to surgical services capable of delivering good quality visual rehabilitation will be met. The effective work of Aravind Hospital System is the testimony of this[2].

Efforts to tackle cataract blindness in India have been going on in earnest for the last three decades. The revolutionary idea of holding surgical eye camps at base hospitals started in the late 1960s and was extremely popular until Aravind Eye Hospital changed this strategy by conducting screening camps and performing surgery at the base hospital[9].

Base hospital approach is an important alternative to peripheral eye camps, which envisages screening of patients in screening camps, their transfer to the base hospital and subsequently surgery in the base hospital using permanent infrastructure

already available. The advantages of base hospital approach stem from the utilization of optimal management for pre-operative, operative and post-operative care for the patient[10].

Due to continued improvement in surgical technique, lens design and manufacturing, the incidence of complications of cataract surgery with intraocular lens implantation have decreased in recent years. Operative complications include posterior capsule rupture with or without vitreous loss, loss of all or part of the lens nucleus, iris damage and stripping of Descement's membrane etc.

Early postoperative complications include pupillary block, hyphema, elevation of the intraocular pressure associated with the use of viscoelastic, persistent uveitis with or without hypopyon and endophthalmitis. Late postoperative complications include IOL malposition, secondary glaucoma, PCO, cystoid macular edema, retinal detachment, and pseudophakic bullous keratopathy[11].

Postoperative complications though inevitable, adequate preventive measures such as timely diagnosis and appropriate management of complications can decrease ocular morbidity. The frequency of postoperative examinations is based on the goal of optimizing the outcome of surgery and swiftly recognizing and managing complications.The study has been undertaken to study the complications after cataract extraction in eye camps at base hospital.

AIM AND OBJECTIVE OF THE STUDY:

1. To study the profile of post operative complications in hospital based camp patients undergoing cataract surgery in BLDE University Shri.B.M.Patil Medical College Bijapur.
2. To suggest the measures to overcome complications.

REVIEW OF LITERATURE

Blindness was present in prehistoric times also. Since the inception of mankind, even the sages were treated by different surgeons for blindness. In those times, Ashwani Kumar and Sushruta used to move from Ashram to Ashram providing treatment for eye diseases and blindness, which we now emulate as "Modern Eye Camp Surgery"[10]

Cataract extraction has undergone a tremendous metamorphosis from the procedure of couching practiced by Sushruta (1000 BC) in India to the latest method of phacoemulsification devised by Charles Kelman (1967).

Couching was practiced by Sushruta in 1000 BC followed by Greeks, Romans and Arabs. In 280 BC Herophilus and Philoxenes practiced cataract surgery in Alexandrian school. A firm foundation for cataract surgery was established in the West when Fabricius of Aguapendents described the anatomic position of the lens in 1600. Stephen Blaukaart in 1668 removed a cataract through a corneal incision[13].

Age of Enlightenment:

Eighteenth century is called the age of enlightenment. It was Micheal Pierre Brisseau in 1705 who first attributed cataract formation to the lens and not a coagulated humor in front of it. An opaque lens was delivered after its accidental anterior dislocation during couching by Charles Saint-Yves in 1707.

In 1736, Benedict Duddell extracted soft cataract by inserting a lancet through the capsule. The first, to make a planned cataract extraction was Jacques Daviel in 1748. George de la Faye (1752) devised a knife to make corneal incision and a cystitome to

incise the lens capsule. Pierre-Francois-Benezet Pamard between 1759 and 1784 advised that the patient lie on his back instead of being seated, used an instrument to fixate the eye and made an incision in the upper part of the cornea.

Wenzel (1786) made an incision in the upper part of the cornea with a keratome instead of a knife. Carl Himly in 1801 introduced mydriasis before the operation, while a preliminary iridectomy was practised in 1862 by Albert Mooren. Julius Jacobson in 1863 practiced a limbal incision. In 1866, Albrecht von Graefe introduced combined "linear extraction". The idea of peripheral iridectomy was advanced by Bajardi (1895) and advocated by Elschnig (1912).

Intracapsular extraction was first planned by Samuel Sharp (1753) who expelled the lens by the pressure of his thumb. Pagenstecher (1866-71) removed lens with a spoon or a loop (vectis). All these procedures involving pressure on the globe formed the basis of extraction of the lens by traction. The first was done by Terson (1870) who grasped the capsule with toothed forceps, Landesberg (1878) used iris forceps, and Eugene Kalt devised a smooth forceps. Stanculeanu (1912), Knapp (1914), Anton (1922), Sinclair (1925) developed the techniques of intracapsular operation using various patterns of capsule forceps. Stoewer (1902) and Hulen (1910) devised suction cup, Barraquer (1924) designed an erisophake, Lacarrere (1932) used diathermo-coagulation and in 1961 Krwawicz employed a cryosurgical probe[13].

Zonular destruction was first done mechanically by LiLuca (1866) α-chymotrypsin was introduced by Barraquer (1958).

Henry Williams introduced corneo-scleral sutures in 1867 and conjunctival sutures in 1869 followed by Kalt (1894), Verhoeff (1916) and Suarez de Mendosa(1891).

Vanlint (1917), Wright (1926) and O'Brien (1929) brought improvement in regional anesthesia and akinesia.

In 1928 Elschnig suggested retrobulbar injection and superior rectus bridle suture. Dunmington (1951) advocated ab-externo incision and limbus based conjunctival flap and practised removal of non-absorbable sutures on the 7th post-operative day. Harold Ridley first implanted intraocular lens into the posterior chamber in 1949.

Richter (1791) noticed vitreous loss, Prof.Barth (1745-1818) observed that the inflammation subsequent to the operation is always much greater in those patients with vitreous loss.

1892 - Collins and Cross emphasized epithelial implantation in the anterior chamber after cataract extraction.

1928 - Descemets detachment was described by Samuel. 1934 - Shapland DC described retinal detachment in aphakia. 1953 Irvine SR defined vitreous touch syndrome following cataract surgery.

1952 - Post-operative macular edema was described by Irvine and aphakic pupillary block glaucoma was first described by Bowman.

1974 – Early ocular hypertension after cataract extraction was described by WJ Rich and co-workers.

1982 – Samuel M Salaman and Bartly J Mondino described peripheral corneal ulcers and scleritis after cataract surgery.

1987 – Regan J Bradfor and CP Wilkinson showed vitreous opacification after cataract extraction.

Literature Survey:

Das T, Venkataswamy G in a study in 1982 compared the surgical results of cataract surgery of patients operated in eye camps and in hospital. Analysis showed that full visual recovery was 46% in hospitals as compared to 19% in camps. Overall operative and postoperative complications occurred more in patients operated in eye camps[14].

Balmer A, Andenmatten R, Hiroz CA in a retrospective study on 1304 patients who underwent cataract extraction between 1982 and 1987 studied the complications of cataract surgery. Loss of vitreous in 2.5%, cystoid macular edema in 1.9%, retinal detachment in 1.1% were the complications occurring frequently. Posterior capsule opacification was noted in 31% of cases[15].

Chitkara DK, Smerdon DL conducted a retrospective study of 1552 patients who underwent cataract extraction between 1987 and 1991. Posterior capsule rupture with vitreous loss (PCR + VL) was seen in 4%.Pseudo-exfoliation diabetes mellitus and a traumatic etiology increased the risk of PCR+VL. Postoperative complications were iris prolapse (1.3%), retinal detachment (0.3%), endophthalmitis (0.2%), pseudophakic bullous keratopathy (0.1%). 21% had symptomatic loose or broken sutures and 11.2% had significant posterior capsule opacification[16].

Goodman DF, Stark WJ, Gottsch JD in the year 1989 studied the complications of cataract extraction with intraocular lens implantation. Complications were categorized into operative and postoperative, both early and late onset. The incidence of complications of cataract surgery with IOL implantation have decreased in recent years

due to continued improvement in lens design, lens manufacturing and surgical technique[11].

Sudhakar J, Ravindran RD, Natchiar G in 1989 analyzed the complications in 1000 cases of posterior chamber intraocular lens implantation. The most common early postoperative complication was striate keratitis followed by iritis and corneal edema. Posterior capsule opacification was the main late postoperative complication observed in 11.5%. This complication can be easily managed by YAG laser capsulotomy. Visual acuity of 6/12 or better was achieved in 80.7%[17].

Lumme P, Laatikainen LJ in 1990 evaluated the risk factors for intraoperative and early postoperative complications on 351 patients. Bleeding into the anterior chamber during the operation, zonular rupture, posterior capsular rupture and vitreous loss were the operative complications noted. Postoperative complications included corneal edema, rise of intraocular pressure and fibrous reaction. Small pupil and exfoliation syndromes were the most important risk factors for both intra and early postoperative complications. Presence of glaucoma increased the risk of vitreous loss, postoperative pressure rise and corneal edema[18].

Lumme P, Laatikainen LT in 1990 studied the factors affecting the visual acuity three months after cataract surgery in 243 patients operated at university hospital. The visual outcome was significantly related to age. The most common causes for low vision were age related macular degeneration and glaucoma. Visually significant posterior capsular opacification was observed in 2.5% and significant postoperative astigmatism ($\geq$3.5D)[19].

Civerchia L, Apoorvananda SW, Natchiar G, Balent A et al in 1991 performed cataract surgery on 379 patients in a public eye camp. Causes of poor visual outcome were retinal detachment, optic atrophy, macular scarring and chorioretinitis. Serious difficulties in peripheral eye camps were associated with measuring initial intraocular lens power, obtaining refractive data, follow up of astigmatism, posterior capsule opacification and associated preoperative pathology[20].

Natchiar G, Robin AL, Thulasiraj RD, Krishnaswamy S (1994) in their study titled Attacking the Backlog of India's Curable Blind described a system of high quality, high-volume, cost-effective cataract surgery using screening eye camps and a resident hospital. The advantages of base hospital approach were described, which include availability of multispeciality clinics, highest quality surgery and costeffectiveness[8].

Madhukar K Reddy (1995) in a study discussed the diagnosis, treatment and prevention of intraoperative and postoperative complications of cataract surgery. Early recognition and prompt treatment of these problems can minimize the risk of ocular morbidity[21].

Verma L, Gupta SK, Murthi GVS, Goyal M, Pant TD in a study conducted on 145 patients in 1996 assessed the visual outcome after camp based intraocular extraction. Less than 50% of the patients had a visual outcome of 6/18 or more. The most common cause of decreased visual acuity was corneal edema followed by vitreous haze, glaucomatous optic atrophy, vitreous hemorrhage and endophthalmitis[22].

Gogate PM, Kulkarni AN in 1996 compared the results of cataract surgery in a base hospital and in peripheral eye camps. Base hospital surgery resulted in better and

earlier visual rehabilitation in 82.7% as compared to 43.7% in peripheral camps. The chief cause of low postoperative vision was vitreous loss.

The surgery in peripheral eye camps was marginally more economical as compared to the base hospital. Considering the quality of surgery, early and better visual rehabilitation, the base hospital approach has much to recommend it[23].

Dandona L, Dandona R, Naduvilath TJ, McCarty CA, Mandal P, Srinivas Met al in 1997 conducted a study to assess the outcome of cataract surgery. Posterior segment causes which included optic atrophy, endophthalmitis, retinal detachment were the main causes of decreased visual acuity. Refractive error was the most common anterior segment cause of decreased visual outcome[24].

Anand R, Gupta A, Ram J, Singh U, Kumar R in 1997 assessed the visual outcome following cataract surgery in rural camps in 300 patients. 17% of cataract operated eyes were blind. 68% of blindness was related to the cataract surgery complications. Corneal edema was the most common followed by retinal detachment and aphakic glaucoma. Among the non-cataract surgery related complications, the most common was traumatic corneal opacity followed by glaucoma, band-shaped keratopathy and climatic droplet keratopathy[25].

Limburg H, Foster A, Vaidyanathan K, Murthy GVS in 1998 compared the visual outcome in camp and hospital operated cataract patients. They have found poor visual outcome (visual acuity <6/60) almost twice as common in camp operated patients compared to the hospital operated patients[26].

Thulasiraj RD, Reddy A, Selvaraj S, Munoz SR, Ellivein LB in 1999 assessed the clinical outcomes of cataract surgery in rural Southern India. Uncorrected aphakia and

other refractive error were the main causes of vision impairment. Poor presenting visual acuity in aphakic eyes was associated with illiteracy, rural residence and surgery in government facilities[27].

Kothari M, Thomas R, Parikh R, Braganza A, Kuriakose T, Muligil J in 1999 reviewed 2095 patients to assess the incidence and visual outcome of vitreous loss for different cataract surgical techniques in a tertiary teaching hospital. Vitreous loss was most common with ECCE followed by the Blumenthal technique and phacoemulsification. Incidence and visual outcome of vitreous loss managed using standard vitrectomy techniques were assessed for different cataract surgical techniques as well as at different levels of surgical training. This complication, managed with standard surgical techniques, is compatible with good visual outcome[28].

Hennig A, Kumar J, Singh AK, Ansari A, Singh S, Gurang R, Foster A in 2002 conducted a cataract surgical workshop during which 2167 cataract extractions were done at Lahan Eye Hospital. Surgical complications was seen in 1.2%. They concluded that well organized cataract screening camps combined with efficient base hospital surgery, which provides early and good visual outcome can be a strategy to reduce the backlog of cataract blindness in rural communities in developing countries[5].

Hennig A, Yorston D, Kumar J, Foster A in 2001 reviewed the outcome of sutureless manual extracapsular cataract extraction (ECCE) at a high volume surgical centre. The major cause of uncorrected visual acuity less than 6/18 was postoperative astigmatism. Intraoperative complications include hyphema, posterior capsule rupture. Postoperatively hyphema accounted for the major complication in 19 eyes. They

concluded that rapid recovery of good vision can be achieved with sutureless manual ECCE at low cost in areas where there is a need for high volume cataract surgery[29].

ANATOMY AND DEVELOPMENT OF LENS

Embryology:

The rudimentary lens is first seen as a thickening of the surface ectoderm, the lens placode at 22 days gestation; it overlies the optic vesicle. The lens placode forms the lens vesicle which consists of a single layer of cells. The cells forming the posterior wall of the lens rapidly elongate and become filled with proteins called crystallins. These densely packed elongated cells are known as the primary lens fibers. Additional fibers are formed by the mitotic division of the anterior epithelial cells at the equator known as secondary lens fibers. New secondary lens fibers are formed throughout life and persist throughout life. The ends of the fibers come into apposition at sites referred to as sutures. In the fetus, the lens grows rapidly, because it is supplied by the hyaloid artery, which forms a plexus on the posterior surface of the lens. The vascular lens capsule is formed from the mesenchyme. The true lens capsule is formed from the thickened basal lamina[30].

Anatomy:

The lens is a transparent, biconvex, elliptical, semisolid, avascular body of crystalline appearance located between the iris and the vitreous[31]. In the adult, the lens measures approximately 10 mm in diameter and 4mm thick. The lens contributes to about 15 diopters to the total power of the eye. The lens is made up of three parts (1) an elastic capsule; (2) a lens epithelium; and (3) the lens fibers.

Capsule: The capsule of the lens is an elastic basement membrane that envelops the entire lens. It is thickest on the anterior and posterior surface close to the equator and thinnest at the posterior pole.

Epithelium: The lens epithelium consists of a single sheet of cuboidal cells spread over the front of the lens, deep to the capsule and extending outwards to the equator.

Lens Fibers: The lens fibers constitute the main mass of the lens. The fibers are formed by the multiplication and differentiation of the lens epithelial cells at the equator. The earliest fiber mass in the center of the lens is known as the embryonic nucleus. This is followed by the fetal nucleus with its Y-shaped sutures. Those fibers that are formed after birth constitute the earliest part of the fiber mass known as the adult nucleus. The area surrounding the adult nucleus, containing the recently formed nucleated fibers is referred to as the lens cortex.

During the development, the lens fibers lose their nuclei and the cytoplasmic organelles become specialized for the production of lens proteins, known as crystallins. The lens is held in position by a series of delicate radially arranged fibers, collectively known as the suspensory ligament of the lens or zonules. The zonules fibers arise from the epithelium of the ciliary process and run towards the equator of the lens.

Physiology and Biochemistry of Lens:

The molecular makeup of the lens is unique in that it is two-thirds water with one-third protein; other constituents represent only about 1% of total lens weight[32].

Composition of the Lens:

Compared to most other tissues, the lens has a particularly high protein content and a low water content. The high protein concentration is necessary for the lens to maintain a high refractive index. The adult human lens is approximately 65% of water. The lens capsule is about 80% water, and the water content of the dense nuclear region of the lens is less than that of the outer cortex.

Lens Proteins:

Protein accounts for about 35% of the net weight of the lens. Based upon their solubility in water, they are separated into two classes.

1. Water soluble lens crystallins – account for nearly 90% of the total lens proteins.
2. Water insoluble proteins – consists of membrane proteins, cytoskeletal proteins and aggregated crystallins.

Lens Crystallins: are a heterogeneous group of structural proteins identified as:

1. Alpha crystallin: Accounts for 35% of total lens protein.
2. Beta crystallin: are the most abundant water-soluble protein, representing about 55%.
3. Gamma crystallin: accounts for 1 to 2% of the total.

Water insoluble proteins: include

1. Membrane proteins accounting for approximately 20 to 30% of water insoluble fraction of lens proteins.
2. Cytoskeletal proteins

Lens lipids include cholesterol, phospholipids and glycosphingo-lipids. About 50 to 60% of the lens lipid is cholesterol. The major phospholipid is sphingomyelin.

Sodium: is in the range of 14 to 26 mEq/Kg lens water and potassium is about 140 mEq/Kg lens water. There is low sodium level and high potassium level in the normal lens[32].

Calcium: is about 0.3 mEq/ Kg lens water. Increased concentrations are cytotoxic and thought to contribute to the development of cataract.

Carbohydrates: Glucose in the lens is approximately about 1.0mM. In the lens energy production is almost entirely dependant upon the metabolism of glucose.

Amino acids: The concentration of amino acids in the lens generally exceed that in the aqueous humor.

Ascorbic acid: Might participate in the modulation of the hexose monophosphate shunt.

Choline: The normal human lens contains about 1 mMP-choline, but this is substantially lower in cataractous lenses perhaps due to increased membrane permeability.

Inositol: Myoinositol, the most abundant isomer of inositol, is actively transported into the lens by sodium-dependent carrier-mediated mechanism. In several forms of cataract, particularly diabetic cataract, myoinositol levels are significantly reduced.

Lens Metabolism: The metabolism of the lens is entirely directed towards the maintenance of transparency. The main location of lens metabolism is in the lens epithelium. Composition and metabolism of the lens undergo significant changes as the lens changes. Certain of these changes might contribute to the development of cataract or atleast render the lens more susceptible to cataractogenic stress.

Carbohydrate metabolism:

The four processes through which carbohydrate metabolism occurs are:

1. Glycolysis

2. Krebs cycle

3. Hexosemonophosphate shunt

4. Sorbitol pathway.

Protein Metabolism: Protein synthesis takes place predominantly in the lens epithelium and the outer cell layers. It involves the transfer of genetic information via m-RNA to the ribosomes. The energy is supplied in the form of ATP derived from carbohydrate metabolism.

Glutathione and oxidation – reduction pathways:

Glutathione plays a central role in protecting the lens from oxidative insult. Glutathione plays a critical role in the oxidative defense mechanisms, by virtue of its sulphydryl group, perhaps in association with ascorbic acid[32].

The Chemical Pathology of the Lens[13]:Changes in the Constituents of the Lens:

Proteins: In age, the protein content of the normal lens increases in all types of cataract. The insoluble protein remains comparatively stable so that its concentration is relatively increased, while the soluble proteins are markedly decreased.

There is a loss of the β-crystallin fraction. In the later stages of development of cataract, proteolysis occurs whereby the soluble proteins are broken down by proteolytic enzymes. Complete hydrolysis increases the osmotic pressure 400 times leading to an enormous imbibition of water with a consequent swelling followed by an almost complete disappearance of the lenticular substance.

Peptides: Glutathione tends to decrease with age and diminishes markedly in all the forms of cataract. This reduction is possibly due to the failure in synthesis. Ascorbic acid shows a comparable diminution in age or with the development of cataract but this is slight and does not occur at an early stage.

Nucleotides: ATP, diphosphopyridine (DPN, DPNH) and triphosphophyridine nucleotides (TPN, TPH) become deficient in age and decrease in cataract.

Lipids: The total lipids increase in age and in cataract.

Inorganic Materials: There is increase in calcium in cataract. The content of potassium simultaneously decreases. Contemporaneously, the sodium increases, chlorides and inorganic phosphates also increases. This shift in the content of cations due to free permeability of the cellular membranes may lead to an increase in osmotic pressure and this participates in the intumescence of the lens[13].

The Pathological Metabolism of the Lens:

Interruption of metabolism affects most acutely the actively dividing epithelial cells. Carbohydrate metabolism is retarded. Anaerobic glycolysis gradually falls with age, with this fall there is a diminution in the production of high energy phosphates, particularly ATP. The HMP shunt diminishes at an early stage. Sorbitol pathway is probably of importance in the development of sugar cataracts. The main evidence of a reduction in oxygenation is the diminution of glutathione at an early stage. The principal change in the protein metabolism in age and most types of cataract is a decrease in the synthesis of the metabolically active soluble proteins.

Cataract: Any opacity in the lens is called cataract. The term cataract was introduced by Constantinus Africanus (AD 1018) as “cataracta” meaning to rush down, as a waterfall or portcullis. The Latin word suffusio was translated by the Arabic scholars as Nuzel-el-ma, a flowing down of water. In Arabian countries cataract is known as ‘the blue water’.

Etiological Theories:

1. Biological

a) an expression of senility

b) genetic tendencies.

2. Immunological

3. Functional, due to excessive accommodation.

4. Local disturbances

• of nutrient supply

• chemistry of lens

• radiational damage.

5. General metabolic disturbances.

Clinical types of Cataract:

1. Congenital cataract

2. Evolutionary cataract

3. Senile cataract

4. Metabolic cataract

5. Syndermatotic cataract

6. Cataract in osseous disease

7. Osmotic cataract

8. Multiple syndromes with cataract

9. Complicated cataract

10. Toxic cataract

11. Traumatic cataract[13].

The Decision to Operate:

Ophthalmic surgeons have taken a much more liberal approach to the indication for cataract surgery. Ophthalmic surgery has progressed far beyond the era of extra-capsular extraction when it was necessary to wait until the cataract was nearly mature with vision reduced virtually to light perception.

A modern attitude towards the technologic explosion insists more than even on conventional points of view about patient's needs. The best guide to decision making will be the thought whether the operation will benefit the patient. There is a wide variance in opinion concerning the advisability of performing cataract surgery on both eyes. Bilateral cataract surgery on the same day is risky and warranted only in the rarest circumstances. There is also a wide diversity of opinion on the need for cataract extraction in the second eye after successful cataract surgery has been performed on the first. Operating the second eye will definitely improve the quality of vision, but it will not reduce the burden of blindness in the country[12].

Preoperative Physical Examination[12]:

Before planning cataract surgery, the surgeon must be aware of the physical condition of the patient. A careful medical history is obtained, and a physical examination is performed. Only rarely need a patient be refused surgery because of a physical debility. The surgeon must use good judgement when considering surgery on feeble, elderly, and imfirm individuals. Cataract surgery is usually unjustified on a patient with an overwhelming medical problem such as terminal stage of malignancy. If the patient has diabetes, control should be adequate and diabetic status monitored. If the patient is on anticoagulation therapy, it should be discontinued to allow the prothrombin level to return to normal before surgery. Severe anemia should be corrected, high blood pressure reduced, and all signs of congestive failure eliminated.

Respiratory problems such as bronchitis and asthma should be controlled. Patients' emotional status should also be taken into consideration. Surgery should not be undertaken on a patient who is suffering from depression or one whose peptic ulcer is

active or whose colitis is uncontrolled. A question about the patient's personal habits, idiosyncrasies to drugs, allergies, and current medication regimen is important. Certain minimum laboratory tests are required on every admitted patient. Other tests may be performed as indicated by the results of the physical examination.

Preoperative Ocular Examination:

Visual acuity should be determined for both near and far distance. The degree of visual impairment caused by the lens opacity should be estimated. Retinoscopy and ophthalmoscopy are useful in evaluating the media. If the cataract does not appear dense enough to reduce vision to the low level found, macular degeneration, optic nerve disease, or amblyopia may be present. With a cataract of mild or moderate density, a careful and accurate visual field examination is possible. Peripheral field may be tested by the finger counting method. Intraocular pressure should be checked. Pupillary reactions to light can be taken as a value for prognosis for restoration of central vision. The ability of the pupil to dilate adequately should be estimated before surgery. The detection of light projection is of utmost importance. It is a test of gross retinal function. Macular function can be assessed by two light discrimination, maddox rod test. The entoptic visualization of retina is also useful for retinal function. Slit lamp examination is done to determine the health of the cornea.

The type of cataract and the condition of the capsule can also be evaluated. Unless the cataract is almost mature, a reasonable adequate fundus examination is usually possible. The indirect ophthalmoscope with its intense illumination system is invaluable.

The possibility of infective complications should be excluded. Gross focal sepsis, such as abscessed teeth, should be eliminated. Conjunctival sac should be examined. If regurgitation is found on pressure on the lacrimal sac area, or a mucocele is present, a nasal drainage surgery should be performed first[33].

History of Cataract Surgery Techniques

Year	Technique	Place	Surgeon
800	Couching	India	Unknown
1015	Needle aspiration	Iraq	Unknown
1100	Needle aspiration	Syria	Unknown
1500	Couching	Europe	Unknown
1745	ECCE inferior incision	France	Daviel
1753	ICCE by thumb expression	England	Sharp
1860	ECCE superior incision	Germany	Von-Graefe
1880	ICCE by muscle-hook zonulysis & lens tumble	India	Smith
1900	ICCE by capsule forceps	Germany	Verhoeff & Kalt
1940	ICCE by capsule suction erysiphake	Europe	Stoewer & Barraquer
1949	ECCE with PC IOL	England	Ridley
1951	AC IOLs	Italy Germany	Strampelli Dannheim
1957	ICCE by enzyme zonulysis	Spain	J.Barraquer
1961	ICCE by capsule cryoadhesion	Poland	Krawicz
1967	ECCE by phacoemulsification	US	Kelman J.Shock
1975	Iris-pupil supported IOLs	Netherlands	Binkhorst Worst
1984	Foldable IOLs	US, South Africa	Mazzocco Epstein

Cataract Surgery:

Cataract extraction is the most frequently performed operation in patients over 65 years of age. A technologic explosion has arisen in the techniques of cataract extraction.

The different methods of cataract surgery are:

1. Standard intracapsular cataract extraction.
2. Standard extracapsular cataract extraction.
3. Phacoemulsification

Intracapsular cataract extraction:

a) Cryoextraction.

b) Capsule forceps extraction

c) Erisophake extraction.

Extracapsular cataract extraction:

a) Conventional ECCE

b) ECCE by small incision cataract surgery (SICS) or small incision manual nucleus fragmentation.

c) Lensectomy

d) Phacoemulsification[33].

The main steps of cataract surgery are:

1. Adequate anesthesia and akinesia by local anesthesia.
2. Exposure of the operative field by lid speculum.
3. Fixation of eye with a superior rectus bridle suture.

4. Limbal incision of 8 mm in cases of ECCE, 6-6.5mm scleral tunnel incision made 2.5mm behind the anterior limbus in manual SICS after taking fornix based conjunctival flap.

5. A side port paracentesis to facilitate intraocular manipulations in manual SICS.

6. A 6-6.5mm can opener capsulotomy or continuous curvilinear capsulorrhexis with a bent26G needle after filling the anterior chamber with a viscoelastic.

7. Delivery of the nucleus with a vectis and muscle hook after extending the incision in ECCE, and in manual SICS delivery using Sinsky hook dialer and vectis after prolapsing into anterior chamber.

8. Aspiration of residual cortical matter using simcoe irrigation aspiration cannula.

9. In the bag implantation of a 6.5 mm optic three piece PMMA IOL.

10. Closure of the wound with 10-0 or 8-0 monofilament interrupted sutures

in ECCE. In manual SICS the incision is self sealing.

Complications of Cataract Surgery:

Over the years, the technique of cataract surgery has evolved into a safe and successful procedure for visual rehabilitation of the cataract blind. The conversion from ICCE to ECCE, advent of microsurgery, availability of fine suture materials, affordable high quality intraocular lens implants and vitrectomy instrumentation have significantly decreased the complications of cataract surgery[11].

Intraoperative Complications:

Anesthesia related complications:

Incomplete local anesthesia and akinesia: The danger is greatest from activity of the rectus muscles. This may lead to reduction of working space in the anterior chamber, bulging of ocular contents, gaping of the wound, and prolapse and vitreous loss[34] .

Acute anaphylactic reaction (neuro ophthalmic reflexes)

Reflex	Afferent pathway	Efferent pathway	Precipitating factor	Symptoms & signs	Prophylaxis & treatment
Oculo cardiac	Long & short nerve to ciliary ganglion	Motor nucleus of vagus	Pressure, torsion, pulling on extra-ocular muscles	Sinus Bradycardia Ectopic beats Sinus arrest	IM/IV Atropine or Glycopyrrolate
Oculo respiratory	Same as oculo cardiac	Via a connection between trigeminal sensory nucleus & pneumatic centre & medullar respiratory centre		Shallow breathing, bradypnoea or respiratory arrest	Controlled ventilation on children undergoing squint surgery
Oculo emetic		Reflex action	Traction on extraocular muscles	vomiting	

Globe Perforation:

This is a rare complication as reported. 1 in 12,000 cases composed of both peribulbar and retrobulbar anesthesia, 1 in a series of 1000 and 3 in a series of 4000 retrobulbar anesthesia[12]. It occurs more frequently in elongated myopic eyes and in deep set eyes. The diagnosis of globe perforation may be suspected by the presence of hypotony, poor red reflex and marked pain at the time of perforation.

In cases with dense cataract, cataract extraction should be performed with peribulbar anesthesia. The post-operative management include examination of the retina to assess the extent of retinal damage and argon laser photocoagulation or cryopexy for retinal breaks, if necessary. If perforation of the eye is suspected and the cataract is not dense, cataract surgery should be postponed. Cryopexy or photocoagulation should be done as soon as possible to close the perforation site.

Prevention: Globe perforation can be prevented by moving the needle to and fro after insertion into the orbital tissue. If the whole globe moves, it means that either the coats of the globe are incarcerated or the needle has passed through the coats and the tip is in the vitreous.

Retrobulbar Hemorrhage:

The incidence of serious retrobulbar hemorrhage has been reported as 1% to 3%[12]. The increased orbital pressure may tamponade the small nutrient vessels in the optic nerve resulting in severe visual loss and late optic atrophy in the absence of retinal vascular occlusion.

Management includes lateral canthotomy, digital pressure, osmotic diuresis, and anterior paracentesis. Cataract surgery should not be attempted when a serious

retrobulbar hemorrhage occurs. The use of blunt needles and the technique of peribulbar anesthesia can decrease the incidence of retrobulbar hemorrhage. Other complications resulting from retrobulbar injection include central spread of anesthetic, retinal vascular occlusion, optic nerve trauma and optic atrophy.

Surgery related complications:

Detachment of Descemet's Membrane: The cause is probably a mechanical one related to faulty instrumentation or technique associated with the added possibility of an inherent predisposition. The detachment looks like small, transparent tag curling inward from the curved lip of the incision. When Descemet's membrane is curled inward, the anterior chamber is filled with air and the curled-up membrane is manipulated with a cyclodialysis spatula.

When Descemet's membrane is separated from the stroma but not curled inward, filling the anterior chamber with air or a viscoelastic material is sufficient. In some cases, the detached edge of Descemet's membrane can be sutured to the area of the surgical incision. If these methods fail, a penetrating keratoplasty should be performed.

Premature entry: Occurs when dissection of the sclera is too deep and the anterior chamber is entered in the AC angle. This will lead to iris prolapse and wound leak. Suturing of the wound is required at the end of the surgery[35].

Iris Prolapse: Iris prolapse is more common during larger incision surgery such as a planned extracapsular cataract extraction. Iris prolapse during cataract surgery is troublesome to the extent that it interferes with the insertion of instruments into the eye

and often causes the pupil to contract. A correctly performed incision that extends into clear cornea will usually prevents its occurrence. Elevation of IOP by excessive injection of fluids, including viscoelastic agents, should be avoided.

Hemorrhage:

Excessive bleeding during the surgery is disturbing because it may reduce the surgeon's visibility. Hemorrhage can arise from scleral bed vessels during sclera tunnel incision and from iris vesels while performing iridectomy or if iridodialysis is produced. Viscoelastic material and air will limit the intraocular bleeding by
tamponading the bleeding site.

Suprachoroidal Hemorrhage:

It is usually venous in origin and is often associated with hypotony accompanying intraocular surgery. Some of these are self limiting. If the globe becomes firm, the incision should be closed. A posterior sclerotomy may be performed to drain the choroidal hemorrhage.

Expulsive Hemorrhage:

Expulsive hemorrhage is one of the most frightening and serious complications of cataract surgery. Incidence of expulsive hemorrhage is reported to be around 0.2%[12]. It usually occurs in aged patients with vascular disease and hypertension immediately after the intraocular pressure has been lowered on completing the incision, or occasionally

some hours or days after surgery. Apart from immediate sclerotomy, the only expedient usually available is evisceration of the globe.

Prophylaxis: Special attention should be given to patients with extensive vascular disease. If an expulsive hemorrhage occurs in the first eye, it should be expected and anticipated in the second. Phacoemulsification is ideally suited for an expulsive hemorrhage because the 3mm incision can be rapidly closed in case of a hemorrhage.

Prevention: The incidence of expulsive hemorrhage can be decreased by ensuring a pre-operative soft eye, treating hypertension adequately and controlling IOP preoperatively, if raised.

Iridodialysis: Iridodialysis can occur during enlargement of the incision during iridotomies, or during insertion of a phacotip or irrigation/ aspiration tip in high pressure eyes. If excessive it should be sutured with 10-0 nylon or 10-0 polypropylene sutures[12].

Implant Insertion Errors:

Faulty implantation of a posterior chamber lens is rare. The most frequent is placing one loop in the capsular bag and one outside the bag. This may be due to a faulty capsulorrhexis, high vitreous pressure or intraoperative bleeding that reduces visibility. In most cases, it is corrected if easily seen.

Iris Sphincter Damage:

Early on, this resulted from inadvertent freezing of the iris sphincter during cryoextraction of the lens nucleus (intracapsular cataract extraction). The most common

cause of iris sphincter damage during an extracapsular cataract extraction is expression of the nucleus through a poorly dilated pupil or during phacoemulsification if the ultrasonic tip inadvertently engages the iris edge.

Operative Loss of Vitreous:

Aside from expulsive hemorrhage, loss of vitreous is the most serious ocular complication that occurs during cataract surgery. The incidence of vitreous loss in the practice of a skilled surgeon performing intracapsular cataract extraction should be approximately 3% and that in extracapsular cataract extraction should be lower.The chances of vitreous loss in the second eye is probably much higher if vitreous was lost during surgery on the first eye.

Diagnosis: Vitreous loss is diagnosed by the presence of vitreous in the anterior chamber, distortion of the pupil, presence of vitreous in the wound and the accompanying posterior capsular rent or zonular dehiscence.

Treatment: If the prolapse occurs immediately after the section is made, a complete iridectomy and extraction is usually necessary. If it accompanies or follows extraction of the lens, a corneo-scleral suture should be immediately tightened, the wound freed from vitreous as much as possible and the anterior chamber filled with air after the remaining sutures has been tied. The aim of vitrectomy in vitreous loss is to prevent vitreous incarceration in the corneoscleral section and adhesion of vitreous to the corneal endothelium.

Prevention: Preoperative softening of the globe is the best method to prevent vitreous loss. Adequate lid and globe anesthesia and minimal external pressure on the globe can decrease the incidence of vitreous loss.

Posterior capsule tear: Posterior capsule tear is a common complication that can occur during cataract surgery. The incidence following ECCE ranges from 0.2% to 10.3%, during phacoemulsification ranges from 0.7% to 16%[36]. The incidence varies with the experience and skill of the surgeon. A higher incidence is associated with cataract with pseudoexfoliation, diabetes mellitus and trauma. The causes of posterior capsule tear during ECCE include the smaller incision size jeopardizing the manual expression of the nucleus, trauma during capsulotomy and irrigation – aspiration, small pupil in the course of cortex aspiration, and high

pressure from the posterior chamber.

The conventional management consists of prevention of mixture of cortical matter with vitreous, dry aspiration, and anterior vitrectomy, if required. If the tear is small, the IOL can be placed in the sulcus, if the capsular rim is available. Scleral fixated posterior chamber lenses and anterior chamber intraocular lens can be implanted when the posterior capsule tear is large[36].

Zonular Dialysis:

The most frequent cause of intraoperative zonular dialysis is the pseudocapsular exofoliation syndrome. Intraocular maneuvers such as traumatic capsulorrhexis, excessive manipulation of the nucleus and inadvertent aspiration of the anterior or posterior capsule can lead to zonular dialysis. When zonular dialysis occurs, a

viscoelastic agent should be placed over the area of dialysis to minimize the risk of vitreous prolapse. If the dialysis is less than 4 clock hours, capsular bag implantation is possible as long as the anterior capsule ring is intact.

Dropped Nucleus and Retained Lens Fragments:

Rupture of the lens capsule and operative loss of the vitreous is a prerequisite for its occurrence. Unencapsulated retained lens material in contrast to dislocated lenses with intact capsules may cause significant inflammation. Pars plana vitrectomy has been recommended in patients with a retained nuclear fragment and any evidence of inflammation or persistent increase in IOP[12].

Dislocated Intraocular Lens:

Dislocation of an IOL into the vitreous may occur in the presence of a large break in the posterior capsule. An intraocular lens resting on the retina or embedded into the vitreous base is often well tolerated. Variable options for treatment include simple observation, removal with or without exchange, and repositioning.

Postoperative Complications:

Complications occurring within first three weeks of surgery are classified as early and those of slower or delayed onset as late[33].

1. **Anterior chamber depth abnormalities:**

a) **Delayed restoration of the anterior chamber**: This complication was seen with far greater frequency before modern cataract surgery techniques. The common causes of

shallow anterior chamber include wound leak, pupillary block, hyposecretion of aqueous and choroidal detachment.

Wound leak: It is the most important primary factor responsible for the shallowing of the anterior chamber. It is caused by many factors.

i) Inadequate incision.

ii) Inadequate suturing.

iii) Poor coaptation of the wound margins.

iv) Accidental sclerotomy and excessive cauterization.

v) Incarceration of material between the lips of the wound such as iris, lens fragments, vitreous, lint, rubber, glass suture pieces and cilia.

vi) Accidental trauma, which causes wound separation.

vii) Poor ocular structures such as thin scleral coats of highly myopic and juvenile eyes and eyes that have had previous intraocular surgery.

viii) Elevation of intraocular pressure.

The eye is usually significantly hypotonic. A choroidal detachment is often found in cases in which the anterior chamber does not spontaneously reform within 48 hours. Wound leak is diagnosed by Siedel's test. Complications of wound leak are glaucoma, hypotension, inflammation and infection, iris prolapse, bullous keratopathy, epithelial down growth and fibrous ingrowth. Preventive measures include meticulous care in wound closure, avoidance of excessive cauterization, prevention of incarceration of

vitreous or iris in the surgical wound in cases of operative loss of vitreous, protection of the operated eye during sleep and treatment of elevated intraocular pressure[12].

Treatment: The treatment of shallow anterior chamber include instillation of a short acting mydriatic and a firm pressure dressing if the anterior chamber is flat 24 hours after surgery. Surgical intervention is indicated if shallow anterior chamber persists for more than 5 to 7 days or earlier if corneal decompensation is evident.

Pupillary block:

Pupillary block is defined as a failure of communication of aqueous between the anterior and posterior chambers due to obstruction of the pupil and surgical openings in the iris.

The causes of pupillary block after cataract extraction include:

1. Leaky wound: Most frequent cause of early pupillary block.
2. Post operative iridiocyclitis – may cause a relatively late pupillary block.
3. Posterior vitreous detachment associated with pooling of retrovitreal aqueous.
4. Dense impermeable anterior hyaloid membrane.
5. Pupillary block by air.
6. Swollen lens material behind the iris.
7. Choroidal detachment and hemorrhage.
8. Scleral collapse – may cause operative loss of vitreous or a compression of the main vitreous mass against the iris.
9. Free vitreous block.
10. Anterior chamber hemorrhage.

Pupillary block can be diagnosed by a shallow anterior chamber with raised intraocular pressure[12].

Treatment:

Prophylaxis: Prophylactic measures to prevent pupillary block include adequate wound closure, reduction of post-operative inflammation, treatment of a leaking wound, early restoration of the anterior chamber, avoidance of large amounts of air within the eye, and performing adequate iris openings during cataract extraction.

Medical Treatment: Consists of dilating the pupil as widely as possible with mydriatics and cycloplegics. Systemic administration of hyperosmotic agent results in dehydration of the vitreous that may displace the IOL and the anterior hyaloids membrane posteriorly from its closely apposed position against the back of the iris, thus allowing mydriasis to occur.

Surgical Treatment:

Surgical therapy is performed when medical treatment fails. It includes:

1. Surgical iridectomy.
2. Laser iridotomy
3. Incision of anterior hyaloid membrane.
4. Partial anterior vitrectomy through a pars plana approach.
5. Through and through incision of the vitreous.
6. Cyclodialysis or cyclocryotherapy when permanent angle damage has occurred.
7. Suprachoroidal tap – if a choroidal detachment is associated with aphakic pupillary block.

Choroidal detachment:

The exact etiology of choroidal detachment is not known. It is not clear as to whether hypotony causes choroidal detachment or vice-versa. Postoperative choriochoroidal detachments may occur immediately after surgery, 7 to 21 days after surgery or it may be delayed, occurring months or years after surgery.

Immediate choroidal detachments were very common after intracapsular cataract extraction. The incidence is uncertain, but O'Brien found an incidence of 93%[37].

Postoperative choroidal detachments occurring 7 to 21 days after surgery may be a persistence of the immediate type or may occur later because of wound leak, delayed wound healing or rupture of an inadequately healed wound. The incidence of this type of choroidal detachment was probably about 5% to 8%. The course is usually benign, with the detachment subsiding in 2 to 3 weeks. Choroidal detachment occurs some months or years after cataract surgery.

The mechanism of delayed choroidal detachments is unclear, although some cases seem to be associated with trauma, with reopening of the wound being likely.

Treatment: Most postoperative choroidal detachments subside within three weeks and therefore require no treatment unless the anterior chamber remains shallow.

Prophylaxis: A precipitous fall in intraocular pressure during surgical decompression should be avoided.

Medical Treatment:

1. Cycloplegic agents, minimize tension on the uvea by releasing the ciliary muscle.
2. Acetazolamide is used when there is wound leak.

3. Systemic administration of a hyperosmotic agent along with cycloplegics if there is posterior misdirection of aqueous.

Surgical Treatment:

Surgical treatment is indicated if the shallow anterior chamber persists for more than 8 days because of the dangers of permanent angle damage from anterior iris adhesions and secondary glaucoma. The surgical procedure consists of:

1. Performing a suprachoroidal tap.

2. Placing air in the anterior chamber.

3. Repairing the leak, if found.

Glaucoma in Aphakia and Pseudophakia: The incidence of glaucoma after all cataract extractions was reported as 12% by Duke Elder[13] and 0.7 to 7% according to a review by Francois[38]. A transient rise in intraocular pressure during the first week of cataract surgery is a common occurrence with all types of cataract surgery. The cause of this pressure elevation is not entirely clear, but it may be swelling of the trabecular meshwork fibers or breakdown of the blood-aqueous barrier. The combination of a rapid phase of aqueous formation with a water tight wound closure may account for this pressure rise.

Mechanisms of Pressure Elevation:

A) Open Angle:

1. Early post-operative period/ reversible, self-limited:

a) **Alpha chymotrypsin**: In the era of ICCE, alpha-chymotrypsin – induced glaucoma, also referred to as “enzyme glaucoma” was common occurring in up to 72% of cases[39].

The exact mechanism of enzyme glaucoma is not clearly understood. Enzyme glaucoma typically appears 2 to 5 days after cataract extraction. Preventive and therapeutic measures include the use of a smaller volume and or a weaker concentration of the enzyme (1:10,000 instead of 1:5000), thorough irrigation of the enzyme solution before delivery of the cataract, intracameral acetylcholine intraoperatively, topical timolol or pilocarpine at the end of surgery, and oral acetazolamide post-operatively.

b) **Blood or other particulate material**: The trabecular meshwork may become temporarily occluded by particulate material such as blood, pigment, inflammatory debris, retained cortical material, or any combination of these. Intraocular pressure elevation under these circumstances is usually self-limited. In cases of intractable glaucoma and or with impending corneal blood staining, surgical evacuation is indicated[40].

c) **Viscoelastic substances**: With the widespread use of viscoelastic substances today, sodium hyaluronate is well recognized as a culprit responsible for postoperative IOP elevation, especially in eyes with impaired aqueous outflow. It is believed that these highly viscous substances block the flow of fluid through the trabecular meshwork. Even when removed, viscoelastics can cause a significant pressure elevation but to a lesser degree than when they are not removed. It has been shown that postoperative IOP is high in most cases within 6 hours of surgery if material is left in the eye. It was recommended that this rise in pressure can be eliminated or greatly minimized by irrigating or aspirating the sodium hyaluronate from the eye at the end of surgery.

d) **Idiopathic**: Intraocular pressure may still rise following cataract extraction without any detectable cause accounting for up to 23% of routine cataract extractions. Gross et al

found a higher incidence of IOP rise after ECCE with intraocular lens implantation than with phaco-emulsification and intraocular lens implantation[41]. The exact mechanism by which IOP rises temporarily in an otherwise perfectly normal eye following cataract extraction is not yet fully understood.

However, several theories have been proposed.

1. Trabecular meshwork edema caused by the surgical trauma itself.
2. Mechanical distortion of the angle deformation by sutures.
3. Inflammation

e) **Neodymium: Yag Laser Posterior Capsulotomy**: Transient IOP elevation following Nd: Yag Laser Posterior capsulotomy is said to occur in 95% of cases especially those with preexisting glaucoma,or those in which the prelaser IOP is 20mm Hg or higher. It is also more common in aphakic than pseudophakic eyes. The magnitude of post-laser IOP rise does not seem to be related to total laser energy used nor to the size of the capsulotomy produced[40].

2. Sustained / Permanent IOP Elevation:

a) Pre-existing Primary Open – Angle Glaucoma.

b) Positive steroid response: If IOP starts rising only a few weeks after surgery, then a positive steroid response has to be kept in mind.

c) Vitreous in the anterior chamber: Gluacoma with free vitreous in the anterior chamber results when vitreous fills the anterior chamber and blocks the trabecular meshwork.

d) Ghost cell glaucoma: Ghost cell glaucoma in the aphakic eye occurs as a result of chronic vitreous hemorrhage. Vitrectomy is frequently necessary.

e) Irreversible trabecular mesh work damage.

f) Inflammation.

g) Late hemorrhage: Occurs months or years after cataract extraction, either as a result of vascularization of the cataract incision, the so called Swan syndrome, or because of iris and or ciliary body chafing or erosion by the intraocular lens.

B) **Closed Angle**:

1. With pupillary block: Synechial closure of the angle is said to be the most common cause of glaucoma in aphakia and pupillary block is the most common cause of angle closure after cataract extraction. The various causes of papillary block in aphakia and pseudophakia include:

a) Air bubble: This is self limited in most cases, or easily reversible by proper positioning of the patient and mydriasis.

b) Vitreous face: Shaffer stressed the role of posterior vitreous detachment in predisposing to pupillary block of vitreous and to malignant glaucoma in aphakic eyes[42]. Pupillary block by vitreous has been described to occur following Nd: YAG laser posterior capsulotomy. The classical treatment for pupillary block in aphakia has always been surgical iridectomy. Other modalities include anterior vitrectomy, mechanical separation of iridovitreal adhesions with a blunt spatula, and mechanical removal of the vitreous through the pars plana, either by vitrectomy or by ultrasonic fragmentation.

c) The posterior lens capsule: In the absence of an intraocular lens, and sometimes in its presence, the posterior capsule may come into firm apposition with the iris, sometimes occluding the pupil. This phenomenon of retrocapsular accumulation of aqueous is referred to as anterior aqueous misdirection or posterior pupillary block.

d) Intraocular lens: Pupillary block has been described for iris – supported lenses, and anterior as well as posterior chamber lenses. The absence of a functioning surgical iridectomy is known to predispose to pseudophakic pupillary block, especially with anterior chamber lenses.

e) SeclusioPupillae: Total posterior synechiae is a cause for pupillary block in aphakic or pseudophakic eyes whenever there is excessive post operative inflammation. Laser iridotomy is usually curative in the early stages.

f) Silicone Oil: This is seen in aphakic patients undergoing retinal reattachment surgery with intraocular silicone oil injection.

2. Aqueous Misdirection: Classical referred to as malignant or ciliary block glaucoma is a rare complication of cataract extraction. The predisposing factors include:

a) Small sized globe.

b) Post operative leakage from the surgical incision.

c) The absence of a functioning surgical iridectomy.

d) A combination of the above factors.

Medical treatment is frequently ineffective. Other options include deep incision and aspiration of the vitreous with anterior chamber deepening, anterior vitrectomy, pars plana vitrectomy, shrinkage of the ciliary process by transpupillary argon laser photocoagulation, or in terminal cases – cyclodestruction.

3. Without Pupillary Block:

The anterior chamber angle may become permanently and irreversibly damaged by peripheral anterior synechaie via mechanisms other than pupillary block.

a) Inflammation/ hyphema.

b) Prolonged anterior chamber shallowing.

c) Iris incarceration in the surgical incision.

d) Intraocular lens Haptics: Anterior chamber intraocular lenses have been shown to cause progressive synechiae in the anterior chamber angle.

e) Pre-existing angle closure: The process of angle closure by any mechanism may precede cataract formation and surgery.

f) Neovascular glaucoma: Cataract extraction, ICCE much more than ECCE, is known to accelerate anterior segment neovascularization in eyes with proliferative diabetic retinopathy. The management of neovascular glaucoma in aphakia should address first the primary cause of ischemia.

g) Epithelial downgrowth: Glaucoma in epithelial downgrowth is though to result from peripheral anterior synechiae, pupillary block caused by the epithelial membrane itself, direct obstruction of the outflow channels by epithelial cells, or necrosis of the trabecular meshwork.

h) Fibrous ingrowth: Glaucoma associated with fibrous ingrowth results from direct involvement of the anterior chamber angle by the ingrowing sheets of fibrous tissue. Treatment is extremely unrewarding.

i) Endothelial proliferation.

Treatment:

A) **Medical Treatment**: Includes miotics and carbonic anhydrase inhibitors. Some cases of pupillary block may benefit from intensive cycloplegics and mydriasis.

B) **Laser Treatment**:

1. Argon laser iridotomy and photomydriasis are useful to relieve aphakic and pseudophakic pupillary block.

2. Nd: YAG Laser: The Nd: YAG laser is particularly useful for iridotomy in pupillary block.

C) **Surgical Treatment**: Glaucoma procedures are much less successful in aphakia and pseudophakia. Cyclocryotherapy and cyclodialysis are reserved for aphakic eyes. The choice of surgical procedure depends on the condition of the eye in question, that of the contralateral eye and the general status of the patient as a whole[37].

Corneal Complications:

Corneal Edema: is one of the most serious complications of cataract surgery. The incidence has decreased with improved surgical management. However, both toxic and mechanical trauma to the endothelium can result in corneal edema especially in predisposed eyes.

According to a study there was no statistically significant difference between the mean endothelial cell loss of 17.1% in the intracapsular group and 13.6% in the extracapsular group[43]. Extracapsular cataract extraction with intraocular lens implantation, even when performed by inexperienced surgeons, caused no greater corneal endothelial damage than intracapsular extraction. Phacoemulsification is associated with endothelial cell loss of about 4-10%[44].

Corneal edema is graded from mild to severe grade as:

1. Striate keratopathy consists of folds in the Descemet's membrane with no significant epithelial changes. Most cases are self limiting.

2. Microcystic edema: consists of microcystic epithelial edema with Descemet's folds and stromal edema.

3. Bullous keratopathy: There are sub-epithelial bullae with stromal edema and descemet's folds. Rupture of bullae leads to exposure of corneal nerves causing pain. The incidence of pseudophakic bullous keratopathy with the current technique of posterior chamber intraocular lens implantation ranges from 1% to 2%[45].

Causes of endothelial cell loss which cause corneal edema are:

1. Preoperative factors

- Fuch's dystrophy, cornea guttata
- Glaucoma
- Previous surgery

2. Intraoperative factors:

- Mechanical trauma
- Introduction of particulate matter into the anterior chamber
- Irrigating solutions
- Epinephrine due to its preservative 0.1% sodium bisulfite.
- Excessive manipulation of the cornea
- IOL contact with endothelium.

3. Postoperative factors:

- Iritis
- Vitreocorneal adherence
- Hyphema.
- Raised intraocular pressure
- Flat anterior chamber with intraocular lens touch.

Independent predictors of endothelial cell loss were high nucleus grade, greater infusion volume, IOL type, large nucleus and total ultrasound energy[44]. The implantation of intraocular lenses have been associated with a greater loss of endothelial cell density than cataract extraction alone.

The acrylic IOL was associated with greatest endothelial cell loss. Preoperative evaluation of cornea by specular microscopy and pachymetry is important.

Causes of persistent corneal edema are:

1. Pre-existing corneal endothelial disease.
2. Trauma during surgery
3. Adherence of vitreous to the back of the cornea or incarceration in the operative wound.
4. Adherence of iris or lens capsule to the back of the cornea or incarceration in the wound.
5. Epithelial downgrowth
6. Endothelial proliferation
7. Descemet's detachment

8. Glaucoma

9. Uveitis

10. Chemical injury

11. Foreign material introduced during surgery.

Treatment is difficult unless the primary cause of the edema can be eliminated. Hypertonic agents, 2% and 5% solution and ointment of sodium chloride and bandage contact lens cause temporary relief of pain and discomfort. Surgical modalities include keratoplasty, conjunctival flap, anterior stromal puncture and cautery of Bowmann's membrane.

The other corneal complications include epithelial disruption, infections, sterile corneal ulceration, stromal melt, stripped Descemet's membrane, and epithelial and fibrous downgrowth. Meticulous cataract surgery with careful attention to protecting the cornea can prevent most serious corneal complications[46].

Retained lens materials:

Inadequate aspiration of the cortex, especially behind the iris at the 12'O clock position, is the commonest cause of retained lens material after cataract surgery[21]. In case with minimal cortical matter, the anterior segment inflammation can be treated with topical steroids and cycloplegics. In cases with significant cortical residue, surgical removal is essential.

Endophthalmitis:

Endophthalmitis is one of the most catastrophic complications of surgery. The incidence of infectious endophthalmitis after cataract surgery has been estimated to be 0.07% to 0.13%[47]. The incidence may depend on the surgical procedure. After extracapsular lens extraction or phacoemulsification and intraocular lens implantation, the incidence is between 0.07% and 0.12%, whereas after secondary IOL implantation it is approximately 0.4%[47,48]. Preliminary data suggest that the incidence is not higher after sutureless phacoemulsification surgery.

The possible sources of infection include:

1. Airborne contaminants.
2. Solutions and medications
3. Tissues
4. Objects and materials
5. Miscellaneous.

Preoperative risk factors include blepharitis, conjunctivitis, canaliculitis, dacryocystitis, lacrimal duct obstruction, contact lens wear, an ocular prosthesis in the fellow orbit, host immunosuppression, diabetes mellitus, and upper respiratory tract infection.

Intraoperative risk factors include inadequate eyelid or conjunctival disinfection, prolonged surgery (longer than 60 minutes), vitreous loss, use of prolene haptic IOLs and inapparent or unplanned ocular penetration during ocular surface surgery. Vitreous loss

and introduction of additional instruments into the eye, may also increase the risk of infection.

Post-operative risk factors include wound leak/ dehiscence, inadequately buried sutures, suture removal, vitreous incarceration in the surgical wound, and the presence of a filtering bleb. Scleral tunnels which lie in relatively nonvascular sclera, create a potential abscess cavity when inoculated by an infectious organism[49] .

Endophthalmitis after cataract surgery shows a higher incidence when no antibiotics are used than when preoperative topical antibiotics and subconjunctival antibiotics are used: The use of topical 5% povidone – iodine solution into the conjunctival sac not only reduces the bacterial load but also decreases the incidence of culture positive endophthalmitis.

The endophthalmitis vitrectomy study determined that of 69% of patients with confirmed microbiologic growth, 70% were infected with coagulase negative micrococcus (mostly S. epidermidis). Others included S. aureus, Streptococcus, Enterococcus, Gram-negative species and certain fungi[49.]

The symptoms and signs of postoperative bacterial infections manifest to some degree in 24 to 48 hours. The patient complains of an inordinate amount of pain, increasing hyperemia, eyelid edema, undue corneal edema and anterior chamber and vitreous reaction. If the process is manifested soon after surgery and the course is fulminating, pseudomonas infection is a likely cause. The various forms of delayed clinical post operative endophthalmitis include mycotic, bacterial, the vitreous wick syndrome and endophthalmitis associated with postoperative filtering bleb. P.acnes must be considered in cases of delayed onset prolonged inflammation after ECCE with PCIOL

implantation. A white plaque on the peripheral lens capsule is frequently seen. The mechanism of inflammation is unclear.

Post operative mycotic endophthalmitis appears to be increasing in frequency. It is unknown whether this increase results from expanded use of steroids, antibiotics or both. The entire tempo of the process is usually low grade. The various fungi include Aspergillus, Cephalosporium, Fusarium volutella, Aeromonium species, candida, Sporotrichum schenkii[12].

Management:

A definitive etiological diagnosis is often difficult. Nonetheless, every effort should be made to establish an etiologic diagnosis. Separate smears and cultures should be taken from material in the conjunctival sac, from the anterior chamber, and from the vitreous because they may differ in their microorganismal content. Slides are prepared for Gram's, Giemsa stains and modified Grocott-Gomori methenamine silver stain for fungi. Cultures are made on blood and chocolate agar, liquid brainheart infusion, thioglycolate and Sabouraud's agar. Negative smears and cultures do not rule out intraocular infection. Although paracentesis of the anterior chamber is a rationale and rewarding diagnostic approach, Forster and co-workers have shown that aspiration of the vitreous proves more specific.

Prophylaxis:

The prevention of post-operative infection should include the elimination of sources of infection. It is important to control or treat any identified risk factors before elective surgery is performed. Prophylactic preoperative topical antibiotics have caused a reduction in the number of potential pathogens. Antibiotic – soaked collagen shields

provide an alternative to eyedrops or subconjunctival injections. The efficacy of postoperative sub-conjunctival antibiotics is unproved. Antibiotics can also be used in the intraocular infusate. The role of heparin in the prevention of endophthalmitis has not yet been identified. Heparin-coated lenses, as well as low molecular weight heparin added to the intraocular infusion, have been proposed.

Treatment:

Administration of intravitreal antibiotics is the mainstay of treatment of acute postoperative endophthalmitis. Usually a combination of two antibiotics is chosen, one for activity against coagulase negative staphylococci and the other for gramnegative bacilli infections.

Subconjunctival and topical antibiotics are often used with intravitreal antibiotics in the treatment of postoperative endophthalmitis. The rationale underlying this approach is to achieve high concentrations of antibiotics within the eye. Controversy exists as to the benefit of systemic antibiotics to treat postoperative infectious endophthalmitis. The EVS has shown that intravenous systemic antibiotics are not useful adjuncts to intravitreal antibiotics in the setting of acute and sub-acute postoperative endophthalmitis and there was no difference in final visual acuity or media clarity with or without the use of systemic antibiotics[50].

Steroids are used to limit post-inflammatory sequences. Intensive topical along with oral steroids are used if there are no systemic contraindications. Several reports suggest that intravitreal corticosteroid therapy when used in conjunction with antibiotics with and without vitrectomy, reduces the intraocular inflammatory process and secondary complications associated with microbial endophthalmitis[51] .

Controversy still exists as to whether therapeutic vitrectomy is necessary in all cases of endophthalmitis. Vitrectomy has the potential advantages of removing the infecting organisms and associated toxins, removing vitreous membranes that could lead to retinal detachment and improving intraocular distribution of antibiotics.

Currently, most ophthalmologists reserve therapeutic vitrectomy for cases with vitreous inflammation severe enough to prevent a view of the posterior pole on indirect ophthalmoscopy, progressive inflammation despite initial antibiotic therapy, and or cases that have not improved despite initial therapy[49].

If fungal endophthalmitis is suspected, Foster recommends initial vitrectomy for both diagnostic and therapeutic purposes with intraocular injection of gentamicin and amphotericin B, 0.005 to 0.1 mg. If a yeast such as candida is cultured 24 to 36 hours after aspiration, 5-fluorocytosine should be given in a dose of 100 to 150 mg/ Kg/ day orally. Nystatin, 50,000 U/ml injected subconjunctivally, may also be administered. In cases of postoperative filamentary fungal endophthalmitis subconjunctival injections of amphotericin B in doses of 0.5 to 1.0 mg are given. The role of steroids in fungal endophthalmitis is controversial[12].

Uveitis:

Cataract surgery results in transitory post-operative uveitis, which subsides rapidly. The causes of uveitis after cataract extraction include:

1. Surgical trauma like iris damage, vitreous loss.

2. Foreign material introduced during surgery such as lint, rubber, cilia, cotton, suture material and intraocular lens.

3. Toxic reaction to drugs or irrigation solutions like saline solution, balanced saline solution, α-chymotrypsin, acetylcholine, epinephrine, viscoelastic material.

4. Exacerbation of pre-existing uveitis.

5. Systemic conditions like rheumatoid arthritis, sarcoidosis, diabetes.

6. Retained lens material

7. Wound incarceration

8. Other post operative complications like epithelial invasion of the anterior chamber, fibrous ingrowth, hyphema, vitreous hemorrhage, retinal detachment, infection[12].

Surgical Trauma: Results in transitory uveitis, which subsides rapidly and usually leaves no permanent sequelae. However, in a small number of patients, the inflammatory response is excessive which results in persistent uveitis.

Aseptic Uveitis:

Aseptic uveitis is generally observed slightly later than an infection, usually 3 to 4 days after surgery. The symptoms and signs are usually less intense. A late form of aseptic uveitis can occur after an uncomplicated ICCE. The prognosis is usually favourable because the uveitis responds well to steroid therapy.

Foreign Matter Introduced During Surgery:

An enormous amount of foreign material is introduced into the eye during cataract extraction. In most instances, this material is tolerated well because the inflammatory response to particulate debris is usually small.

Pre-Existing Uveitis:

Pre-existing uveitis is not likely to cause much difficulty post operatively if cataract surgery is performed while uveitis is inactive.

Systemic Conditions:

Postoperative uveitis is a very rare complication of cataract surgery in a patient with chronic rheumatoid arthritis. Diabetes mellitus is considered as a cause of increased incidence of postoperative uveitis[52].

Wound incarceration:

The incarceration of ocular structures in the operative wound often leads to an irritable eye associated with uveitis. Iris and vitreous are most frequently involved.

Phacoanaphylactic Uveitis:

It is an acute uveal tract inflammation when the eye is exposed to lens proteins liberated within it after ECCE, discission trauma to the lens, or spontaneous rupture of its capsule. This is thought to result from a hypersensitivity reaction to retained lens protein, which continues after the extracapsular cataract extraction[11]. It usually occurs after extracapsular extraction, 2-4 weeks later. However, the reaction has been reported to occur as early as several hours or as late as several months following capsular rupture.

Generally, the symptoms abate as the lens material absorbs. The lens material may be irrigated from the eye. It should be suspected in any eye after ECCE or phacoemulsification complicated by granulomatous intraocular inflammation.

Phacotoxic uveitis:

Although some of the ocular reactions to retained lens material are anaphylactic, others represent a purely toxic reaction to lens protein. This condition was called phacogenic chronic non-granulomatous uveitis by Zimmerman. The clinical findings and histologic picture is similar to that of phacoanaphylactic uveitis.

Iris Prolapse:

It is now a relatively uncommon complication as a result of improved methods of wound closure and better suture material. The causes of iris prolapse include:

1. Poorly executed incision.
2. Inadequate wound closure
3. Vomiting, coughing and wheezing
4. Accidental trauma.
5. Pupillary block[12].

It usually occurs within 48 hours after the surgery but may be a late event appearing 10 to 15 days later[13]. There is usually a distortion of the pupil. Complications of untreated iris prolapse include defective wound healing, cystoids cicatrix, excessive astigmatism, flat anterior chamber, hypotension, striate keratopathy or corneal edema, iridocyclitis, endophthalmitis, secondary glaucoma, epithelial down growth, fibrous ingrowth, sympathetic ophthalmitis.

A recent iris prolapse should be treated promptly. Surgical excision or reposition is the treatment of choice. Other methods like photocoagulation, cryothermy coagulation and chemical cautery are not desirable. Adequate wound closure can decrease the incidence of iris prolapse to a certain extent.

Hyphema:

Hyphema may occur as an early postoperative complication,either as an isolated finding or in combination with persistant uveitis and glaucoma (UGH syndrome)[11]. Post-operative hemorrhage into the anterior chamber has been less common. Bleeding may arise from the operative wound, the iris or the ciliary body. It usually occurs between the second and seventh day.

Causes of hyphema are:

1. Defective wound healing.
2. Sutures – catgut sutures incite a greater vascular response.
3. Trauma
4. Excessive scleral incision is a cause of late hyphema occurring months or years after cataract surgery.
5. Excessive cauterization of vessels.
6. Damage to ciliary body – while performing iridectomy.
7. Abnormal vascularization of the iris.
8. Blood dyscrasias.
9. Anticoagulant therapy[12] .

A small hyphema is absorbed spontaneously in a few days. A large hyphema can overwhelm the eyes absorption mechanisms and cause a secondary glaucoma. The chief complications of hyphema are secondary glaucoma and blood-staining of the cornea. The best preventive measures for hyphema is the performance of adequate incision and closure of the wound. The eye should be protected against trauma.

The most important principle of management of hyphema is conservative.

1. Most hyphema absorb between 2 and 6 days after occurrence. Management consists of:
2. Bed rest with head of the bed elevated.
3. Carbonic anhydrase inhibitors, timolol, and osmotic agents to lower intraocular pressure.
4. Bandaging both eyes if there is a large hyphema.
5. Surgical evacuation of the blood is indicated when there is blood staining or wound dehiscence.

Vitreous Hemorrhage:

Vitreous hemorrhage is rare occurrence after cataract extraction. It is a common accompaniment of operative loss of vitreous. The most common origin of postoperative vitreous hemorrhage is blood spreading posteriorly from the anterior or posterior chamber. Most are caused by rupture of blood vessels in connection with horse shoe break or large lacerations of the retina. Retinal tears need immediate surgical management.

The complications of vitreous hemorrhage are:

1. Fibroplasia.
2. Hemosiderosis
3. Hemophthalmitis.

Intravitreal injection of urokinase has been used with success in the treatment of vitreous hemorrhage. Pars plana vitrectomy is indicated if there is lack of spontaneous absorption after 5 to 6 months[12].

Delayed Complications:

1. **Posterior capsule opacification**: Opacification of the posterior capsule caused by post-operative proliferation of cells in the capsular bag remains the most frequent complication of cataract intraocular lens surgery. The overall incidence of PCO is now rapidly decreasing from 50% in the earlier days to less than 10% currently. It is not the capsule, which opacifies; rather, an opaque membrane develops as retained cells proliferate and migrate onto the posterior capsular surface. The interval between the surgery and PCO varies widely resulting from three months to four years after the surgery. Although the causes of PCO are multifactorial as reported in several studies, there is an inverse risk with age. The incidence of PCO depends upon the time interval from the cataract surgery and upon the age of the patient. The incidence of PCO in pediatric cataract surgery approaches 100%[53].

There are two morphologic forms:

1. Epithelial pearls
2. Fibrous membranes

Postoperative proliferation of lens epithelial cells contributes to opacification membranes. This response represents aberrant attempts of the epithelium to form new lens fibers. The prevention of an after cataract is of the greatest importance.

Surgery-related factors to reduce PCO:

1. Hydro-dissection enhanced cortical clean up: Allows more efficient removal of cortex and LECs. Sealed capsular irrigation (SCI) is a type of sealed irrigation system applied to the internal eye. This isolates the internal lens capsule, and facilitates removal of residual cortical material as well as lens epithelial cells, and thus prevents/ delays capsular bag opacification.
2. In the bag capsular fixation enhances the IOL optic barrier effect.
3. Capsulorhexis edge on IOL surface: CCC diameter should be slightly smaller than that of IOL optic. This sequesters the optic in the capsular bag from thesurrounding aqueous humor.

IOL-Related Factors to Reduce PCO:

1. IOL biocompatibility: Compared with PMMA and silicone lenses, the Acry Soflenses were associated with less PCO.
2. Maximal IOL optic-posterior capsule contact by posterior angulation of IOLhaptic and posterior convexity of the optic.
3. Barrier effect of the IOL optic: A truncated, square edged optic rim appears tocause a complete blockade of cells at the optic edge, preventing capsularopacification[53].

Pharmacological Prevention of PCO:

Intraocular application of pharmacologic agents such as antimetabolites, anti-inflammatory substances, hypo-osmolar drugs, and immunologic agents has been

investigated. Toxicity to corneal endothelium and other ocular structures remains one of the major concerns for using these agents.

Treatment:

Treatment is usually undertaken for optical reasons. In the past, treatment required surgical procedure consisting of a discission of the capsule.

1. Aspiration of Elschnig pearls and polishing of the posterior capsule.
2. Neodymium: YAG laser posterior capsulotomy[12].

Cystoid Macular Edema:

CME is the most frequent visual complication after uncomplicated, contemporary cataract surgery. This entity was first referred to by Vogt in 1918 and in 1953 was defined by Irvine[54]. Gass and Norton demonstrated this entity on Fluorescein angiogram and was given the name of Irvine-Gass syndrome. Intact posterior capsule lessens the incidence of CME in eyes with implants compared with eyes that have had intracapsular extraction. Moses reported a lower incidence of CME in eyes after phacoemulsification or planned extracapsular cataract extraction compared with intracapsular cataract extraction[55].

Taylor et al found a 2% incidence of clinical CME in ICCE patients without IOL, 9.9% in ICCE with iris supported IOL and 1.2% in ECCE with PC IOL[56]. Kraff and coworkers found that patients 60 years of age and older had a significantly higher incidence of CME than younger individuals[57]. There is a higher incidence of CME in diabetics. It is about 32% in patients without a preoperative retinopathy and 81% in patients with preoperativeretinopathy[58]. The incidence of CME rises abruptly with

surgical complications such as operative loss of vitreous, uveitis and postoperative rupture of the anterior hyaloids membrane.

Pathogenesis:

The cause of aphakic or pseudophakic CME is still unknown. CME is thought to result from the increased permeability of perifoveal capillaries, either due to intraocular inflammation or from direct traction on the macula following vitreous shifts[11].

Some of the many proposed mechanisms include:

1. Mechanical disturbances such as vitreous incarceration in the wound, vitreous traction at the macula, hypotension, and turbulence retinopathy associated with endophthalmodonesis.
2. Inflammatory disturbances that cause incompetence of capillary walls, osmotic gradients between vitreous and serum, or primary retinal vein phlebitis.
3. Systemic factors such as hypertension or diabetes mellitus.
4. Other proposed causes, such as topical adrenergic compounds, hyaluronidase in the anesthetic solution, photic damage to the fovea from exposure to the light of the surgical microscope, inflammation and prostaglandins.

Binkhorst attributed CME to microconcussions of the retina caused by turbulences inside the eye. Worst has proposed a hypothesis attributing several biotoxic effects to aqueous humor (aqueous biotoxic complex factors). Mujake postulated that aphakic CME may occur as a result of prostaglandin synthesized intraoperatively in the iris.

Acute CME usually develops within the first three months following surgery. Chronic CME is defined as clinically significant CME persisting for more than six months. Chronic CME can cause macular degeneration, macular hole and macular

pucker. CME occurs following uneventful cataract extraction with initial good visual acuity. Visual acuity decreases 1 to 3 months after surgery. On ophthalmoscopy, there is loss of the foveal reflex and a yellowish reflex or spot that appears to lie deep in or behind the retina. Fluorescein angiography is an important diagnostic tool.

Prophylaxis: The best treatment for CME is prevention. Various studies suggest that topical prostaglandin inhibitors and perhaps systemic inhibitors are effective in the prophylaxis of CME[59]. A comparative study by Ahluwalia et al showed that, though topical indomethacin has a definite role in reducing the incidence and severity of aphakic CME, its superiority over steroids cannot be authenticated[60].

Treatment:

A trial of systemic, topical, or periocular corticosteroids and systemic prostaglandin synthesis inhibitors should be used in cases of established CME. A trend to intervene surgically exists today when vitreous incarceration is associated with CME.

Loose Sutures

Complaints of congestion, discharge and irritation of the ocular surface may indicate loose or broken sutures. These sutures should be removed to provide comfort to the patient[21].

Epithelial Downgrowth:

It is a clinically rare occurrence. Incidence of 1.1% was reported by Theobald and Hass[61]. The pathogenesis of epithelial downgrowth is not fully understood. Most instances result from faulty surgical technique with inadequate wound closure or wound healing. Early

studies stated that corneoscleal sutures, fornix based flap was probably responsible for a higher incidence of epithelial downgrowth. This has been disproved by later studies.

A high incidence is found in young patients, high myopia and diabetes. Epithelial downgrowth leads to glaucoma, bullous keratopathy. The diagnosis is confirmed by:

1. Seidel's test.
2. Photocoagulation of the surface of the iris.
3. Scraping of the back of the cornea.
4. Biopsy
5. Specular microscopy.

Treatment is usually unsuccessful. Various techniques include cryotherapy of the cornea, photocoagulation of the involved iris, surgical excision of the involved iris, ciliary body and vitreous; scraping, peeling, curettage, alcohol swabbing, irridation and deep lamellar resection.

Fibrous Ingrowth:

Fibrous ingrowth is characterized by an ingrowth of connective tissue elements into the anterior chamber. The incidence of fibrous ingrowth is comparable with epithelial down growth. Dunnington[62] observed in 32.8%, Allen[63] in 36% and Bettman61 in 34% of eyes enucleated after cataract extraction. Contemporary extracapsular surgery has significantly reduced the incidence of fibrous ingrowth.

Fibrous ingrowth is related to poor wound healing, incarceration of vitreous, iris and or lens matter in the wound, prolonged intraocular inflammation, excessive bleeding into the anterior chamber. The consequences of fibrous ingrowth include bullous keratopathy, glaucoma, retinal detachment and pthisis. Treatment is directed to a

particular consequence of the fibrous ingrowth. A fibrous membrane covering the pupillary aperture may be incised, secondary glaucoma treated by cyclodialysis or cyclocryothermy and retinal detachment treated by severing the fibroblastic bands and performing buckling procedure[12].

Intraocular Lens Complications:

Lens precipitates may be seen on the IOL post operatively. There are three types:

1. Pigment deposits arising from the pigment epithelium of the iris.
2. Grayish white precipitates that are the residues of lens material.
3. Deposits that resemble keratic precipitates.

The most frequent intraoperative complications include hyphema, vitreous injury, pupil deformation with an IOL in the anterior chamber, and hyphema and corneal lesions with an IOL in the posterior chamber. The early postoperative complications of ACIOL are striate keratopathy and uveal flare and postoperative transitory ocular hypertension and striate keratitis with a PCIOL. Late postoperative complications with ACIOL include CME, bullous keratopathy, retinal detachment, uveitis, posterior capsule opacification, eccentric displacement of the IOL, and less frequently, cystoid macular edema with PCIOL[65]. Lens precipitates tend to lessen with time.

Malpositions of Intraocular Lenses:

Malpositions of posterior chamber lenses are seen less frequently with a reported incidence of 0.4%. Capsulorrhexis ensures capsular bag fixation and better centration of a posterior chamber lens. The four kinds of malpositions associated with posterior chamber

lenses are: (1) Pupil capture; (2) Decentration; (3) Windshield wiper syndrome; (4) Sunset syndrome[12].

Pupil capture is caused by a section of the optic portion of the lens passing anterior to the iris. It may lead to an irregular pupil, limit full dilation and may cause inflammation. It occurs more frequently with planar posterior chamber lenses. Pupillary dilatation in the early postoperative period may predispose to the formation of pupillary capture. It is best managed by vigorous pupillary dilatation followed by miosis once the lens has been relocated, with the patient in the supine position. Decentration of the optic of a minor degree with position of the optic stable is of little consequence. If the decentration is such that the edge of the optic lies in the centre of the pupil, creating aphakic and pseudophakic areas, the optical disturbances may be considerable.

Windshield wiper syndrome results when the implant is too small for the eye. It is common in myopic eyes, loops in the ciliary sulcus in the vertical position. It may be corrected with a McCannel suture around the superior loop.

Sunset syndrome is usually found within first 6 weeks after surgery, resulting from an unrecognized inferior zonular dialysis during surgery. Forceful rubbing of the eye may be a cause in the late postoperative period. It may be managed by McCannel suture, creating a pupillary capture, or removal of the lens, vitrectomy and substitution with an anterior chamber lens. If the lens is dislocated into the vitreous, it is best left there because of a high risk of retinal detachment on attempting removal.

Intraocular lens membranes, more common with iris plane lens since it lies closer to vitreous face. Recurrent iritis and retained lens remnants may also cause such

membrane Corneal edema, recurrent uveitis, hyphema and glaucoma are more common with anterior chamber lenses.

Retinal Detachment:

It is the most common potentially blinding complication of cataract extraction. The incidence of retinal detachment varies from 0.66% to 3.6%[66]. The possibility of a postoperative retinal detachment is greater after ICCE than ECCE. Retinal detachment after cataract extraction is primarily due to changes in the vitreous and retina.

Risk factors:

1. Axial myopia.
2. Retinal detachment in the opposite eye
3. Operative loss of vitreous
4. Others include patients younger than 50 years of age, uveitis, open-angle glaucoma, congenital cataract surgery, Marfan syndrome, and atopic dermatitis. An intact posterior capsule may lessen the incidence of postoperative retinal detachment[12].

Prophylactic therapeutic measures, especially in the second eye are useful. Cryogenic applications may be safer for small, peripheral holes.

Postoperative Astigmatism:

Surgically induced astigmatism is an unavoidable consequence of cataract surgery. Location of the incision, suture material, type of sutures, and length and architecture of the incision influence the development and amount of post operative

astigmatism. Anterior incisions result in greater astigmatism, probably related to the delayed wound healing with anterior incisions. Non absorbable suture material (nylon) leads to with-the-rule astigmatism whereas absorbable or removable suture (chronic catgut or silk) leads to against-the-rule astigmatism.

Wound compression induces with-the-rule astigmatism. Factors that increases wound compression are:

1. Deeply inserted sutures.
2. Wide suture bites
3. Tightly tied sutures
4. Greater number of sutures.
5. Fine sutures .

Wound gape causes against-the-rule astigmatism. Causes are:

1. Large sutures that must be removed.
2. Sutures that tend to disintegrate early
3. Loose sutures
4. Wound slippage after suture removal[12].

Sutures tend to cause with-the-wound astigmatism, and both sutured and unsutured incisions move in the direction of against-the-wound astigmatism overtime. A preoperative and postoperative keratometric measurements must be taken to calculate the effect of particular technique on corneal astigmatism.

Effective method of reducing high degrees of corneal astigmatism caused by wound compression is cutting a suture in the meridian of steepest corneal curvature. It is better to

cut suture one at a time, and cut few sutures than too many, because the wound will likely slip or stretch overtime, and this is accelerated by cutting the sutures.

Optic nerve abnormalities:

The common optic nerve abnormalities responsible for decreased vision after cataract surgery are primary optic atrophy, anterior ischemic optic neuropathy or glaucomatous optic atrophy in presence of normal intraocular pressure. Postoperative anterior ischemic optic neuropathy results from a predisposing vascular insufficiency complicated by lens extraction and possibily precipitated by a change in intraocular pressure during the postoperative period[12].

MATERIALS AND METHODS:-

This is a prospective study conducted at Shri B.M.Patil Medical College Hospital and Research Centre , Bijapur. The study included patients from base camps conducted during a 18months period from November 2011 to May 2013 . During this study period 313 patients who underwent cataract surgery at our hospital were selected by simple random sampling and were studied and followed up for a period of 6 months. All the patients who underwent cataract surgery were screened at various community based screening camps [6 PHC around 60 kms from bijapur] and transferred to the base hospital.

Sample size:

Using statistical formula, n= [1.96]2 p q/L2

where n-sample size, p-proportion, q-confidence interval, L-error.

From the total number of cases coming to camp for eye check ups, we considering 60% as a proportion of cataract patients and allowing 10% error with 95% confidence interval, minimum calculated sample size was 256.

Data was analyzed using diagrams and percentage.

Inclusion criteria : -

1) Mature cataract .

2) Immature cataract.

3) Hypermature cataract.

Exclusion criteria :-

1) Traumatic cataract.

2) Cataract with diabetis mellitis.

3) Cataract with retinal diseases.

4) Congenital cataracts.

5) Complicated cataract.

6) Cataract with glaucoma.

7) Pseudoexfoliated cataract.

8) Cataract with other ocular abnormalities.

Each case was examined with detailed history regarding their complaints, the onset and duration of complaints. Preliminary examination with torch and loupe was done. Preoperative visual acuity was determined . Detailed slit lamp examination and fundus examination was done. Intraocular pressure measurement was done using Schiotz tonometer and sac syringing to test patency of lacrimal passages was done in all cases. Keratometry readings were taken , A-scan for axial length and biometry was done to determine IOL power. Routine blood investigations like RBS and urine analysis was done in all cases.

Prophylactic topical antibiotic drops (Ciprofloxacin eye drops 4 hourly) were instilled preoperatively and a single dose Tab Ciprofloxacin 500mg was given previous night . All patients underwent small incision cataract surgery (SICS) with posterior

chamber intraocular lens (PCIOL) implantation in majority (8cases SICS+ ACIOL and 1 plain SICS) under operating microscope. Retrobulbar anesthesia was given under strict aseptic condition .

All patients were operated by senior consultant . The eye to be operated was painted and draped. In all the cases superior rectus muscle, bridle suture was placed. Limbal based conjuctival incision was taken ,bleeding vessels cauterized with either wet or dry cautery. Scleral tunnel was created using crescent and a keratome, side port made at around 9'o clock position, In some cases tryphan blue was used to stain the capsule under the air bubble. Reforming the anterior chamber constantly with viscoelastic substance, first the continuous curvilinear capsulorrhexis (CCC) or capsulotomy was done , Hydroprocedures-hydrodissection and hydrodelineation, were performed through the tunnel wound. Then the nucleus was dislodged from the posterior to the anterior chamber. Using two instruments to sandwich the nucleus between them or by viscoexpression the nucleus was finally removed from the scleral tunnel. If difficulty in delivery of hard cataract the tunnel was extended ,cortex was aspirated using aspiration –irrigation method (using simmcoe cannula).

Intraoperative complication if any and their management were noted. Subconjunctival (0.5-1ml) dexamethasone and tobramycin was given at the end of the procedure. Average operating time was 10-15minutes .

Post operatively all patients received oral antibiotic (Tab. Ciprofloxacin 500mg for 5 days with Tab.Ibuprofen 400mg for 3days) and topical antibiotic steroid (oflaxacin with dexamethasone) eye drops for 6 weeks in tapering dose along with Flurbiprofen eye drops 8^{th} hourly for 3 weeks . Subsequently all of them were

followed up post operatively on day 1 , 1st week and 6th week,3rd month and 6th month. At every visit slitlamp and fundus finding were recorded, in addition to visual acuity. Astigmatism was recorded on every visit . Subjectively spectacle correction was given at the end of 6th week .

RESULTS :-

The present study was carried out for a period of 18months from November 2011 to May 2013. A total of 313 patients who were screened at community based screening camps and underwent cataract surgery were evaluated.

Table 1: Gender wise distribution

Gender	No of patients	Percentage
Female	214	68.37%
Male	99	31.63%
Total	313	100%

Female patients were 214(68.37%) of the total number of cases, while males constituted 99(31.63%).

Figure 1: Gender wise distribution of cases

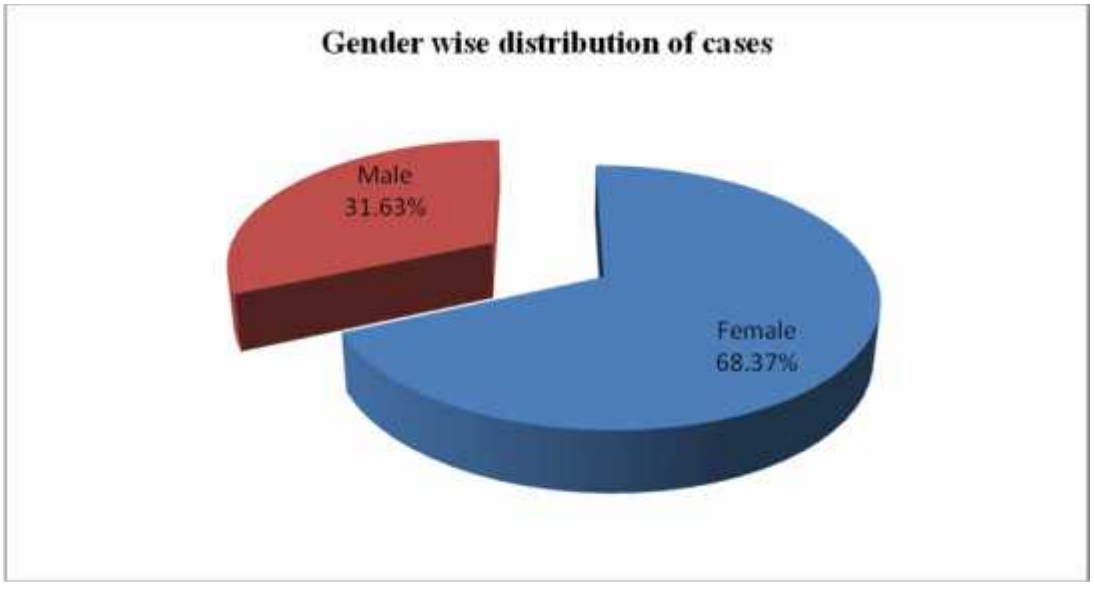


Table 2: Age wise distribution

Age	No of patients	Percentage
41-50	37	11.83%
51-60	79	25.24%
61-70	158	50.48%
71-80	38	12.14%
81-90	1	0.31%
Total	313	100%

The above tabular column shows the distribution of cataract in different age groups. Mean ± standard deviation of age was 64.02± 8.55. Maximum number of patients 158 (50.48%) were in the age group of 61-70 years. There were 79 patients (25.24%) in the 51-60 years age group. 38 patients (12.14%) in the 71-80 years age group, 37 (11.83%) patients in the 41-50 years age group and 1 patients (0.31%) in the 81-90 years age group.

Figure 2: Age wise distribution of cases

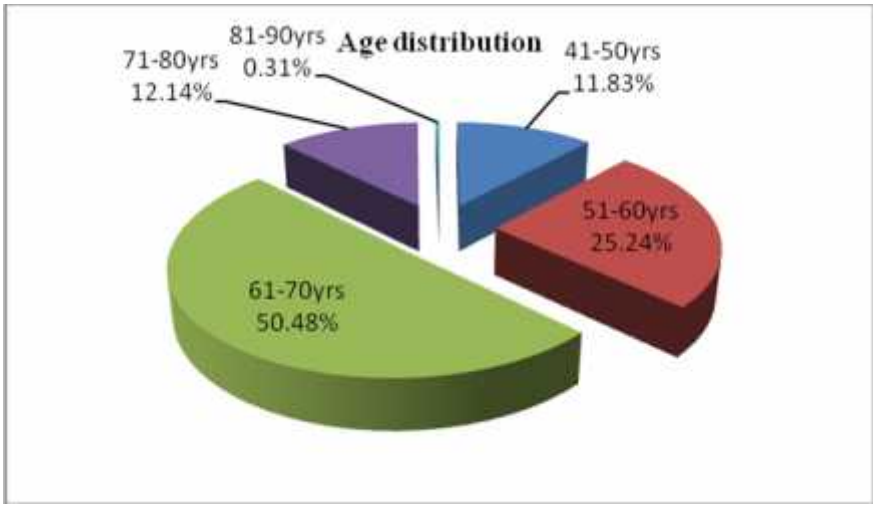


Table 3: Type of Cataract

Type	No of cases	Percentage
Immature	206	65.82%
Mature	106	33.87%
Hypermature	1	0.31%
Total	313	100%

Out of 313 cases operated, 206 (65.82%) were immature cataract, while 106 (33.87%) were mature and 1 (0.31%) was hypermature.

Figure 3:Type of cataract

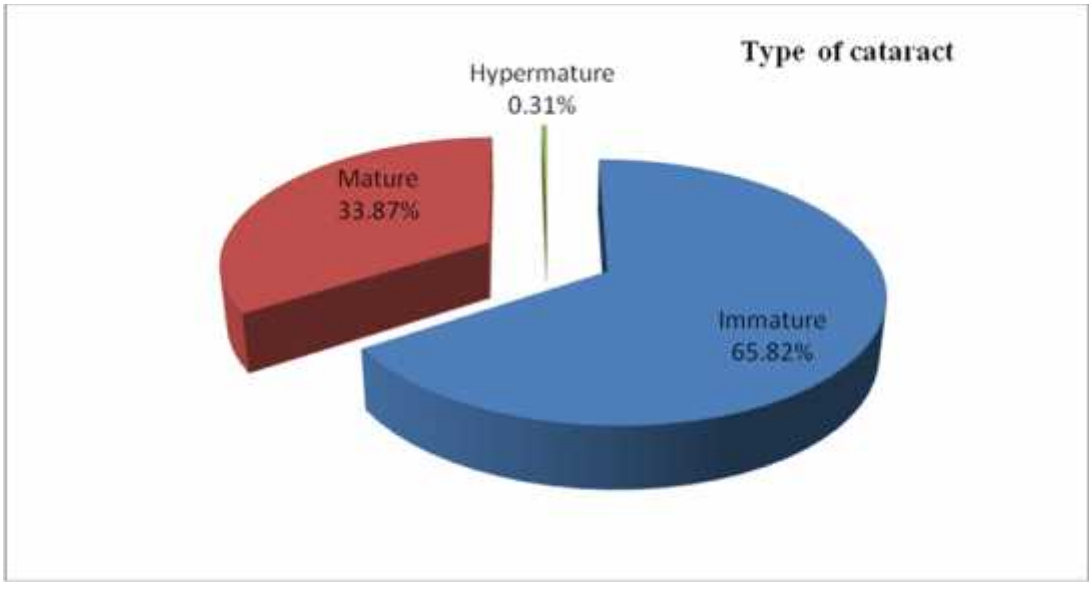


Table 4: **Distribution of type of immature cataract**

Type of SIMC	No of patients	Percentage
PSC	26	8.30%
PSC+Cortical	99	31.62%
PSC+NS	5	1.59%
Cortical	21	6.70%
NS	32	10.22%
NS+Cortical	12	3.83%
NS+PSC+Cortical	12	3.83%
Total	206	66.1%

Out of 206 immature cataract cases 99 cases (31.62%)were posterior subcapsular with cortical cataract, 32 cases (10.22%) were nuclear sclerosis grade 1-3, 26 cases(8.30%) were posterior subcapsular type, 21 cases (6.70%) were cortical type, 12 cases each (3.83%)were nuclear sclerosis with cortical type and nuclear sclerosis with cortical with posterior subcapsular type and 5 cases(1.59%) were posterior subcapsular with nuclear sclerosis type.

Figure 4: Distribution of type of immature cataract

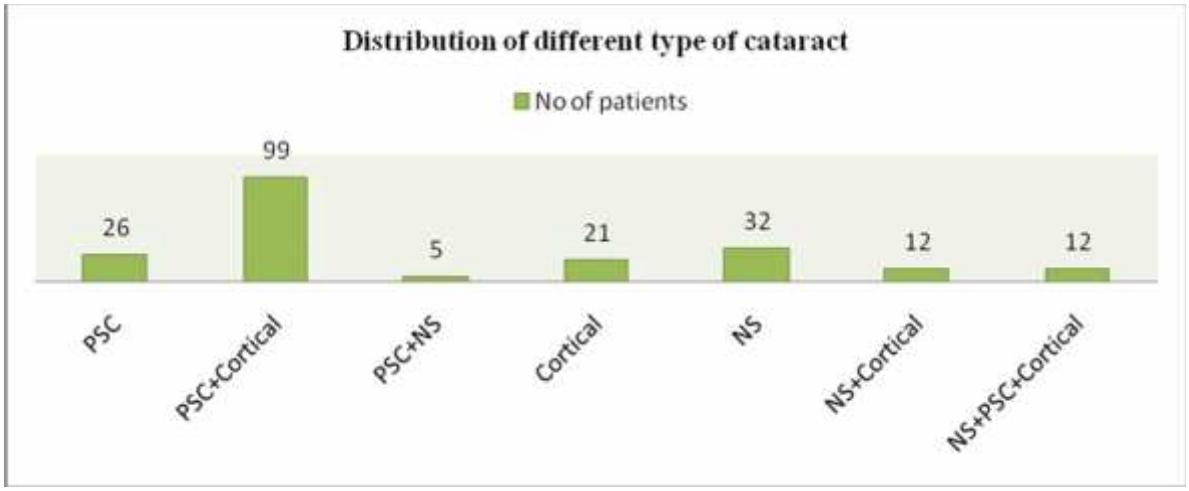


Table 5: Distribution of patients and preoperative uncorrected visual acuity

Visual acuity	No of cases	Percentage
PL+ PR +	68	21.72%
PL+	4	1.28%
HM	51	16.30%
CF CTF	21	6.70%
CF 1/2M	32	10.22%
CF 1M	62	19.80%
CF 2M	35	11.20%
CF 3M	28	8.95%
6/60	10	3.19%
6/36	2	0.64%
Total	313	100%

Out of 313 patients, preoperative visual acuity in 301 patients (96.17%) was counting fingers less than 3 meters . 10 patients (3.19%) had preoperative visual acuity of 6/60 and 2 patients (0.64%) had pre operative visual acuity 6/36.

Figure 5: Distribution of patients and preoperative uncorrected visual acuity

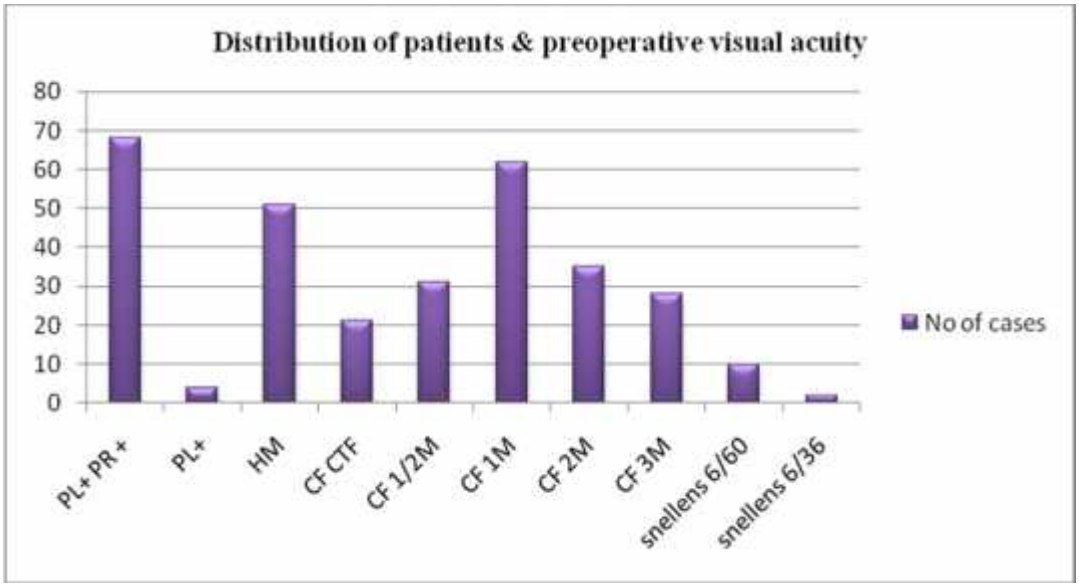


Table 6: Types of surgery performed

Procedure	No of patients	Percentage
SICS+PCIOL	304	97.13%
SICS+ACIOL	8	2.56%
SICS	1	0.31%
Total	313	100%

The above tabular column shows that SICS with PC-IOL implantation was performed in 304 cases (97.13%), SICS with AC-IOL implantation in 8 (2.56%), plain SICS without intraocular in 1 case (0.31%).

Figure 6: Types of surgery performed

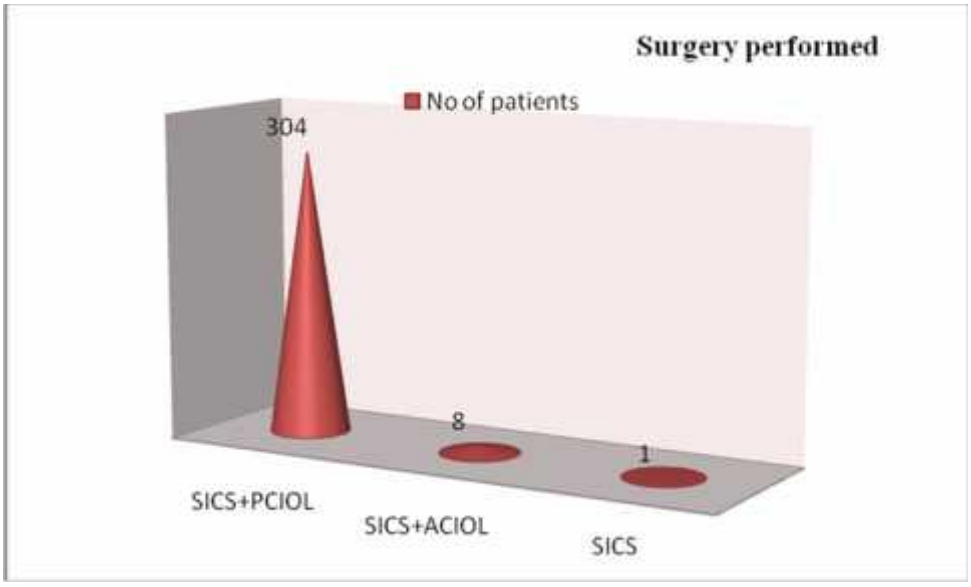


Table 7: Intraoperative complications

Complication	No of patients	Percentage
None	288	92.01%
Posterior capsule rupture	10	3.20%
Posterior capsule rupture with vitreous leak	5	1.60%
Premature entry	2	0.64%
Premature entry+posterior capsule rupture+iridodialysis	1	0.31%
Iris tear	2	0.64%
Iridodialysis	2	0.64%
Descemets membrane detachment	3	0.96%
Total	313	100%

Of the 313 patients, surgery was uneventful in 288(92.01%). Patients intraoperative complications were seen in 25 patients (7.99%). Posterior capsule rupture without vitreous leak occurred in 10 cases (3.20%), posterior capsule rupture with vitreous leak occurred in 5 patients (1.60%). The other intraoperative complications in descending order are Descemet's membrane detachment in 3 cases (0.96%), iridodialysis in 2 cases (0.64%), premature entry in 2 cases (0.64%) and iris tear in 2 cases(0.64%) and premature entry with posterior capsule rupture with iridodialysis in 1 case (0.31%).

Figure 9: Intraoperative complications

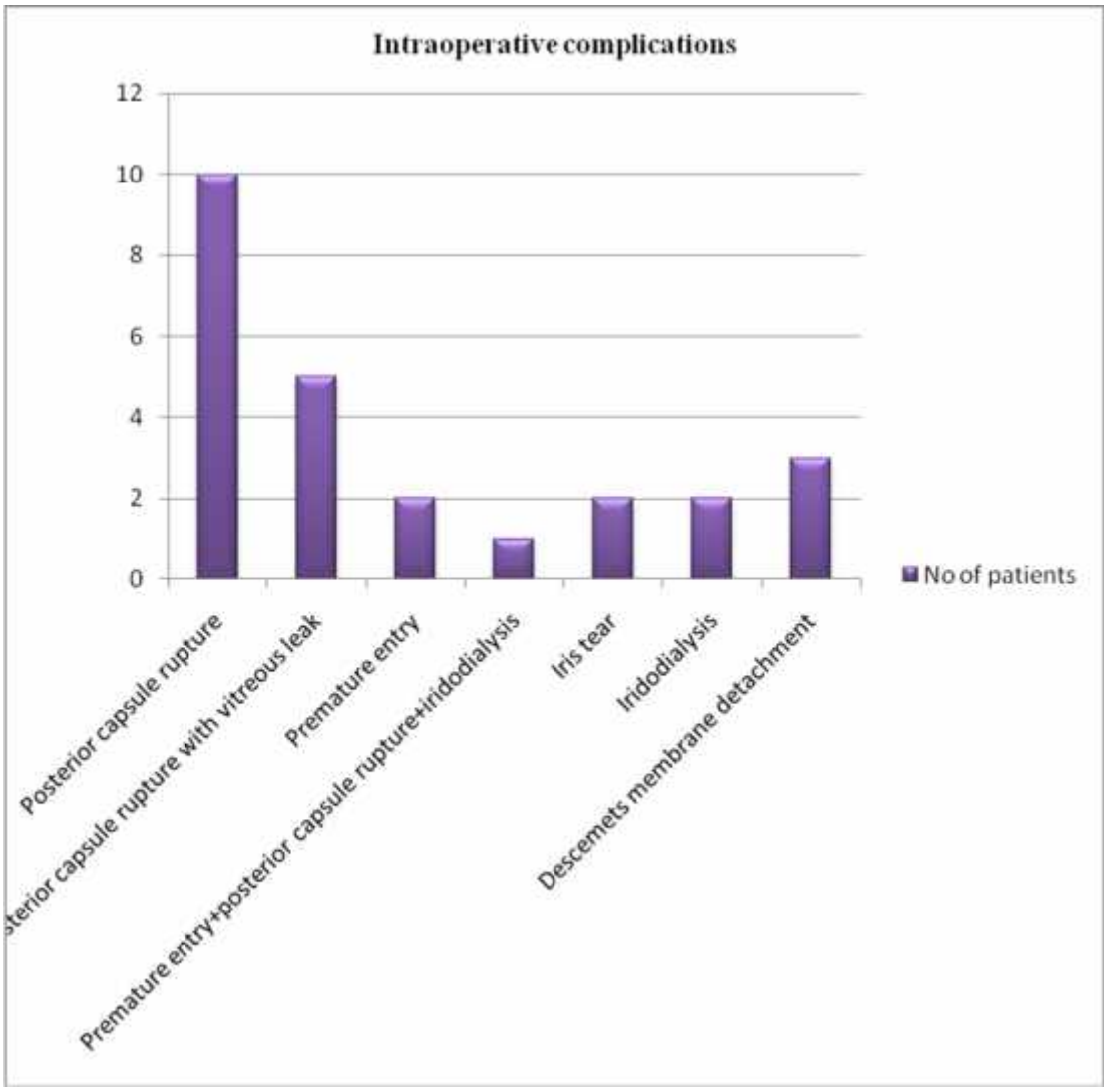


Table 8: Immediate post operative complications(Day 1)

Complications	No of cases	Percentage
Corneal edema(primary)	39	12.46%
Striate keratitis	28	8.95%
Uveitis	23	7.04%
Secondary glaucoma+ corneal edema	11	3.52%
Residual cortex	11	3.52%
Pre exixting posterior capsular opacification	3	0.97%
Iris prolapse	2	0.64%
Shallow anterior chamber	1	0.31%
Hyphema	1	0.31%
Endophthalmitis (sterile)	1	0.31%
Iris incarceration	1	0.31%
None	193	61.66%
Total	313	100%

Of the 313 patients, surgery was uneventful in 179 patients. Complications were seen in 120 (38.34%) cases on the first postoperative day. Primary corneal edema was the most common complication seen in 39 cases (12.46%). Striate keratitis was the second most common complication seen in 28 (8.95%) patients.

Other complications in the descending order of frequency were uveitis in 23 cases (7.04%), secondary glaucoma with corneal edema and residual cortex in 11 cases each(3.52%), pre existing posterior capsular opacification in 3 cases (0.97%) each ,iris prolapsed in 2 cases (0.64%),shallow anterior chamber, hyphema, sterile endophthalmitis and iris incarceration in 1 case(0.31%) each.

Figure 8: Immediate post operative complications (day 1)

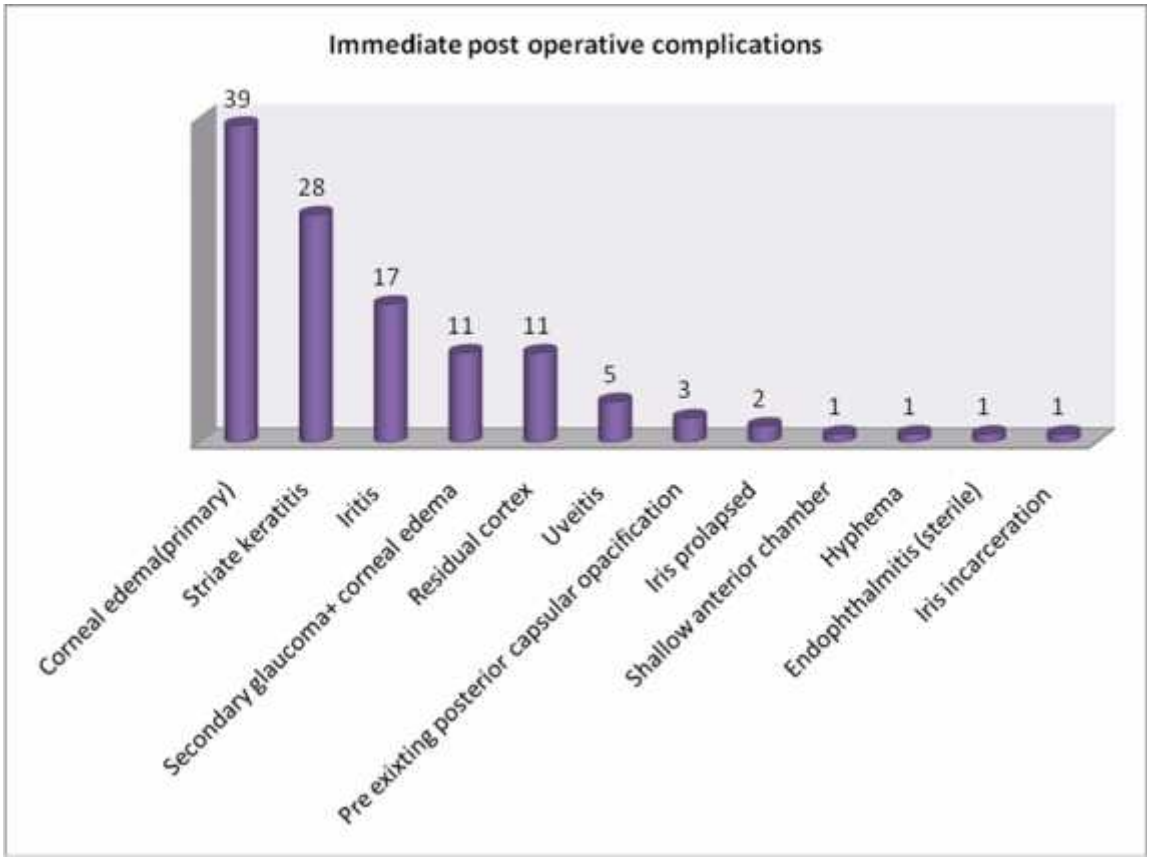


Table 9: Postoperative complication at the end of first week

Complication	No of patients	Percentage
Persistent uveitis	12	3.84%
Corneal edema	10	3.20%
Striate keratitis	6	1.93%
Pre existing PCO	5	1.60%
Secondary glaucoma	5	1.60%
Residual cortex	1	0.31%
Displaced IOL	1	0.31%
Optic capture	1	0.31%
None	272	86.90%
Total	313	100%

At the end of first week, complications were seen in 41 (13.10%) cases. In decreasing order persistent uveitis in 12 cases(3.84%), corneal edema in10 cases (3.20%) , striate keratitis in 6 (1.93%), secondary glaucoma and preexisting posterior capsular opacification in 5 (1.60%) case each and residual cortex ,displaced IOL and optic capture each in 1 (0.31%) case were noted.

Figure 9: Postoperative complication at the end of first week

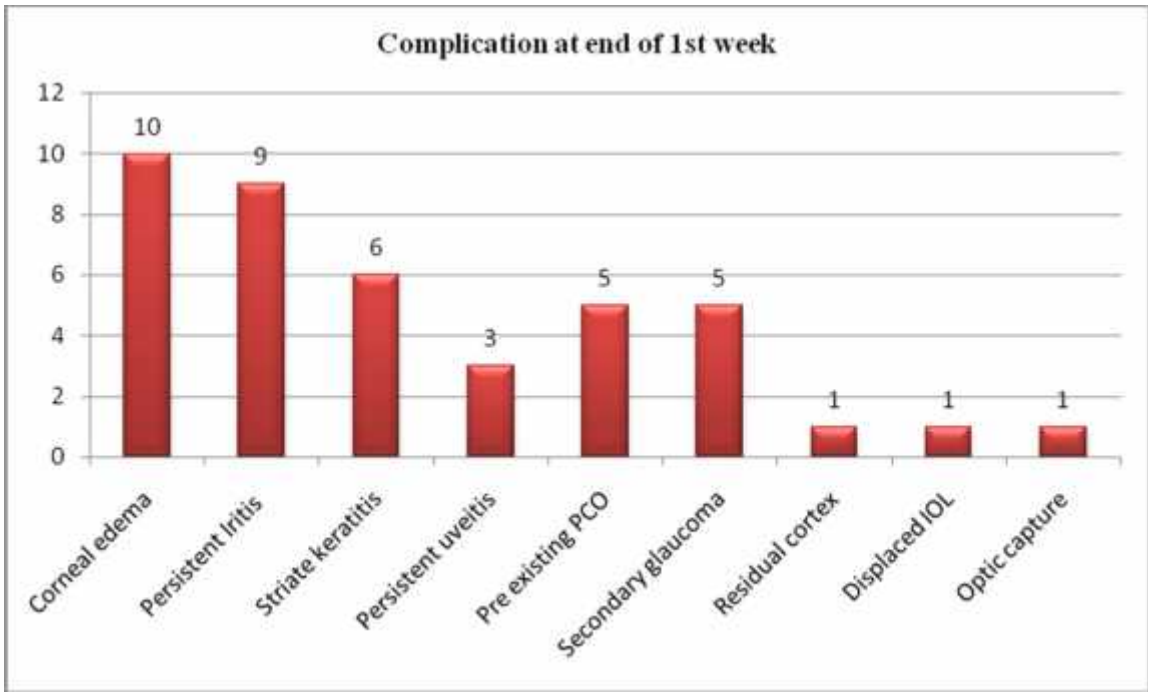


Table 10: Postoperative complication at the end of 6weeks

Complication	No of patients	Percentage
PCO	9	2.88%
CME	1	0.31%
Optic capture	1	0.31%
None	302	96.50%
Total	313	100%

At the end of 6 weeks, 9 patients (2.88%) had PCO, 1 case (0.31%) each had cystoid macular edema and optic capture .

Figure 10: Postoperative complication at the end of 6weeks

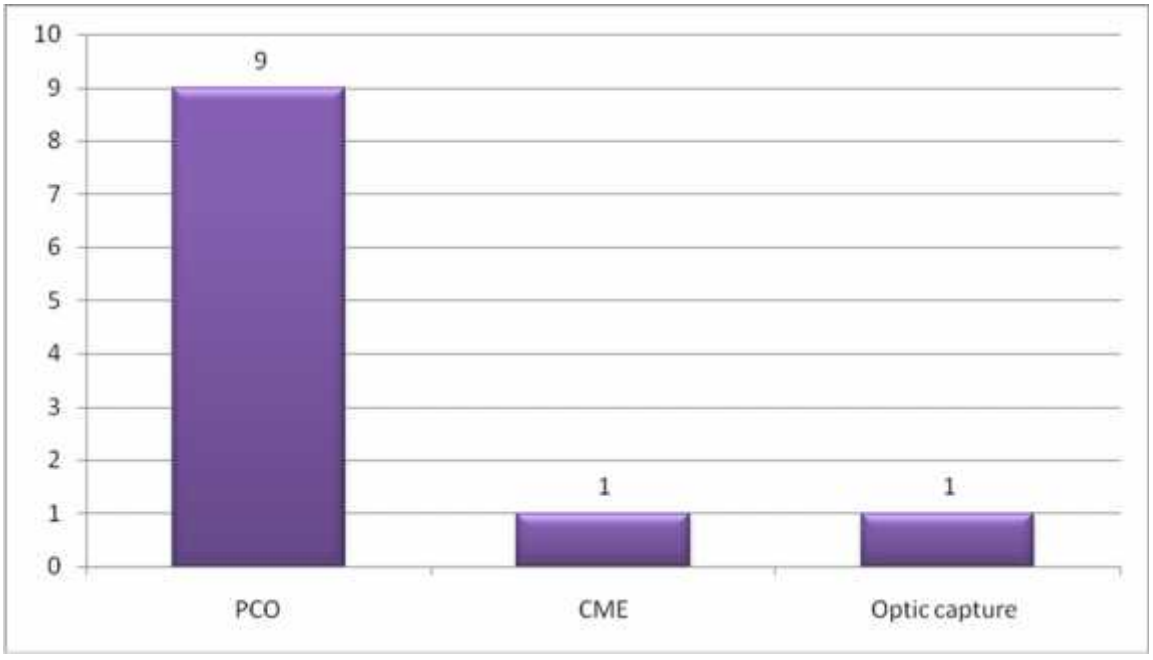


Table 11: Postoperative complication at the end of third month

Complication	No of patients	Percentage
PCO	8	2.55%
None	305	97.45%
Total	313	100%

At the end of third month, 8 patients (2.55%) had developed PCO. Overall PCO patients in our study were 17 cases (5.42%).

Figure 11: Postoperative complication at the end of third month

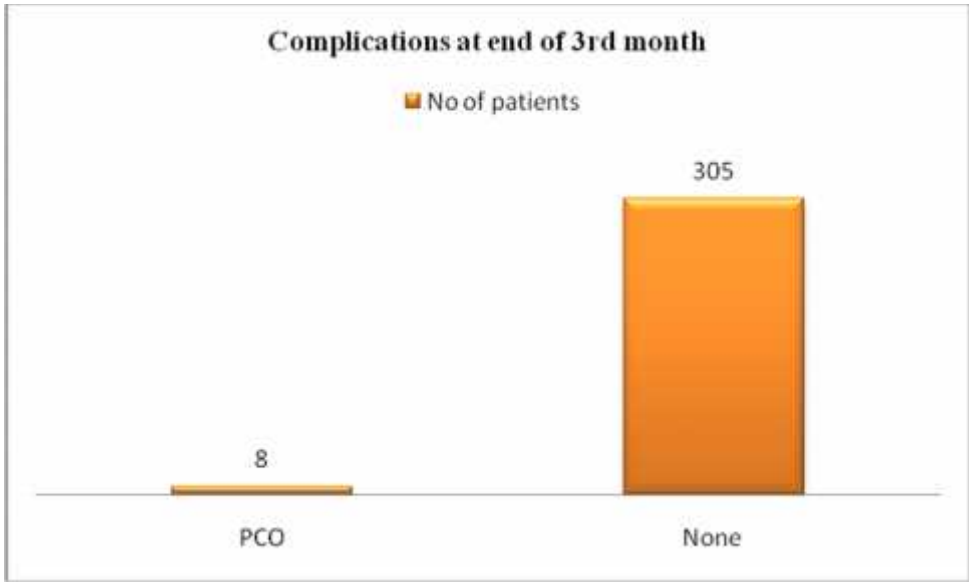


Table 12: Postoperative complication at end of 6 months

Complication	No of patients	Percentage
Retinal detactment	2	0.63%
None	311	99.37%
Total	313	100%

At the end of 6 months, 2 cases (0.63%) had retinal detachment.

Figure 12: Postoperative complication at the end of 6 months

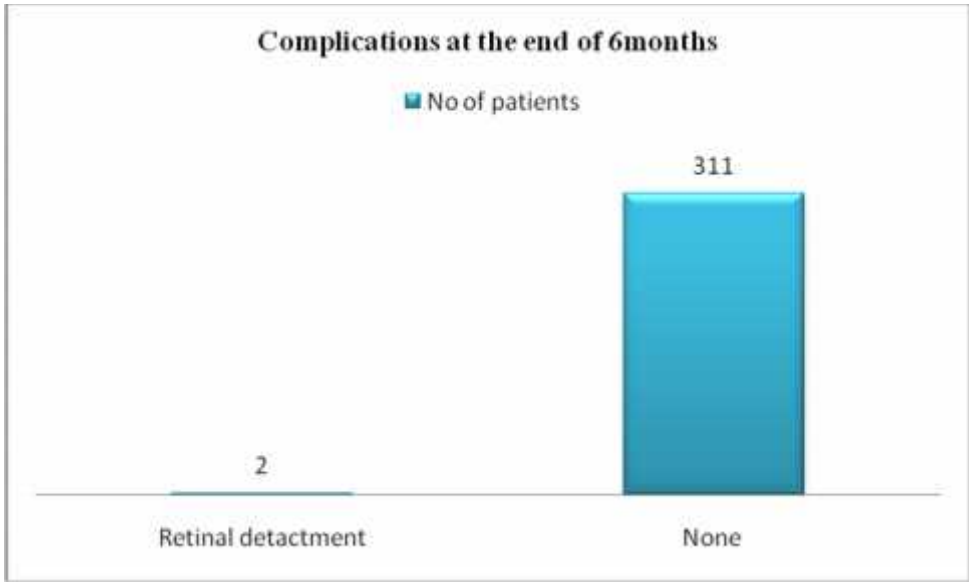
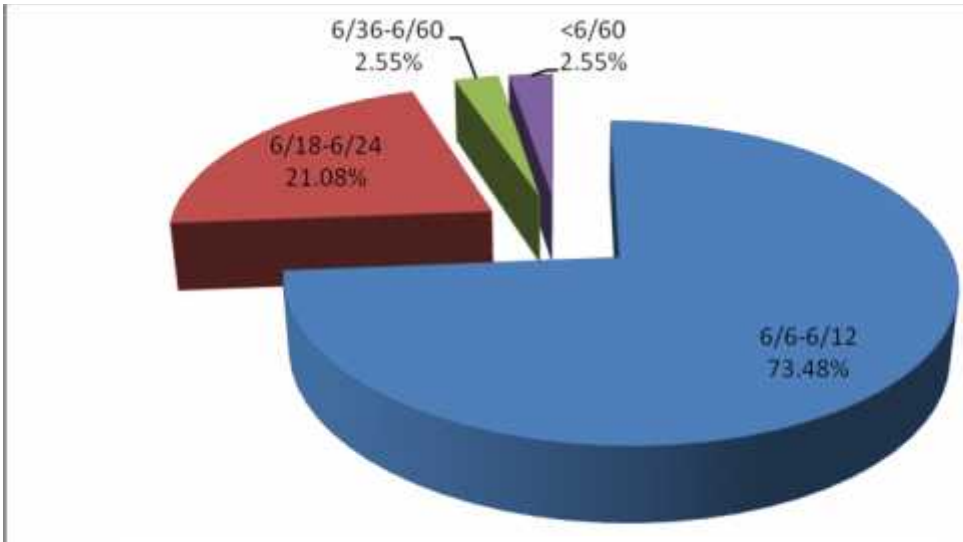


Table 13: Distribution of patients with postoperative best corrected visual acuity (BCVA)at end of 6weeks.

Visual acuity	BCVA	
	No of cases	Percentage
6/6-6/18	261	83.39%
<6/18-6/60	42	13.42%
<6/60	10	3.19%
Total	313	100%

Out of 313 patients ,261 patients (83.39%) had BCVA between 6/6-6/18, 42 patients(13.42%) had BCVA between 6/18-6/60, 10 patients(3.19%) had BCVA less than 6/60.

Figure 13: Distribution of patients with postoperative best corrected visual acuity (BCVA)at the end of 6weeks.



Postoperative BCVA at the end of 6weeks.

Table 14: Post operative astigmatism at the end of 6 weeks

Astigmatism	No of patients	Percentage
0.25-1D	98	31.31%
1.25-2D	144	46.01%
2.25-3D	46	14.70%
3.25-4D	9	2.87%
4.25-5D	2	0.64%
No astigmatism	2	0.64%
Poor vision	12	3.83%
Total	313	100%

Among 313 cases astigmatism was present in majority of patients. Post operatively in decreasing order 144 cases(46.01%) with astigmatism between 1.25 to 2 diopters, 98 cases(31.31%) with astigmatism between 0.25-1diopters ,46 cases (14.70%) with astigmatism between 2.25-3diopter, 9 cases (2.87%) had astigmatism between 3.25-4diopters, 2 cases (0.64%)with astigmatism between 4.25-4 diopters. 2 cases(0.64%) had no astigmatism . Remaining 12 cases (3.83%) had poor vision.

Figure 14: Post operative astigmatism at the end of 6 weeks

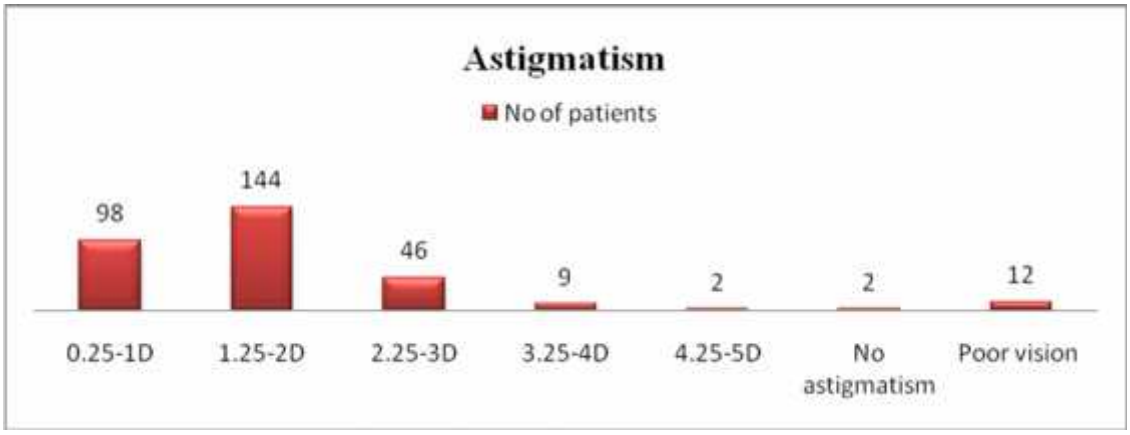


Table 15: Type of astigmatism at the end of 6 weeks

	No of patients	Percentage
WTR	12	4.79%
ATR	213	68.05%
Oblique	74	27.15%
NA	2	1.28%

With the rule astigmatism were 12 cases(4.79%) and against the rule astigmatism were 213 cases(68.05%). This signifies ATR astigmatism is more common in MSICS.

Figure 15: Type of astigmatism at the end of 6 weeks

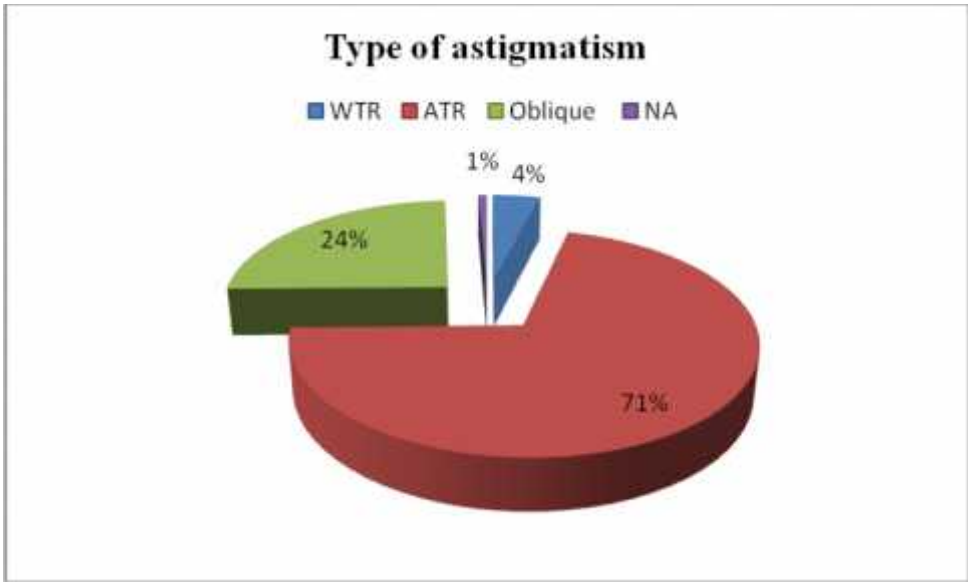


Table 16: Association of type of cataract and intraoperative complications

Intra operative Complications	Type of cataract					
	Mature	**Hypermature**	**PSC**	**Cortical**	**Nuclear**	**Combined**
PCR	2	-	-	-	3	6
PCR+VL	2	-	-	-	1	2
Tunnel problem	1	-	-	-	-	2
Iris tear	2	-	-	-	-	1
Iridodialysis	2	-	-	-	-	-
DMD	2	-	-	-	-	1
None	95	1	26	21	28	116

The above table shows that most intra complications were associated with mature type (3.51%) and combined type of immature cataract (3.83%).

Table 17: Relationship of BCVA to the intra operative complications

Intra operative Complications	**Visual acuity**		
	6/6-6/18	**<6/18-6/60**	**Less than 6/60**
PCR	7	4	-
PCR+VL	2	-	3
Tunnel problem	1	2	-
Iris tear	2	-	-
Iridodialysis	2	-	1
DMD	1	-	2
None	247	36	5

Three patients with posterior capsular rupture with vitreous leak, 2 patients with Descemets membrane detachment and one patient with iridodialysis had BCVA less than 6/60. 4.79% patients had good vision inspite of intraoperative complications.

Table 18 :Relationship of post operative complication and age.

Post operative Complications	Age(years) of the patient				
	41-50	**51-60**	**61-70**	**71-80**	**>80**
Corneal edema	2	9	24	5	-
Striate keratitis	-	3	19	3	-
Iritis	-	6	8	2	-
Secondary glaucoma	-	1	7	2	-
Residual cortex	-	2	5	2	-
Uveitis	-	2	1	-	-
PPCO	1	-	1	-	-
Iris prolapse	-	-	4	-	-
Shallow AC	-	-	-	1	-
Hyphema	-	1	-	-	-
Sterile Endoph	-	-	1	-	-
Iris incareation	-	-	-	-	-
Persistent iritis	-	-	-	-	-
Persistent uveitis	-	-	2	1	-
Displaced IOL	-	1	1	-	-
Optic capture	-	-	2	-	-
PCO	-	2	7	1	-
CME	-	-	1	-	-
RD	-	1	1	-	-
None	14	52	75	10	1

Most post operative complications (26.83%) were in the age group between 61-70years of age .

Table 19: Relationship of type of cataract and postoperative complication.

Post operative Complications	Type of cataract					
	Mature	**HMC**	**PSC**	**Cortical**	**NS**	**Combined**
Corneal edema	16	-	2	3	8	18
Striate keratitis	9	-	2	4	3	10
Iritis	6	1			3	9
Secondary glaucoma	2	-	1	-	3	5
Residual cortex	4	-	-	-	2	5
Uveitis	4	-	-	-	2	2
PPCO	-	-	-	1	-	2
Iris prolapse	1	-	-	-	-	2
Shallow AC	1	-	-	-	-	-
Hyphema	-	-	1	-	-	-
Sterile Endoph	1	-	-	-	-	-
Iris incareation	1	-	-	-	-	-
Persistent iritis	5	-	-	-	1	1
Persistent uveitis	1	-	-	-	-	3
Displaced IOL	1	-	-	-	-	1
Optic capture	1	-	-	-	1	-
PCO	2	-	-	3	3	7
CME	-	-	-	-	-	1
RD	-	-	-	-	-	2
None	51	-	20	10	6	61

Most post operative complications were present in combined type of immature cataract (21.72%) and mature type (17.57%) followed by nuclear type (8.30%) and others .

Table 20 : Causes of good , borderline & poor visual outcome (presenting vision) after cataract surgery.

Principle causes of reduced vision	**Good outcome** (6/6-6/18)	**Borderline outcome** (<6/18-6/60)	**Poor outcome** (<6/60)
Related to cataract surgery			
Pertaining to anterior segment			
Corneal opacity	-	-	1
DMD	-	-	2
PCO	6	1	2
Aphakia	-	-	2
Displaced IOL	-	-	1
Optic capture	1	-	-
Pertaining to posterior segment			
Cytoid macular edema	-	1	-
Retinal detachment	-	1	2
Unrelated to cataract surgery			
Pertaining to anterior segment			
Polar cataract	-	1	-
Pertaining to posterior segment			
Age related macular degeneration	3	10	2
Optic atrophy	-	1	1
Myopic degeneration (amblyopia)	2	1	-
Old BRVO	-	1	-
Old CNVM	-	-	1
Posterior staphyloma	-	-	1

The above table gives an overview of poor vision . Principle cause of poor visual outcome pertaining to anterior segment were 2 cases each of DMD, aphakia and PCO . One case of each with displaced IOL and corneal opacity.Causes pertaining to posterior segment are 2 cases of retinal detachment.

Causes of poor outcome unrelated to cataract outcome are 2 cases of dry ARMD, 1case each of optic atrophy, old CNVM and posterior staphyloma .

DISCUSSION

Cataract surgery continues to be evolutionary. Dealing with such a growing burden of cataract blindness requires a cheaper and faster method of surgery that also guarantees good post operative visual acuity. Modern cataract surgery has evolved from a simple operation involving Graefe knife and no lens replacement to a refractive surgical procedure capable of improving both uncorrected and best corrected visual acuity. With the introduction of the intraocular lens and consequent improvement in prediction of IOL power, the spherical component of patients refractive error has become reasonably predictable.

Phacoemulsification may not be an affordable technique due to cost involved in the developing countries. Manual small incision cataract surgery (MSICS) has evolved as an effective alternative to phacoemulsification in present times. Recent studies have shown that manual SICS is cost effective and has more benefits as compared to conventional ECCE[67].

MSICS has become popular in India in the last decade. Quality of vision and early rehabilitation are two of the critical parameters that determine the success of modern cataract surgery.The astigmatic component of refractive error following cataract surgery remains the greatest obstacle to achievement of this goal. By 2020, cataract surgical coverage in India is predicted to increase to 7.63 million per year (Murthy et al, 2008). This demonstrates significant progress in addressing surgical aspects; however, visual recovery after surgery is poor in about 25 % of cases (Limburg et al, 1999a; Anand et al, 2000; Dandona et al, 1999).

This study has been undertaken with the principle aim to find out the incidence of complications of cataract surgery in base camps – indirectly pointing to the efficiency of such camps. Totally 313 patients who were screened at community based screening camps and underwent small incision cataract surgery were studied and evaluated for postoperative complications.

Gender :

In our study females constituted the majority (68.37%) of the patients as compared to males (31.63%) .

Age of presentation:

The age at presentation ranged from 41-82 years. Majority of the patients (50.48%) were in the age group of 61-70 years. The incidence was less in the age group of 81-90 years (0.31%). Age is the most important risk factor for developing a cataract,[91,92] but increasing age is also known to influence the visual acuity achieved after cataract surgery.[93] Natural aging and delayed surgery negatively impact postoperative visual acuity as well as surgical outcomes in elderly patients.[94,95]

Type of cataract :

In our study, senile immature cataract cases (65.82%) were more than mature (33.87%) and hypermature (0.31%)types. Among immature cataracts ,majority were posterior subcapsular with cortical type (31.62%), followed by 10.22% nuclear sclerosis, 8.30% were posterior subcapsular type,6.70% were cortical , 3.83% each were nuclear with cortical and nuclear plus cortical with posterior subcapsular type and 1.59% were nuclear with posterior subcapsular type.

Pre operative visual acuity :

Pre operative visual acuity in 96.17% patients was counting fingers less than 3 meters and 3.85% had visual acuity between 6/36-6/60.

High rate of illiteracy in elderly population and lack of awareness about undergoing cataract surgeries on time could be responsible for late presentation. Patients ignorance and economic status was the reason which did not allow them to meet ophthalmologist at the earliest as all of them were from rural population.. Hence, majority of the patients in our study were legally blind (visual acuity less than 6/60) preoperatively.

Operative procedure :

Manual small incision cataract surgery with PC-IOL implantation was the procedure of choice. Majority of patients (97.13%) underwent posterior chamber intraocular lens implantation , ACIOL was implanted in 2.56%,plain SICS was done in 0.31%.

Intra operative complication :

The operative complication rates were low. Of the 313 patients only 7.99% had intraoperative complications. Venkatesh et al[67] in 2003 reported an incidence of 1.9% of intraoperative complications.

Posterior capsular rupture without vitreous leak:

The incidence of posterior capsule rupture without vitreous leak was the commonest intra operative complication in our study(3.20%) and this was similar incidence to that reported in other series (1.0% to 4.3%)[68,69]. Chitkara et al[16] in 1991

reported an incidence of 4% and is comparable with our study results. Posterior capsule rupture occurred most commonly during nucleus delivery and irrigation aspiration.

Early detection of posterior capsule rupture on theoperating table and withdrawal of irrigation and aspiration prevented vitreous loss. Of the 10 cases, in 5 cases ACIOL was implanted were rent was inferior but difficult to place PCIOL.In few cases PCIOL implantation was done in sulcus as the posterior capsule rupture was small. Oshner and Cionni et al[73] reported a similar incidence of 1% in their study.

Posterior dislocation of IOL into the vitreous was seen in 1 case (0.3%), which was advised referral to vitreoretinal surgeon for explantation .

Posterior capsular rupture with vitreous leak:

Vitreous loss following posterior capsular rent was the second commonest complication occurring in 5 cases (1.60%). In hospital based study of 898 patients, Natchiar et al[70]reported a similar incidence of vitreous loss (1.7%).

Table 21 : Vitreous loss in base hospitals Study

Incidence of vitreous loss	**Percentage**
Gogate et al[23]	3.30%
Ravindra et al[71]	2.66%
NCS[68]	1.00%
Venkatesh et al[647]	0.70%
Hennig et al[29]	0.20%
Present study	1.59%

The above tabular column highlights the incidence of posterior capsule rupture with vitreous loss in base hospital surgeries.

Descemet's membrane detachment

Descemet's membrane detachment was seen in 3 cases (0.95%). This occurred probably due to inadvertent entry of the instruments (26 Gauge Cystitome) into AC. Detachment was small in 1case and sufficiently large in 2 cases and placement of air bubble facilitated its apposition in two cases, but one patient had potential vision loss . According to study of Rosenthal [77] incidence of Descemet's membrance detachment is 4%. In 1964 Sugar [78] conducted a survey & found incidence of Descemet's detachment as 11%.

Table 22: Incidence of Descemets membrane detachment in various studies.

Studies	Percentage
Rosenthal[77] (1988)	4%
Sugar [78](1964)	11%
Sparks [79](1967)	5.43%
Our study	0.95%

Iridodialysis:

Iridodialysis was noted in 2 cases (0.94%) one was small and did not have any significance in the final visual outcome. 1 case had poor visual outcome. Premature entry was seen in 3 cases (0.95%). Suturing of the wound was done. Iris trauma (tear) was present in 2 cases(0.63%).

Immediate post operative complications

Of the 313 patients, 193 patients (61.66%) did not have any complications on the first postoperative day. Immediate postoperative complications were noted in 120 cases (38.34%).

Primary corneal edema :

The most common immediate postoperative complication was primary corneal edema 12.46%. The oedema varied in severity from mild to severe, with a corresponding effect on the visual acuity .

In MSICS, the prolapse of nucleus into the anterior chamber and its delivery through the tunnel involve manipulations very close to the iris and the cornea.where sandwich technique was used to deliver the nucleus, so postoperative inflammation and corneal edema was too common.Trauma to the corneal endothelium by instruments, lens fragments, forcible flow of irrigation fluids,retained viscoelastic substance or IOL insertion may be the cause of the oedema.

Patients complained of hazy or misty vision, “like looking through ground glass”, but generally experience no or only mild discomfort. However, in the majority of cases, even marked corneal oedema resolved within the first week. Most studies of MSICS report a transient corneal edema, which clears off by the first week[80,81,82,83,84,85].The trial in Pune had nine (4.5%) cases of postoperative corneal edema on the first day in the phaco arm and four (2%) cases in the MSICS arm[80].

Striate keratitis:

Striate keratitis (8.95%) was the second most common complication during early post operative day . Sudhakar et al[17] in their study found a similar incidence (7.3%) of striate keratitis. Striate keratitis is common during MSICS if enough care is not taken to place the viscoelastic between the nucleus and the cornea. In the various series of cataract surgeries conducted in rural camps by Anand et al[25] and Verma et al[22] corneal edema constituted the major complication accounting for 23.3% and 43% respectively,striate keratitis in 23 cases (7.7%), which cleared within 3 days. The reason could be due to excessive manipulation during surgery like prolonged cortical wash.

Secondary corneal edema:

Secondary corneal edema due to transient rise in intraocular pressure was noted in 3.52%. Intervention was required in approximately 1.91% of patients. Troublesome raised IOP post-operatively was minimised by a prophylactic course of oral acetazolamide 250mg and topical timolol maleate eye drops within 3 days in 6 cases. Raised IOP was probably due to retained viscoelastic substances in the anterior chamber at the time of surgery, residual cortex or because of inadequate wash. Sudhakar et al[17] and Venkatesh et al[67] reported an incidence of 1.7% and 1.5% respectively. Hennig et al[29] reported an incidence of 0.4% in his study.

Postoperative uveitis:

Postoperative uveitis was seen in 7.04%, which was mild in 5.44% and moderate in 1.60%. It was treated with topical and systemic steroids depending on its severity. The probable cause of iritis was surgical trauma. Sudhakar et al[17] reported an incidence of

4.8%. Kratz et al[74] found an incidence of 3.3%. Venkatesh et al[67] reported an incidence of 1.5%. The MSICS involves touching the iris at some point of time. This may lead to higher incidence of postoperative iritis and cystoid macular edema[86,88].

Nevertheless, the studies so far have not shown any difference or increase in these complications[80,87,89]. The series from south India had mild iritis in 6% and moderate iritis in 3% in the first postoperative week[88].

Residual cortex:

Residual cortex was seen in 3.52% cases. Though this was higher than obtained by Venkatesh et al[67] (0.2%), it did not have effect on final visual outcome and none of the cases required resurgery.

Iris prolapse:

Iris prolapse was noted in 0.64% cases. Iris abscission with suturing was done in both of these cases The incidence of iris prolapse was 1.3% and 0.5% in the various studies by Chitkara et al[16] and Balmer et al[15].

Shallow anterior chamber:

Shallow anterior chamber was noted in 0.31% case. Shallow anterior chamber was secondary to wound leak . Anterior chamber was reformed and suturing was done. Ravindra et al[71] reported an incidence of 0.4% of wound gape.

Hyphema:

The other complications included hyphema (0.31%) which got cleared in one week with oral & topical steroids.Venkatesh et al[67] reported an incidence of 0.2% of

hyphema. Das et al[14] in his study reported a higher incidence of hyphema (5.31%) in patients operated in rural camps as compared to patients operated in hospitals (1.06%).

Iris incarceration:

Iris incarceration was present in 0.31%, which was managed with pilocarpine eye drops and did not have significance in the final visual outcome .

Endophthalmitis:

Sterile endophthalmitis (0.31%) was present in 1 case and was treated with only topical steroids hourly which resolved in 20 days. PCIOL was displaced in vitreous and so patient was given aphakic glasses. Postoperative endophthalmitis is a rare but dreaded complication of cataract surgery, with a reported incidence currently in the range of 0.04% to 0.41%.[99,100]. Sudhakar et al[17] encountered an incidence of 0.5% of endophthalmitis in his study. Chitkara et al[16] reported an incidence of 0.2%. A higher incidence was reported by Anand[25] (1.4%) and Verma et al[22] (4.3%) in surgeries conducted in rural camps.

Follow up was done at an interval of one week, 4-6 weeks, 3 month and 6 months.

Postoperative complication at end of first week

At the end of first week, complications were seen in 13.10% cases.

Corneal edema

Corneal edema was present in 3.20% cases,

Persistent uveitis

Persistent uveitis was seen in 3.84%. It was treated with tapering dose of topical and systemic steroids.Stark et al[75] documented a 0.4% to 1.2% incidence of persistent uveitis with various IOL types over a 12 month period. Sudhakar et al[17] reported an incidence of 1.4% of persistent uveitis.Straite keratitis was present in 1.93%.

Pre existing PCO and secondary glaucoma

Pre existing PCO and secondary glaucoma were present in 1.60% each. One case each (0.31%) was displaced IOL,optic capture and residual cortex.

Post operative complications at end of 6weeks

Posterior capsule opacification

At the end of 6 weeks fibrous type of posterior capsule opacification was seen in 2.88% cases.

The cause of early posterior capsule opacification in our study could be due to capsular tag remnants, residual cortex, few immature fibres sticking to posterior capsule and uveitis in the postoperative period. Sudhakar et al[17] reported a higher incidence (11.5%). McDonnell et al[76] reported that up to 50% of adults develop posterior capsule opacification within 3 to 5 years following extracapsular cataract extraction. A higher incidence of 31% was reported by Balmer et al[15]. A large systematic review of posterior capsular opacification (PCO) rates in 1998 had put it at 11.8% at one year and 28.4% at five years[90].

Cystoid macular edema and optic capture

One case of cystoid macular edema and optic capture (0.31%) were noted. Similar incidence of pseudophakic cystoid macular edema (0.3%) was reported by Sudhakar et al[17] . The incidence of 3.4-6.7% studied by Jaffe and Claymen[97] (1978).

Post operative complications at the end of third month

Posterior capsular opacification

At the end of 3rd month posterior capsular opacification was noted in 2.55% cases. YAG-laser capsulotomy was done . Emery[98] & colleagues found PCO in 28% of their patients in 2-3 years follow up. Few patients had lost the follow up .

Post operative complication at the end of sixth month

Retinal detachment

At the end of 6th month 2 cases (0.63%) were noted to have retinal detachment.

Post operative best corrected visual acuity

Of the 313 patients of best corrected visual acuity at the end of 6 weeks was 6/18 or better in 83.39% , <6/18 to 6/60 in 13.42% and less than 6/60 in 3.19% patients. Presenting vision was graded based on the WHO recommendations for acceptable outcomes after cataract surgery into good (6/18 or better), borderline (<6/18 to 6/60) and poor outcome (<6/60).Venkatesh et al[67] in 2002 in their study achieved BCVA of 6/18 or better in 94.4%, Ravindra et al[71] in 1994 reported a BCVA of 6/18 or better in 80.7%, Hennig et al[29] and Das et al[14] in their study reported a BCVA of 6/18 or better in 96.2% and 88.3% respectively. Das et al[14], Ravindra et al[71] and Gogate et al[23] in their

comparative studies between camps and hospitals reported that full visual acuity was higher in hospitals than in camps.

Table 23: **Post operative best corrected visual acuity in different studies**

Study	Percentage of cases		
	6/18 or better	<6/18-6/60	<6/60
Ravindra et al[71]	80.70%	16.80%	2.50%
Venkatesh et al[67]	94.40%	4.00%	1.60%
Das et al[14]	88.32%	10.62%	1.06%
Hennig et al[29]	96.2%	3.6%	0.2%
Our study	83.39%	13.42%	3.19%

Cataract and age related macular degeneration are both common causes of visual loss and reduced quality of life in the elderly. The conditions may coexist, in which case the presence of macular disease may limit the final visual result after cataract surgery[96].The reasons of poor visual acuity in our study were those related to cataract surgery and those unrelated to cataract surgery. Cataract surgery related causes were aphakia ,displaced IOL, corneal opacity due descemets membrane detachment, retinal detachment. Surgery unrelated causes were dry ARMD, optic atrophy,old CNVM and posterior staphyloma.

Astigmatism :

Astigmatism was graded and classified according to Holmström's gradation as

No astigmatism, when it was < 0.25 D

Non-significant, when it was ≥ 0.25 D but <1.00 D

Significant, when it was ≥ 1.00 D but < 2.00 D

High, when it was ≥ 2.00 D.

Post operative recorded astigmatism in our study was non significant (<0.25-1D) in 31.31% patients, significant (1-2D) in 46.02% and high (>2D) in 18.2%. Only spherical correction was accepted in 4.47% cases.

The axes of astigmatism were divided into three classes,"With the rule" (minus cylinder at 180 ±20),"Against the rule" (minus cylinder at 90 ±20) and "Oblique" (minus cylinder at 21 – 69 & 111 – 159). In present study, post operative, against the rule astigmatism(68.05%) was seen in majority of cases. With the rule astigmatism was present in 4.79%, oblique astigmatism was in 27.15% cases .However, the study lacks the record of surgically induced astigmatism .

In present study, association of type of cataract and intraoperative complications showed most of the mature type and combined type immature cataracts were associated with most of the intraoperative complications, but this was statistically not significance.

A relationship between intraoperative complications and best corrected visual acuity showed that only three cases of posterior capsular rupture with vitreous leak, two cases of descemets membrane detachment and one case of iridodialysis caused poor visual outcome. However this was not statistically significant.

We also studied relationship between post operative complications and age , which showed most complications were present in the age group 61-70 years .

A relationship between type of cataract and post operative complications was studied and the results showed that post operative complications were more common in combined type of immature cataract followed by mature type but statistical significance was not found.

CONCLUSION

Cataract remains the major cause of blindness which is thought to be increasing by 1-2 million per year. Developing countries has a growing number of patients awaiting cataract surgery. Manual small incision cataract surgery(MSICS) has become very popular technique of cataract surgery in India, and it is often used as an alternative to phacoemulsification.

Although MSICS demands skill and patience from the cataract surgeon, it is a safe, effective, and economical alternative to competing techniques and can be the answer to tackle the large backlog of blindness due to cataract.

In the current study 313 cases were screened at community based screening camps .Patients undergoing cataract extraction were observed during operative, early post-operative and late post-operative periods for the incidence of complications and following conclusions were drawn:

Intraoperative complications were seen in 7.99%. Posterior capsule rupture without vitreous loss (3.20%) was the most common intraoperative complication followed by posterior capsular rupture with vitreous loss(1.60%). A good vitrectomy should be done in cases of vitreous loss to prevent the postoperative sequence. The other intraoperative complications included Descemet's detachment(0.96%), iridodialysis(0.64%), premature entry(0.95%) and iris trauma(0.64%).

The most frequently occurring **complication during the immediate postoperative** period were in decreasing order corneal edema (12.46%),striate keratitis(8.95%),uveitis(7.04%) and secondary glaucoma & residual cortex(3.52%). The

other complications noted were iris prolapse(0.64%),shallow anterior chamber, hyphema, sterile endophthalmitis and iris incarceration(0.31%),

These complications can be prevented by minimizing excessive intraocular manipulations during surgery. Shallow anterior chamber, iris prolapse and hyphema should be treated promptly to prevent the sequalae. The incidence of endophthalmitis was low (0.31%). The considerable handling inside the anterior chamber during nucleus delivery increase the chances of iris injury, striate keratitis, and posterior capsular rupture. The surgeon has to be extra careful in the construction of the scleral tunnel and to achieve a good capsulorrhexis. Delivery of the nucleus through a small tunnel or rectangular tunnel can cause damage to the corneal endothelium resulting in early post operative corneal edema, which is sometimes recalcitrant to treatment.

The **post operative complications at the end of first week** included persistent uveitis(3.84%), corneal edema(3.20%), striate keratitis(1.93%) and secondary glaucoma(1.60%). Others were residual cortex, displaced IOL and optic capture(0.31%). The final astigmatism is less than that in the extracapsular cataract surgery and almost comparable to that in phacoemulsification.

Post operative **complications at end of 6 weeks** were PCO(2.88%), CME and optic capture(0.31%).

The most common late postoperative **complication at end of 3 months** was PCO (2.55%) and retinal detachment (0.64%) at end of 6^{th} month. There is, however, a concern of posterior capsular opacification in the long term, which needs to be addressed.The incidence of posterior capsule opacification can be reduced by thorough cortical clean up and posterior capsule polishing.

Best corrected visual acuity at end of 6 weeks in our study was 6/18 and even better in majority of cases(83.39%), between 6/18-6/60 in 13.42% and poor (3.19%) in remaining. Vision after cataract surgery could be compromised due to other eye diseases.

In this study we used preoperative vision as a proxy indicator of co-morbidity. If vision in eye with poor preoperative vision is regained, such cases were given more credit and were included in the 'excellent' grade of visual gain.

The World Health Organization (WHO) recommendations for acceptable outcomes after cataract surgery are good outcome (6/18 or better) in >85 % of cases, borderline outcome (<6/18 to 6/60) in <10 %, and poor outcome (<6/60) in <5 % of cases (WHO, 1998). Our study results are of significant relevance for the WHO's "Vision 2020" programme.

The overall complications of cataract extraction in base camps were very low in general. The complication of small incision cataract surgery is less and the procedure is well suited in our country, where there is large number of backlog of cataract cases. And it should be emphasized that SICS with PCIOL is safer than expected to rehabilitate the cataract patients and therefore the commonest cause of blind in this world can be cured by these operations which are gaining popularity as scientific and clinical results are immensely satisfactory.

SUMMARY

In the present study, the incidence of post operative complications in hospital based camp patients undergoing cataract surgery in 313 eyes have been evaluated. Manual small incision cataract surgery with posterior chamber intraocular lens implantation. was performed in majority of patients.

All the selected patients were examined, investigated treated and followed up for evaluating post operative complications after small incision cataract surgery with intraocular lens implantation .

All the facts described earlier are summarized as under:

- Females constituted the majority of patients.
- Age of the patients varies from 40 to 80years.
- Majority of cases are combined(posterior subcapsular with cortical) immature cataract and senile mature cataract .
- Majority of patients were legally blind.
- SICS was the procedure of choice, and 97.13% underwent PCIOL and 2.56% ACIOL implantation and 0.31% left aphakic.
- Overall incidence of intra operative complications were low(7.98%).
- Postoperatively oral antibiotic and analsegics were given. Patients also received topical antibiotic-steriod eye drops & antiiflammatory eye drops.
- All cases were followed up on day 1, 1st week, 4-6th week, 3rd month and at 6th months from the date of surgery.

Peroperative complications were

Posterior capsule rupture without vitreous loss – 3.20%

Posterior capsule rupture witht vitreous loss-1.60%

Descemet's membrane detachment – 0.96%

Tunnel related problem(premature entry)– 0.95%

Iris trauma– 0.64%

Iridodialysis – 0.64%

Early postoperative complications(day 1) were

Primary corneal edema—12.46%,

Striate keratopathy 8.95%,

Iridocyclitis – 7.04%.

Secondary glaucoma-3.52%

Residual cortex-3.52%

Sterile endophthalmitis – 0.31%

Iris prolapse – 0.64%

Shallow anterior chamber,hyphema,iris incarceration-0.31%

Postoperative complications at end of 1st week were

Persistence uveitis-3.84%

Corneal edema-3.20%

Striate keratitis-1.93%

Secondary glaucoma-1.60%

Residual cortex,displaced IOL,optic capture-0.31%

Postoperative complications at end of 6th weeks were

PCO-2.88%

CME,optic capture-0.31%

Late postoperative complications were-

Posterior capsular opacification – 2.55% at the end of 3rd month.

Retinal detachment—0.64% at the end of 6th month.

Final visual acuity

>6/18(good) --83.39%

<6/18-6/60 (borderline)—13.42%

<6/60 (poor)---3.19%

Astigmatism

<1 diopters – 31.31%

1-2 diopters – 46.01%

>2 diopters – 18.21%.

Base hospital approach has many advantages over peripheral eye camps.

These include:

• Adequate sterilization procedures.

• Availability of adequate equipment.

• Good preoperative and post-operative care.

• Early and better management of complications.

REFERENCES

1. Parsons.The Lens. Parsons Diseases of the Eye; Sihota Ramanjit,Tandon Radhika; 21st edition 2011 Elsevier.
2. GVS Murthy,Gupta Sanjeev K. Current status of cataract blindness and Vision 2020: The right to sight initiative in India . IJO. 2008; 56:6:489-94
3. Garry Brian, Hugh Taylor. Cataract blindness – challenges for the 21st Century. Bulletin of the World Health Organization. 2001; 79: 249-56.
4. Park K, Park's Textbook of Preventive & Social Medicine, 18th edn, Banarasidas Bhanot, Jabalpur.2005: 319-23.
5. Hennig A, Kumar J, Singh AK, Ansari A, Singh S,Gurung R, Foster A.World Sight Day and Cataract Blindness. Br. J. Ophthalmol.2002; 86: 830-31.
6. Dandona L, Dandona R, Anand R . Outcome and number of cataract surgeries in India: Policy issues for blindness control. Clin. Experiment Ophthalmol. 2003; 31: 23-31.
7. Gogate Parikshit; Complications And Mini-Review. Indian J Ophthalmol. 2009;57:45-9.
8. Natchiar G, Robin AL, Ravilla D . Attacking the backlog of India's curable blind. Arch Ophthalmol .1994; 112: 987-93.
9. 9) Venkatesh N, Prajna NV, Venkataswamy G. Cataract blindness – The Indian Experience Bulletin of the World Health Organization .2001; 79(3): 259-60.
10. Khosla PK. Community Ophthalmology: An Indian Perspective – Current Scientific Literature. New Delhi.1992: 113-16.
11. Daniel F, Goodman DF, Walter J, Stack WJ, Goltsch JD. Complications of cataract extraction with intraocular lens implantation. Ophthalmic Surgery. 1989; 20(2): 132-40.
12. Jaffe NS, Jaffe MS, Jaffe GF. Cataract surgery and its complications, 6th edn, St. Louis: Mosby, 1999.
13. Duke Elders SS. Diseases of the Lens and Vitreous, Glaucoma and Hypotony, In: System of Ophthalmology, Vol. XI, London; Mosby. 1969: 63-294.

14. Das T, Venkataswamy G. Surgical results – Comparison of patients operated in eye camp with patients operated in hospital. Indian J. Ophthalmol. 1983; 31: 924-27.
15. Balmer A, Andenmatten R, Hiroz CA. Complications of cataract surgery: Retrospective study of 1304 cases. Klin-Monstbl-Augenheilkd .1991; 198(5): 344-6.
16. Chitkara DK, Smerdon DL. Risk factors, complications and results in extracapsular cataract extraction. J. Cataract Refract Surg. 1997; 23: 570-74.
17. Sudhakar J, Ravindra RD, Natchiar G. Analysis of complication in 1000 cases of posterior chamber intraocular lens implantation. Indian J.Ophthalmol .1989; 37(2): 78-9.
18. Lumme P, Laatikainen LT. Risk factors for intraoperative and early postoperative complications in extracapsular cataract surgery. Eur-J Ophthalmol.1994; 4(3): 151-8.
19. Lumme P, Laatikainen LT. Factors affecting the visual outcome after cataract surgery. Int. Ophthalmol.1993-94; 17(6): 313-9.
20. Civerchia L, Apoorvananda SW, Natchiar G, Balent A, Ramakrishnan R, Green D. Intraocular lens implantation in rural India. Ophthalmic Surg. 1993; 24(10): 648-52.
21. Madhukar K Reddy. Complications of cataract surgery. Indian J. Ophthalmol.1995;43:201-9.
22. Verma L, Gupta SK, Murthi GVS, Goyal M, Pant TD. A follow up study on visual outcome after camp based intracapsular cataract extraction. J. Tropical Med and Int. Health .1996; 1: 342-47.
23. Gogate PM, Kulkarni AN. Comparison of cataract surgery in a Base Hospital and in Peripheral Eye Camps. Journal of Community Eye Health. 2002; 42.
24. Dandona L, Dandona R, Naduvilath TJ, McCarty CA, Mandal P, Srinivas M et al. Population based assessment of outcome of cataract surgery in an urban population in Southern India. Am. J. Ophthalmol . 1999; 127: 650-55.
25. Anand R, Gupta A, Ram J, Singh U, Kumar R, Visual outcome following cataract surgery in visual Punjab. Indian J. Ophthalmol. 2000; 48(2): 153-58.

26. Limburg H, Foster A, Vaidyanathan K, Murthy GVS. Monitoring visual outcome of cataract surgery in India. Bull WHO .1999; 77(6): 455-60.
27. Thulasiraj RD, Reddy A, Selvaraj S, Munoz SR, Ellivein LB. The Sivaganga Eye Survey-II: Outcomes of Cataract Surgery. Ophthalmic Epidemiol . 2002; 9(5): 313-24.
28. Kothari M, Thomas R, Parikh R, Braganza A, Kuriakose T, Muliyal J. The incidence of vitreous loss and visual outcome in patients undergoing cataract surgery in a teaching hospital. Indian J. Ophthalmol. 2003; 51(1): 45-52.
29. Hennig A, Yorston D, Kumar J, Foster A. Sutureless cataract surgery with nucleus extraction: Outcome of a prospective study in Nepal. Br. J. Ophthalmol .2003; 87(3): 266-70.
30. Snell RS, Lemp MA. Clinical anatomy of the eye, 2nd ed., Boston: Blackwell Science Inc., 1998; 197-204.
31. Bron AJ, Tripathi RC, Tripathi BJ. Wolff's Anatomy of the Eye and Orbit. 8th ed., London, Chapman & Hall; 1997: 411-42.
32. Patterson CA, Delamere NA. The Lens In: Hart WM Eds. Adler's Physiology of the Eye. 9th Ed. St. Louis: Mosby, 1992: 348-83.
33. Sihota R, Tandon R, Parson's Diseases of the Eye, 19th Ed., Oxford: Butterworth-Heinemann; 2003; 273-98.
34. Roper Hall MJ, Stallard's Eye Surgery, 7th Ed., London: Butterworths. 1989: 282-331.
35. Bernd Schroeder. Sutureless cataract extraction: Complications, Management and Learning Curves. J. Comm Eye Health 2003; 16: 48-58.
36. Vajpayee RB, Sharma N, Dada T, Gupta V. Management of posterior capsule tears. Survey of Ophthalmol, 2001; 45(6): 473-87.
37. O'Brien CS. Further observations on detachment of the choroid after cataract extraction. Arch. Ophthalmol. 1936; 16: 655-56.
38. Francois J. Aphakic glaucoma. Ann. Ophthalmol. 1974; 6: 429-42.
39. Podolsky MM, Ritchy R. Secondary glaucoma in aphakia. Ritch R, Shields MB (Eds). The Secondary Glaucomas. St. Louis, CV Mosby. 1982; 345-66.

40. Tomey KF, Traverso CE. The glaucomas in aphakia and pseudophakia. Surv. Ophthalmol, 1991; 36(2): 79-111.
41. Gross JG, Meyer DR, Robin AL et al. Increased intraocular pressure in the immediate postoperative period after extracapsular cataract extraction. Am. J. Ophthalmol, 1988;105:466-69.
42. Shaffer RN. The role of vitreous detachment in aphakic and malignant glaucoma. Trans Am Acad Ophthalmol Otolaryngol .1954; 58: 217-31.
43. Bourne W, Waller RR, Leisegang TJ, Brubaker RF. Corneal trauma in intracapsular and extracapsular cataract extraction with lens implantation. Arch. Ophthalmol 1981; 99: 1375-76.
44. Ajay Sharma. Corneal edema – Management. Ophthalmology Today 2003; 4(5): 170-71.
45. Apple DJ, Mamalis N, Loftfield K et al. Complications of intraocular lenses: A historical and histopathological review. Surv. Ophthalmol 1984; 29: 1054.
46. Hwang DG, Smith RE. Corneal complications of cataract surgery. J. Refract Corneal Surg. 1991; 7(1): 77-80.
47. Javitt JC, Vitale S, Canner JK et al. National outcomes of cataract extraction. Endophthalmitis after inpatient surgery. Arch. Ophthalmol .1991; 109: 1085-89.
48. Kattan HM, Flynn HW Jr, Pfugfelden SC et al. Nosocomial endophthalmitis survey: Current incidence of infection after intraocular surgery. Ophthalmology.1991; 98: 227-38.
49. Kresloff MS, Castellarin AA, Zaibin MA. Endophthalmitis. Surv. Ophthalmol. 1998; 43(3): 193-224.
50. Mamalis N, Nagpal M, Nagpal K, Nagpal PN. Endophthalmitis following cataract surgery. Ophthalmology Clinics of North America, 2001; 14(4): 661-74.
51. Cole HP III, Stallman J, Diamond J et al. High dose intravitreal dexamethasone for infectious endophthalmitis (abstract). Invest Ophthalmol Vis Sci 30 (Suppl), 1989; 509.
52. Dhanda RP, Kalevar VK. Cataract surgery in diabetics. Antiseptic 1962; 59: 477-80

53. Pandey SK, Apple DJ, Werner L, Maloof AJ, Milverton EJ. Posterior capsule opacification: A review of the etiopathogenesis - Experimental and clinical studies and factors for prevention. Indian J. Ophthalmol. 2004; 52: 99-112.
54. Irvine SR. A newly defined vitreous syndrome following cataract surgery. Am. J. Ophthalmol. 1953; 36: 599-619.
55. Moses L. Cystoid macular edema and retinal detachment following cataract surgery. J. Am. Intraocul Implant Soc. 1979; 5: 326-29.
56. Taylor DM, Sachi SW, Stern AL. Aphakic cystoid macular edema: Longterm clinical observations. Surv Ophthalmol. 1984; 28: 437-41.
57. Kraff MC and others. Prophylaxis of cystoid macular edema with topical indomethacin. Ophthalmology .1982; 89: 885-90.
58. Nagpal M, Nagpal K, Nagpal PN. Post cataract cystoid macular edema. Ophthalmology Clinics of North America, 2001; 14(4): 651-7.
59. Flach AJ and others. Prophylaxis of aphakic cystoid macular edema without corticosteroids. Ophthalmology .1990; 97: 1253-8.
60. Ahluwalia BK, Kalra SC, Parmer IPS, Khurana AK. A comparative study of the effect of antiprostaglandins and steroids on aphakic cystoid macular edema. Ind. J. Ophthalmol .1988; 36(4): 176-8.
61. Theobald GD, Hass JS. Epithelial invasion of the anterior chamber following cataract extraction. Trans Am Acad Ophthalmol Otolaryngol .1948; 52: 470-2.
62. Dunnington JH. Complications of wound healing after cataract surgery. In Haik GM editors: Symposium on diseases and surgery of the lens, St. Louis, CV Mosby; 1957.
63. Allen JC. Epithelial and stromal ingrowths. Am. J. Ophthalmol. 1968; 65:179-82.
64. Bettman JW Jr. Pathology of complications of intraocular surgery. Am. J. Ophthalmol. 1969; 68: 1037-50.
65. Lorussu V, Moramerco A, Pacelle E, Balacco-Gabriel C. Intraocular lens complications. Ann Ophthalmol. 1990; 22(10): 377-81.
66. Coonan P and others. The incidence of retinal detachment following extracapsular cataract extraction: A ten year study. Ophthalmology 1985; 92: 1096-101.

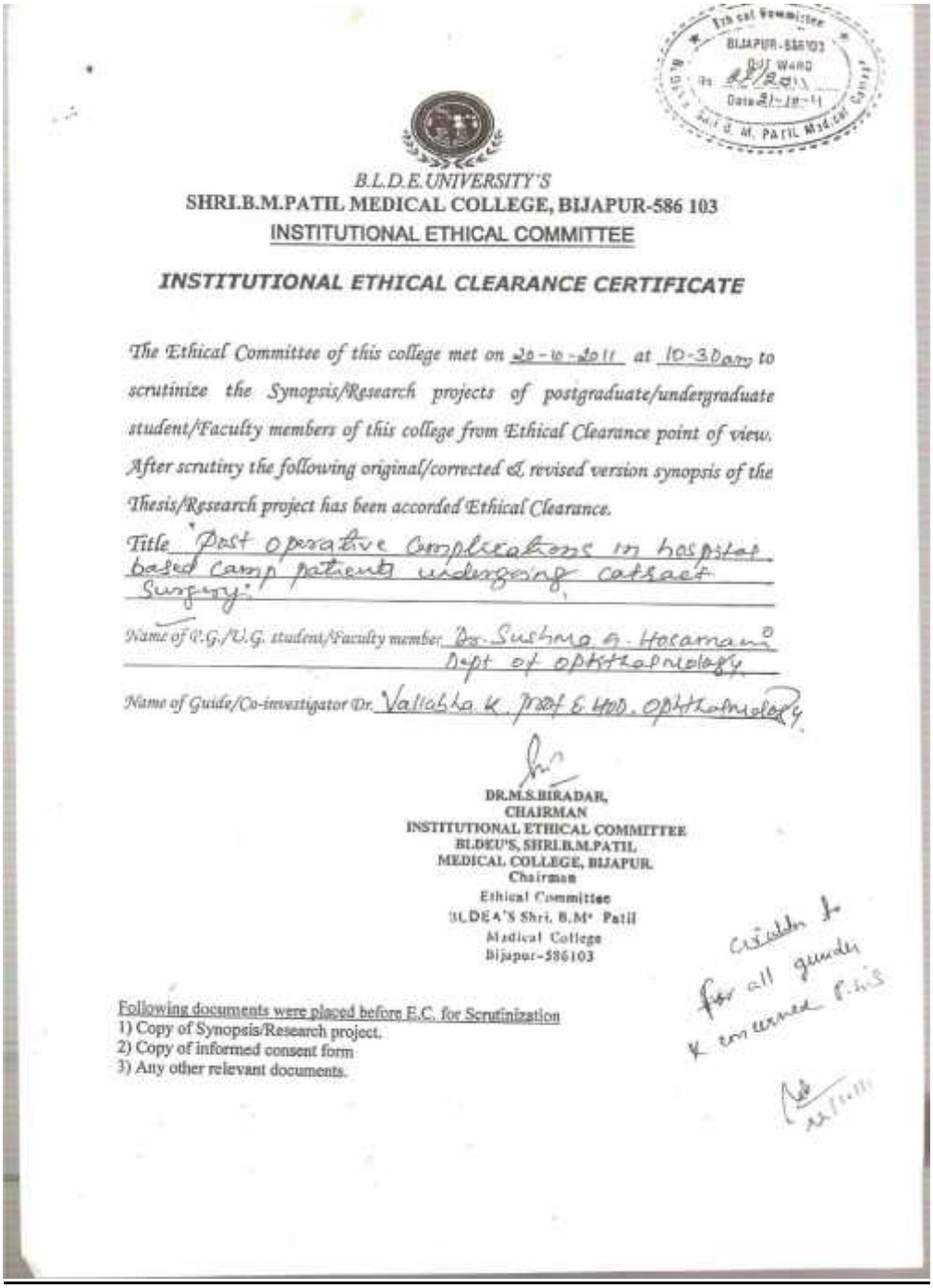
67. Venkatesh R, Muralikrishnan R, Balent LC et al. Outcomes of high volume cataract surgeries in a developing country. Br. J. Ophthalmol 2005; 89: 1079-83.
68. Desai P. The National Cataract Surgery Survey II. Clinical Outcomes Eye 1993; 7: 489-94.
69. Nishi O. Vitreous loss in posterior chamber lens implantation. J. Cataract Refract Surg. 1987; 13: 424-7
70. Natchiar G, Robin AL, Nalgirkar AR, Krishnadas R. Posterior capsule tears during extracapsular cataract surgery in India. Arch. Ophthalmol 1993; 111: 706-8.
71. Ravindra MS, Rekha G. Rural eye camp versus Base Hospital Camp. Indian Ophthalmology. 1996; 303-4.
72. Kapoor H, Chatterjee A, Daniel R, Foster A. Evaluation of visual outcome of cataract surgery in an Indian Eye Camp. Br. J. Ophthalmol. 1999; 83: 343-6.
73. Osher RH, Cionni RJ. The torn posterior capsule, its intraoperative behaviour, surgical management and long-term consequences. J. Cataract Refract Surg 1990; 16: 490-4.
74. Kratz RP, Mazzacco TR, Davidson B et al. The shearing intraocular lens: A report of 100 cases. J. Am. Intraocular Implant Soc, 1981; 7: 55.
75. Stark WJ, Worthern DM, Holladay JR et al. The FDA report on intraocular lenses. Ophthalmology. 1983; 90: 311-7.
76. McDonnell PJ, Zarbin MA, Green WR. Posterior capsule opacification in pseudophakic eyes. Ophthalmology 1983; 90: 1548-1553.
77. Rosenthal AL, Defaller JM, Mc Donald O, Bajjel GD, Beasley GH, Person DG, and Disbron DT : A review of world wide IOGEL intraocular lens implantation. Implants in Ophthalmology. : 1988; 3 , 39-44
78. Sugar HS : Prognosis in stripping of Descemet's membrane in cataract extraction, Am J Ophthalmol .1967; 63: 140-3.
79. Sparks GM : Descemetopexy : Surgical reattachment of stripped Descemet's membrane, Arch ophthalmol .1967;78 : 31-4.
80. Gogate PM, Kulkarni SR, Krishnaiah S, Deshpande RD, Joshi SA, Palimkar A, et al. Safety and efficacy of phacoemulsification compared with manual small

incision cataract surgery by a randomized controlled clinical trial: Six weeks results. Ophthalmology. 2005;112:869–74.

81. Bayramlar H, Cekic O, Totan Y. Manual tunnel incision extra capsular cataract extraction using the sandwich technique. J Cataract Refract Surg. 1999; 25:312–5.
82. Duch Mestres F, Mathieu A, Torres F, Lillo J, Castilla M. Intra-operative complications of planned extra capsular cataract extraction versus manual nucleofragmentation. J Cataract Refract Surg. 1996;22:1113–5.
83. Hepsen IF, Cekic O, Bayramlar H, Totan Y. Small incision extracapsular cataract surgery with manual phaco trisection. J Cataract Refract Surg. 2000;26:1048–51.
84. Thomas R, Kuriakose T, George R. Towards achieving small incision cataract surgery 99.8% of the time. Indian J Ophthalmol. 2000;48:145–51.
85. Guzek JP, Ching A. Manual small incision extracapsular cataract surgery in Ghana, West Africa. J Cataract Refract Surg. 2003;29:57–64.
86. Ruit S, Tabin G, Chang D, Bajracharya L, Kline DC, Richheimer R, et al. A prospective randomized clinical trial of phacoemulsification vs. manual sutureless small incision extracapsular cataract surgery in Nepal. Am J Ophthalmol. 2007;143:32–8.
87. Gogate PM, Deshpande MD, Nirmalan P. Why do phacoemulsification? Manual small incision cataract surgery is almost as effective and more economical. Ophthalmology. 2007;114:965–8.
88. Venkatesh R, Das M, Prasanth S, Muralikrishnan R. Manual small incision cataract surgery in eyes with white cataracts. Indian J Ophthalmol. 2005;53:173–6.
89. Venkatesh R, Tan CS, Kumar TT, Ravindran RD. Safety and efficacy of manual small incision cataract surgery for phacolytic glaucoma. Br J Ophthalmol . 2007;91:279–81.
90. 90) Shaumberg DA, Dana MR, Christen WG, Glynn RJ. A systemic overview of the incidence of posterior capsular opacification. Ophthalmology. 1998;105:1213–21.
91. Lundstrom M, Stenevi U, Thorburn W. Cataract surgery in the very elderly. J Cataract Refract Surg. 2000; 26:408-14.

92. Monestam E, Wachmeister L. Impact of cataract surgery on the visual ability of the very old. Am J Ophthalmol. 2004; 137:145-55.
93. McCarty CA, Mukesh BN, Fu C, Taylor H.R. The epidemiology of cataract in Australia. Am J Ophthalmol. 1999; 128:446-65.
94. Reidy A, Minassian DC, Vafidis G, et-al. Prevalence of serious eye disease and visual impairment in a north London population based, cross sectional study. BMJ. 1998; 316:1643-6.
95. Willerscheidt AB, Healey ML, Ireland M. Cataract surgery outcomes: importance of comorbidities in case mix. J Cataract Refract Surg. 1995; 21:177-81.
96. Minassian DC, Reidy A, Desai P, et al.The deficit in cataract surgery in England and Wales and the escalating problem of visual impairment: epidemiological modelling of the population dynamics of cataract. BJO : 2000; 84:4–8.
97. Clayman I .M., Jaffe N.S. Light D.G., Gascayady J.C. Intraocular lenses axial length and retinal detachment. Am. J. Ophthal 1980; 92: 778-80.
98. Emery JM, Wilhelmus KR,Rosenberg S:Complications of phacoemulsification. Ophthalmology. 1978; 85: 141.
99. Miller JJ, Scott IU, FlynnHWJr, Smiddy WE, Newton J, Miller D. Acute-onset endophthalmitis after cataract surgery (2000–2004): incidence, clinical settings, and visual acuity outcomes after treatment. Am J Ophthalmol. 2005; 139:983–87
100. Bell CM, Hatch WV, Cernat G, Urbach DR. Surgeon volumes and selected patient outcomes in cataract surgery. Ophthalmology. 2007; 114:405–10

ANNEXURE I- ETHICAL CLEARANCE



B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 20-10-2011 at 10-30am to scrutinize the Synopsis/Research projects of postgraduate/undergraduate student/Faculty members of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis/Research project has been accorded Ethical Clearance.

Title "Post operative complications in hospital based camp patients undergoing cataract Surgery."

Name of P.G./U.G. student/Faculty member Dr. Sushma G. Hosamani
Dept of Ophthalmology

Name of Guide/Co-investigator Dr. Vallikha K. Prof & HOD. Ophthalmology

DR.M.S.BIRADAR,
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.
Chairman
Ethical Committee
BLDEA'S Shri. B.M. Patil
Medical College
Bijapur-586103

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

Consent to be for all gender & concerned P.I's

ANNEXURE 2-

SAMPLE INFORMED CONSENT FORM:

TITLE OF PROJECT: " A CLINICAL STUDY OF POST OPERATIVE COMPLICATIONS IN HOSPITAL BASED CAMP PATIENTS UNDERGOING CATARACT SURGERY".

GUIDE : DR.VALLABHA.K

PRINCIPAL INVESTIGATOR : DR.SUSHMA.A.HOSAMANI

PURPOSE OF RESEARCH : I have been informed that this study will analyse the post operative complications in camp patients undergoing cataract surgery.

PROCEDURE : I am aware that in addition to routine care received and I will be asked series of questions by the investigations.I have been asked to undergo the necessary investigation,which will help the investigator as a part of routine management.

RISKS AND DISCOMFORT : I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study are not expected to these feeling which are associated with the usual course of treatment.

BENEFITS : I understand that my participation in this study will help to analyze post operative complications of cataract surgery.

CONFIDENTIALITY : I understand that medical information procedure by this study will become a part of my Hospital records and will be subject to the confidentiality and privacy regulation of the said hospital information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigators research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and videotapes or audio will be used only with my special written permission . I understand I may see the photographs and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION : I understand that i may ask more questions about the study at any time. **Dr Sushma.Hosamani** is available to answer my questions or concerns.I understand that I will be informed of any significant new findings discovered during the course of this study,which might influence my continued participation.

If during the study,or later,I wish to discuss my participation in or concerns regarding this study with a person not directly involved,I am aware that the social worker of the hospital is available to talk with me.A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OR PARTICIPATION: I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present of future care at this hospital.I also understand that **Dr.Sushma.Hosamani** may terminate my participate in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

INJURY STATEMENT : I understand that in the unlikely event of injury to me resulting directly from my participation in this, if such injury were reported promptly, then medical treatment would be avaibale to me, but no further compensation would be provided.I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained _____(patients/relavant name)the purpose of the research, the procedures risks and benefits to the best of my ability in patients own language.

Investigator : Date:

I confirm that **Dr.Sushma.Hosamani** has explained to me the research ,the procedure that I will undergo, and the possible risks and discomforts as well as benefits that I may experience.I have read and I understand this consent form.Therefore,I agree to give my consent to participate as a subject in this research project.

Participant / Gaurdian: Date:

Witness to signature : Date:

ANNEXURE 3-

PROFORMA

NAME : **DATE OF ADMISSION:**

AGE : **IP NUMBER:**

SEX:

HISTORY OF PRESENT ILLNESS:

C/O Diminision of vision Sudden/ Incidious onset

Duration: Right Eye/Left Eye

Associated symptoms

GENERAL PHYSICAL EXAMINATION: General Condition:

Pulse: BP: RR: Temp :

SYSTEMIC EXAMINATON: CVS/RS/CNS

RIGHT EYE	**OCULAR EXAMINATION**:	LEFT EYE
	External appearance	
	Ocular movements	
	Lids	
	Conjunctiva	
	Cornea	
	Sclera	
	Iris	
	Pupil	
	Lens	
	Visual acuity	
	Iop	
	Sac	
	Fundus	

INVESTIGATIONS:

Urine sugar: present/absent RBS:

DIAGNOSIS: Mature /Immature/ Hypermature cataract

SURGERY UNDERWENT: SICS /ECCS **DATE OF SURGERY:**

DATE OF DISCHARGE:

FOLLOW UP : AT 1 week, 1 month, 3 months , 6 months

POST OPERATIVE	1st day	1st visit	2nd visit	3rd visit	4thvisit
COMPLICATIONS (present/absent)	RE/LE	RE/LE	RE/LE	RE/LE	RE/LE
1)Wound leakage					
2)Wound dehiscence					
3)Corneal oedema					
4)Corneal straiae					
5)Detached Descemets membrane					
6)Anterior chamber depth defect					
7)Astigmatism					
8)Hyphema					
9)Prolapsed iris					
10)Iris trauma					
11)Uveitis					
12)Epithelial ingrowth					
13)Fibrous downgrowth					
14) Retained lens material					
15)Pupillary capture					
16)Lens dislocation					
17)Toxic lens syndrome					
18)Posterior capsular opacity					
19)Secondary glaucoma.					
20)Vitreous haemorrhage/loss					
21)Choroidal detachment/haemorrhage					
22)Cystoid macular edema					
23)Endophthalmitis					
24)Retinal detachment.					

INTERVENTIONS:

ANNEXURE 4 –

KEYS TO MASTER CHART

M-male

F-female

RE-right eye

LE-left eye

BCVA- best corrected visual acuity

SIMC-senile immature cataract

SMC-senile mature cataract

PSC-posterior subcapsular cataract

NS-nuclear sclerosis

CF-counting fingers

CTF- close to face

HM-hand movements

PL PR-perception of light ,projection of rays

Sph- spherical

m-meter

SICS- small incision cataract surgery

PCIOL-posterior chamber intra ocular lens

ACIOL-anterior chamber intra ocular lens

IO compln -intraoperative complications

PE-premature entry

ID-iridodialysis

PCR-posterior capsular rupture

VL-vitreous leak

DM Tear – Descemets membrane tear

SK- striate keratitis

CE-corneal edema

IR- iritis

LM-lens matter

EB –epibullae

PrUv-persistent uveitis

PPCO-pre exixting posterior capsular opacification

PCO-posterior capsular opacification

CME-cystoid macular edema

Ir In-iris incarseration

SAC-shallow anterior chamber

Hyp-hyphema

Uv-uveitis

SG-secondary glaucoma

RD-retinal detachment

OC-optic capture

IP-iris prolapsed

DIOL-displaced intra ocular lens

Endoph –endophthalmitis

ARM – age related maculopathy

ARMD- age related macular degeneration

CO-corneal opacity

Prim.optic atrophy- primary optic atrophy

BRVO- branch retinal vein occlusion

CNVM-choroidal neovascular membrane

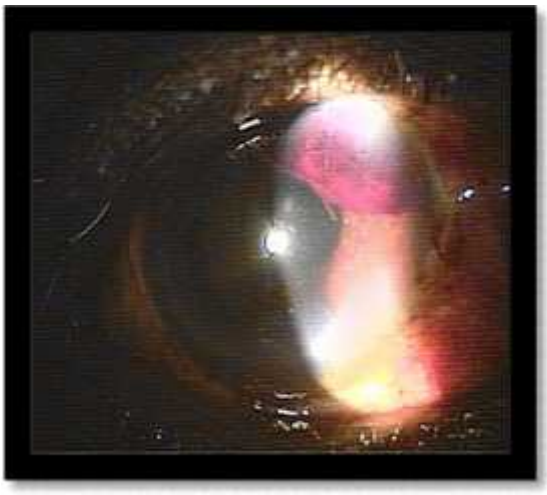
Post staphyloma- posterior staphyloma

ANNEXURE 5-

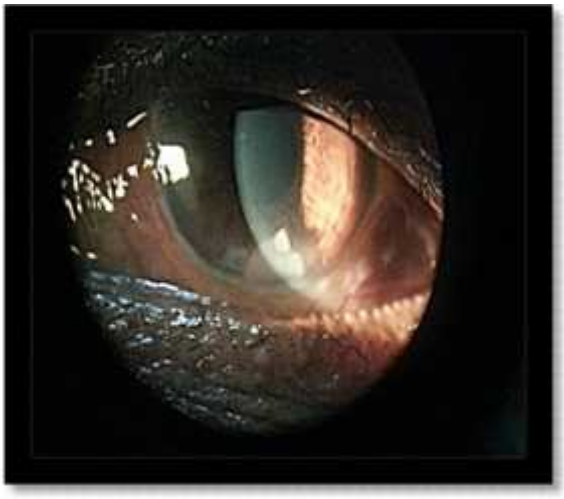
MASTER CHART

ANNEXURE 6-

A.Patients after cataract surgery



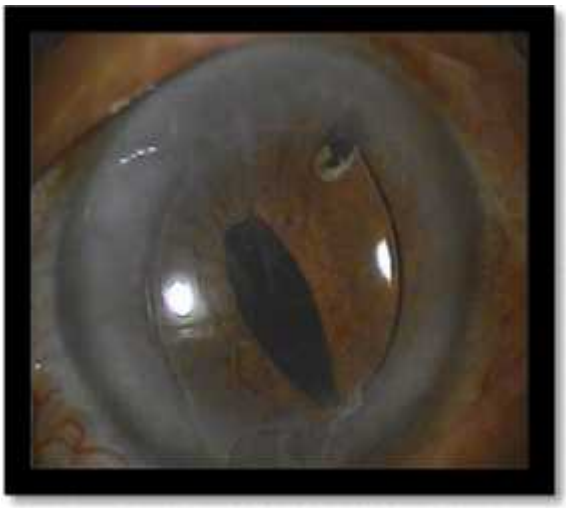
B.Hyphema on 3rd Post Operative



C.Residual lens matter in AC

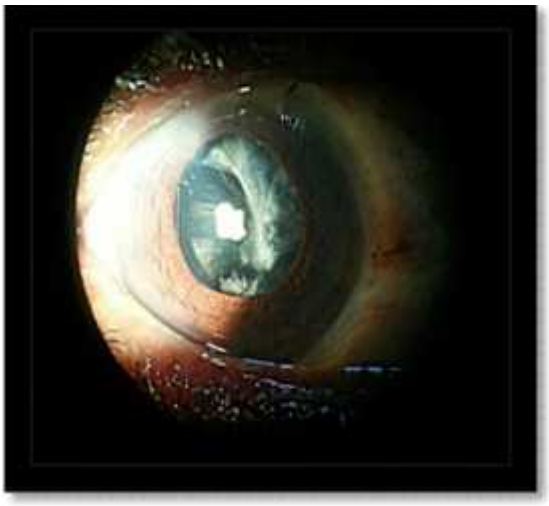
D.Iris incarceration in wound

E.Sterile Endophthalmitis



F.AC IOL implanted after intra op PC rent with vitreous leak

G.Optic capture with iris atrophy



H.Posterior capsular opacification

I.Posterior polar cataract

MASTER CHART

Sl no	NAME	IP No	AGE	SEX	EYE	Diagnosis:Cataract type	Fundus	Pre op BCVA	Procedure	IO compln	POST OPERATIVE COMPLICATION					POST OPERATIVE BCVA			Astigmatism	causes of low vision
											DAY 1	1 Week	1 Month	3 Month	6 Month	Day 1	1 Week	6 Week	1 Month	
1	Neelawwa Devar	22211	70	F	RE	SIMC - cortical	normal	CF 1/2m	SICS + PCIOL	None	CE,SK	SK	Nil	Nil	Nil	6/36P	6/9	6/9P	-2.5 X 90°	Nil
2	Mahadev Takkalaki	22216	70	M	LE	SIMC - cortical	normal	CF 3m	SICS + PCIOL	None	SK	Nil	Nil	Nil	Nil	6/60	6/12	6/12	-1.75 X 70°	Nil
3	Andanappa Biradar	22221	65	M	LE	SIMC - cortical	normal	HM	SICS + PCIOL	None	CE,SK,EB	EB	Nil	Nil	Nil	6/12	6/9	6/9	-2.5 X 100°	Nil
4	Bannewwa Indi	22220	80	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	CE ,SK	EB,CE,SK	Nil	Nil	Nil	CF 2m	6/36	6/60	-3.0X90°	ARM changes
5	Saraswati Badiger	22225	60	F	LE	SMC	no glow	HM	SICS + PCIOL	Iris trauma	CE,SK,IR	IR	Nil	Nil	Nil	6/24	6/18	6/18	-2.25X20°	Nil
6	Kallawwa Roogi	22210	65	F	RE	SMC	no glow	CF 1m	SICS + PCIOL	None	SK	IR	Nil	Nil	Nil	6/12	6/18P	6/18	-2.5X80°	Nil
7	Laxmibai Bandi	22214	70	F	RE	SMC	no glow	HM	SICS + PCIOL	None	IR	IR	Nil	Nil	Nil	6/36	6/36P	6/36P	-1.75X100°	dry ARMD
8	Shantawa Math	22218	50	F	RE	SIMC - PSC	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9P	6/9P	-1.5X100°	Nil
9	Sabu Naik	22219	65	M	LE	SIMC- cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/6P	6/9p	6/9P	-1.00X90°	Nil
10	Neelabai	23212	68	F	RE	SIMC- cortical+NS2+PSC	normal	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	-1.50X30°	Nil
11	Madiwallaww Kumbar	23651	70	F	LE	SIMC-PSC+cortical	normal	6/60	SICS + PCIOL	None	LM	ED,PrUv	Nil	Nil	Nil	6/18p	6/24p	6/24	-3.00x80°	Nil
12	Geeremma Biradar	23667	41	F	RE	SMC	glow +	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24	6/24	-1.00x100°	Nil
13	Laxmanna Soddagi	23659	75	M	LE	SIMC-cortical+ PSC	normal	6/60P	SICS + PCIOL	None	LM,IR	PrUv	Nil	Nil	Nil	6/18	6/12	6/12	-1.5x120°	Nil
14	Avamma Setti	23670	70	F	LE	SIMC-cortical+ PSC	normal	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/9	6/9	-1.25X15°	Nil
15	Sangamma Pattar	23666	80	F	RE	SIMC-cortical+ PSC	normal	CF 1m	SICS + PCIOL	None	CE	CE	Nil	Nil	Nil	CF 3m	6/24	6/24	-2.5X110°	Nil
16	Somalinga	23662	50	M	RE	SIMC-cortical+ PSC	normal	CF 1m	SICS + PCIOL	None	PPCO	PPCO	PCO	PCO	Nil	6/60	6/18	6/18	+0.25X160°	Nil
17	Bagarewwa Madari	23650	45	F	RE	SIMC-NS+cortical	normal	6/60P	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/9	6/9P	-3.5X105°	Nil
18	Boramma	23653	65	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	CE,LM	Nil	Nil	Nil	Nil	6/60	6/18	6/18P	-1.5x120°	Nil
19	Bangarewwa Kadani	23673	70	F	RE	SIMC-cortical+ PSC	normal	6/60	SICS + PCIOL	None	LM,IR	IR	Nil	Nil	Nil	6/60	6/24	6/24	-3.0x100°	Nil
20	Sangamma Hiremath	23669	65	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/24	6/24	-2.5X110°	Nil
21	Bhimawwa Adavi	23677	75	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/12	-2.0X80°	Nil
22	Neelagangavva Marshaalli	23665	75	F	RE	SIMC-NS3-4	no glow	HM	SICS + PCIOL	None	CE,SK	CE,SK	Nil	Nil	Nil	CF 1m	CF 1m	CF 1m	Nil	CO
23	Gurulingavva Shabadi	23649	65	F	LE	SIMC-PSC+cortical	normal	CF 1m	SICS + PCIOL	None	CE,SK	Nil	Nil	Nil	Nil	6/60	6/24	6/24	-1X70°	Nil
24	Appu Shimagar	23658	54	M	RE	SMC	no glow	PL PR	SICS + PCIOL	None	LM	LM	Nil	Nil	Nil	6/60	6/18	6/18	-1.5X90°	Nil
25	Shreshail	23655	50	M	RE	SIMC-PSC+cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	-1.5x120°	Nil
26	Darmanna.Madar	23654	55	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	SK	Nil	Nil	Nil	Nil	6/24	6/9	6/9	+ 2x 10	Nil
27	Sakeen. Asanchal	23675	70	F	LE	SIMC- NS 3 + Cortical	normal	PL PR	SICS + PCIOL	None	LM,CE	Nil	Nil	Nil	Nil	CF 3m	6/24	6/24	-1.5X90°	Nil
28	Ashimbee.Makanadar	24243	70	F	RE	SIMC- PSC+ Cortical	normal	CF 2m	SICS + ACIOL	PE,ID,PCR	CE,SK,IR	IR	CME	Nil	Nil	CF 2m	6/24	6/18	+0.25	Nil
29	Shantabai. Salunke	24268	70	F	LE	SIMC- cortcal	normal	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/9	6/9	-1x150°	Nil
30	Sonawwa.Salagarakar	24266	70	F	LE	SIMC-PSC+cortical	normal	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18	6/18	-2.75X102	Nil
31	Malakari. Badiger	24244	48	M	LE	SIMC-PSC+cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	-1X70°	Nil
32	Chandram.Pattar	24249	70	M	RE	SIMC- Cortical	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/9	6/9	-2.0X80°	Nil
33	Bagawwa	24251	60	F	LE	SIMC- NS4	no glow	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18P	6/12	-2.0X30°	Nil
34	Muttawwa.Govar	25990	80	F	RE	SIMC-NS3-4	no glow	PL PR	SICS + PCIOL	None	Ir	IR	Nil	Nil	Nil	6/60	6/36	6/24	-2.5x70	Nil
35	Kallawwa Hajeri	25988	65	F	RE	SIMC-PSC+cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/24	6/12	-2.00X80	Nil
36	Ratanawwa.Pujari	25992	70	F	RE	SIMC- PSC+ Cortical	normal	CF 1/2m	SICS + PCIOL	PE	Ir	Nil	Nil	Nil	Nil	6/36	6/36	6/24	-2.25x60	Nil
37	Nirmala.Bandekar	26001	45	F	RE	SMC	no glow	HM	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/24	6/9	6/9	-0.75X90	Nil
38	Shahebbi	25993	50	F	LE	SMC	no glow	CF 1/2m	SICS + PCIOL	None	CE,LM,Ir In	Nil	Nil	Nil	Nil	6/24	6/18	6/12	-2.0x70	Nil
39	Saraswati Vattar	26000	50	F	LE	SMC	no glow	CF 1/2m	SICS + PCIOL	None	CE,LM	Nil	Nil	Nil	Nil	6/36	6/18	6/18	-1.75X100°	Nil
40	Jakkawwa .Malakari	25989	65	F	RE	SIMC-NS 2-3	normal	CF 2m	SICS + PCIOL	None	CE,LM,Ir	Nil	Nil	Nil	Nil	6/24	6/12	6/12	-0.25X80	Nil
41	Chandrawwa.Gayakwad	25999	70	F	RE	SIMC - cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/24	6/12	-1.5X90°	Nil
42	Satirawwa.Hattalli	27118	70	F	LE	SIMC-PSC+cortical	PPA	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/12	6/12	-2.0X90	Nil
43	Shabira.Sutar	27116	65	F	RE	SIMC-PSC+cortical	no glow	CF 1m	SICS + PCIOL	None	PPCO	PPCO	PCO	Nil	Nil	6/18	6/18	6/12	-1.75X100°	Nil
44	Shivayogi	27117	45	M	RE	SIMC-PSC	normal	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/18	6/12	-1.5X70	Nil
45	Danabai.Rathod	293	75	F	RE	SIMC-PSC+cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/12	-2.0X90	Nil
46	Bhimashi Koli	291	65	M	RE	SIMC-PSC	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	-2.00X90	Nil
47	Indubai	296	65	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/9	-1.5X100°	Nil
48	Sarojini .Kyadi	287	62	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/12	6/9	-1.25X85	Nil
49	Dannamma Goni	289	80	F	RE	SIMC-NS3-4	no glow	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24P	6/24P	-2.5X80°	Nil
50	Kashibai Biradar	295	70	F	LE	SIMC-PSC+cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/24	6/24	-2.75X80	Nil
51	Padmawwa	292	60	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/6P	6/6	-1.25X90	Nil
52	Jaayashree	294	72	F	RE	SIMC-PSC+cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	-0.5X80	Nil
53	Laxmibai	288	56	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6P	6/6	-1.0X90	Nil

54	Gadigeppa	294	70	M	RE	SMC	no glow	PL PR	SICS + PCIOL	PE	Nil	Nil	Nil	Nil	Nil	6/36P	6/12	6/12P	-1.50X30°	Nil
55	Chandrakant Kale	290	67	M	LE	SIMC-PSC	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/9P	6/9P	-0.75X100	Nil
56	Basalingawwa	1423	45	F	LE	SIMC-PSC+cortical	normal	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/9	6/9	-1.00X90°	Nil
57	Prabhavati	1401	65	F	LE	SIMC-Cortical	PPA	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	PCO	Nil	6/18	6/9	6/9	-0.75X90	Nil
58	Chandrawwa Ambiger	1393	65	F	RE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/9	6/9P	-1.25X100	Nil
59	Siddappa Padagnur	1414	75	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9P	6/9	-0.75X90	Nil
60	Krishnabai Mane	1420	60	F	RE	SIMC-NS4	no glow	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/6P	6/6	-1.75X100°	Nil
61	Nabilala	1392	48	M	LE	SIMC-Cortical	no glow	HM	SICS + PCIOL	None	PPCO	PPCO	PCO	PCO	Nil	6/12	6/6	6/6	-1.25X130	Nil
62	Allisab Angadi	1398	60	M	RE	SIMC-Cortical	normal	6/36p	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/18p	6/18	-2.25x170	Nil
63	Bhagawwa Kambale	1395	65	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/24	6/12P	-1.50X90	Nil
64	Dastagerisab Jainapur	1388	50	M	LE	SIMC-Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	+0.75X150	Nil
65	Sangawwa Bijapur	1400	70	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12P	6/12	-1.0X90	Nil
66	Tirtawwa Badiger	1391	71	F	LE	SIMC-NS4	no glow	CF 1m	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	CF CTF	6/18	6/18	-1.50X70	Nil
67	Jayabai Karpe	1390	60	F	RE	SIMC-Cortical	normal	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6	6/6	-1.00X90°	Nil
68	Bimashi	1402	60	M	RE	SIMC-Cortical	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/6P	6/6P	-2.50X90	Nil
69	Lalasab Magare	2599	75	M	RE	SMC	no glow	CF CTF	SICS + PCIOL	None	SAC	Nil	Nil	Nil	Nil	6/60	6/18	6/18P	-3.50X40	Nil
70	Laxmibai	2601	65	F	RE	SIMC-PSC+cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/9	6/9	+0.50X90	Nil
71	Subadra Badiger	2600	75	F	RE	SIMC-NS2-3	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/12P	6/9P	-1.75X110	Nil
72	Dundawwa	2604	75	F	LE	SIMC -NS1-2	normal	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/18P	6/18	2.50X90	ARM changes
73	Yallappa	3765	70	M	LE	SIMC-cortical+NS2	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	PCO	Nil	6/18	6/12	6/36	-2.00X170	Nil
74	Kamalabai	3770	45	F	RE	SIMC-PSC	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/9	6/18	-0.75X75	Nil
75	Gowrawwa	3773	70	F	LE	SIMC-PSC	normal	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/9	6/9P	+0.50X90	Nil
76	Gangabai	3768	65	F	RE	SIMC-Cortical+PSC+NS2	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/9	6/9	+0.50X90	Nil
77	Kemu Rathod	3785	65	M	LE	SIMC-NS3-4	no glow	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/12P	6/12	-1.50X100	Nil
78	Parvathi	3764	80	F	LE	SIMC-NS3+Cortical	glow +	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/18P	6/12	-1.75X90	Nil
79	Manohar	3772	52	M	RE	SIMC-PSC	normal	CF 2m	SICS + PCIOL	None	Hyp(day 3)	Nil	Nil	Nil	Nil	6/12	6/12	6/12	-1.00x70	Nil
80	Bhagawan Rao	3775	72	M	LE	SIMC-Cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/18	6/12	-2.5X50	Nil
81	Sharanawwa Gaudappagol	3677	60	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Uv	Nil	Nil	Nil	Nil	6/12	6/12P	6/9	+1.00X90	Nil
82	Siddappa Shegunsi	3766	65	M	LE	SIMC-PSC +Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/9	+0.5X90	Nil
83	Noorjan Mirji	3771	50	F	RE	SIMC-NS4	no glow	CF 1/2m	SICS + PCIOL	None	CE	Nil	Nil	PCO	Nil	6/24P	6/24	6/12	+0.50X90	Nil
84	Bhagubai	5417	75	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/18	6/9	-1.00X90°	Nil
85	Satawwa	5426	65	F	RE	SMC	no glow	PL	SICS + PCIOL	PCR	Uv	Nil	Nil	Nil	Nil	6/24P	6/24	6/18P	-2.00X90	Nil
86	Babu Heralagi	5418	52	M	RE	SIMC-PSC+cortical	normal	6/60P	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9P	6/6	6/6	+0.50X80	Nil
87	Sangappa Jawanar	5419	68	M	RE	SIMC-Cortcal	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/6	6/6	-1.25X100	Nil
88	Balamma Myageri	5423	53	F	RE	SIMC-Cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/9	6/6P	-1.50X90	Nil
89	Makanabai Lamani	7526	60	F	LE	SIMC-NS2-3	normal	CF 2m	SICS + PCIOL	None	Uv,LM	Nil	Nil	Nil	Nil	CF 2m	6/12	6/12	-1.25X10	Nil
90	Gangabai Jadav	7525	45	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/6	6/9P	6/9P	-1.50X90	Nil
91	Kalavati	7520	65	F	LE	SIMC-PSC+cortical	PPA	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/6	6/6	-1.00X90°	Nil
92	Ballawwa Bajantari	7522	50	F	LE	SIMC-Cortical	normal	6/60P	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12P	6/12	-1.25X40	Nil
93	Bangarewwa Sarawad	7521	70	F	LE	SIMC-PSC	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/12	6/12	-1.50X90	Nil
94	Nagawwa Vagadurgi	7529	70	F	RE	SIMC- PSC+ Cortical	PPA	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/24P	6/24	-3.50X70	Nil
95	Laxmibai Shinde	9161	70	F	LE	SIMC - PSC+Cortical	glow +	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/12	6/9p	-2.00x70	Nil
96	Vittal Karigar	9160	70	M	LE	SIMC- PSC+ Cortical	glow +	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/12	6/12P	-3.50X40	Nil
97	Gowrawwa Awarsang	9162	70	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	CE	Nil	Nil	Nil	6/9	6/24P	6/12P	-2.5X6O	Nil
98	Irappa Vadder	9659	65	M	RE	SIMC-PSC +Cortical	normal	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9P	6/9P	-1.00x100°	Nil
99	Yamanawwa	9658	70	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18P	6/18	-2.50X90	Nil
100	Sidappa Jumanal	10861	80	M	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 1m	6/24	6/18	+0.50X90	Nil
101	Sharanabai Talbawadi	10862	42	F	RE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6P	6/6	-1.50 Sph	Nil
102	Gouramma Badiger	10873	65	F	LE		glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/24P	6/12P	-2.50X80	Nil
103	Janakabai Rathod	10869	75	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	SK	Nil	Nil	Nil	Nil	6/24	6/24	6/9	-1.00X85	Nil
104	Sabu Indi	10870	62	M	LE	SIMC-PSC +Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/18	6/9	-1.00X90°	Nil
105	Shakuntala Chawan	10863	55	F	LE	SIMC-PSC +Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/18	6/12	-0.50X100	Nil
106	Yeshubai Sendage	10864	71	F	RE	SIMC-NS4	glow +	PL	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/18	6/9P	-2.50X120	Nil
107	Janabai Tambe	10866	60	F	LE	SIMC-NS4	glow +	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/12	6/9	-1.75X40	Nil
108	Shankremma Patil	11432	60	F	LE	SIMC-PSC+NS2	glow +	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/18	6/9	-0.75X60	Nil
109	Savalgeppa Nula	11421	65	F	RE	SIMC-PSC +Cortical	glow +	CF 2m	SICS + PCIOL	None	SK,CE	Nil	Nil	Nil	Nil	6/24	6/12P	6/9	-0.75X70	Nil
110	Budawwa Baragal	11447	65	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24P	6/18P	-2.50X75	Nil
111	Samawwa Malinmani	11438	65	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/9P	6/6	-0.75X70	Nil
112	Sangawwa Manavi	11455	70	F	RE	SIMC-PSC +Cortical	normal	CF 1m	SICS + ACIOL	PCR	SG,CE	CE	Nil	Nil	Nil	CF CTF	CF 3m	6/18	-2.50X90	Nil
113	Mahadevi Hadalageri	11429	65	F	RE	SIMC-PSC+cortical	normal	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/36	6/9P	-1.50X90	Nil
114	Sitawwa Chalwadi	11456	60	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/12	6/12P	-1.00X90°	Nil

115	Basamma Harnal	11433	68	M	LE	SIMC-PSC +Cortical	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/24P	6/18	-2.00X90	Nil
116	Lalabeen Nadaf	11453	68	F	RE	SIMC -Cortical	normal	HM	SICS + PCIOL	None	CE ,SK	Nil	Nil	Nil	Nil	CF CTF	6/18	6/9	-2.50X90	Nil
117	Ladamma Nadaf	11458	70	F	RE	SIMC-NS3	normal	CF 2m	SICS	PCR,VL	SG,CE,SK	CE ,SK	Nil	Nil	Nil	CF CTF	CF 3m	CF 3m	Sph +12	Nil
118	Basavaraj Kumbar	11463	43	M	RE	SIMC-Cortical	glow +	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/12	6/9	-0.75X110	Nil
119	Ramanna Badiger	11444	60	M	LE	SIMC-Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/12	6/9	+1.50X180	Nil
120	Pandit Shambewad	11459	60	M	RE	SIMC-NS 3	no glow	CF 1/2m	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/24P	6/24	6/12P	-3.00x90°	Nil
121	Sayabawwa Almel	11442	65	M	RE	SIMC-PSC+ Cortical	normal	CF 1m	SICS + PCIOL	None	CE SK	Nil	Nil	Nil	RD	6/36P	6/36	6/24P	-4.50X90	Nil
122	Somalu Chawan	11452	70	M	RE	SIMC-PSC +Cortical	glow +	PL PR	SICS + PCIOL	None	CE SK	CE	Nil	Nil	Nil	CF 2m	6/18P	6/12	-1.75X90	Nil
123	Nibewwa Kolur	11449	70	F	RE	SIMC-NS3-4	glow +	HM	SICS + ACIOL	PCR	CE SK	CE	Nil	Nil	Nil	CF 1m	6/18	6/24	+2.5X90	Nil
124	Nenewwa Dodamani	11439	70	F	LE	SIMC -PSC +Cortical	glow +	CF 2m	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	CF 3m	6/18	6/9P	-1.50X100	Nil
125	Devamma Gour	11465	70	F	RE	SIMC- PSC+ Cortical	glow +	CF CTF	SICS + PCIOL	None	CE SK	Nil	Nil	PCO	Nil	CF 1m	6/24	6/24	-1.25X90	Nil
126	Siddanna Biradar	11460	65	M	RE	SMC	no glow	HM	SICS + PCIOL	Iris trauma	CE SK	Nil	OC	Nil	Nil	6/24	6/18	6/9	-2.00X90	Nil
127	Sangappa Bagali	11441	70	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	CE SG	SG CE	Nil	Nil	Nil	CF CTF	HM	6/18P	-1.75X90	Nil
128	Bagawwa Harijan	11448	70	F	RE	SIMC - PSC+Cortical	glow +	CF 1m	SICS + PCIOL	None	CE SK	Nil	Nil	Nil	Nil	6/36P	6/36P	6/24	-1.50X90	ARM changes
129	Prabakar Joshi	11428	70	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/18	6/9P	-1.00X90°	Nil
130	Ratnabai Kattimani	11431	70	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/36	6/12	-2.50X120	Nil
131	Shivappa Hebbal	11426	70	M	RE	SIMC -PSC +Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/9P	-1.00X80	Nil
132	Ramanabai Chawan	11428	60	F	LE	SIMC -PSC +Cortical	normal	HM	SICS + PCIOL	None	CE,SG	CE,SG	Nil	Nil	Nil	HM	6/60	6/24P	-3.50X60	Nil
133	Bhimabai	11462	60	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/24P	6/9P	-1.00x100°	Nil
134	Bheemashankar Herakal	11446	65	M	LE	SIMC-NS4	glow +	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	PCO	Nil	6/18	6/18	6/60	-3.25X120	Nil
135	Kantawwa Pujari	11454	70	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	HM	CF CTF	CF 1m	Nil	Dry ARMD
136	Lakshmibai Hosamani	11430	70	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18P	6/12	-1.75X70	Nil
137	Gangabai Biradar	11427	68	F	RE	SIMC-PSC +Cortical	normal	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/18	6/12	-1.50X90	Nil
138	Shantabai Mangalewal	11437	62	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/24P	6/24	-3.50X80	ARM changes
139	Bouramma Biradar	11443	65	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/18	6/12	-1.00X90°	Nil
140	Kasumbi Haranal	11440	70	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12P	6/12P	-2.50X100	Nil
141	Siddanna Shabad	11425	70	M	LE	SIMC -PSC	normal	CF CTF	SICS + PCIOL	None	SG	Nil	Nil	Nil	Nil	CF 3m	6/24	6/18	-1.00x100°	Nil
142	Sangawwa Mathpati	12512	65	F	RE	SIMC-PSC	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/18P	6/9P	-2.50X90	Nil
143	Malewwa Kumbar	12515	65	F	LE	SIMC -PSC +Cortical	glow +	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 2m	6/12P	6/9	-1.75X60	Nil
144	Dundawwa Tambad	12499	65	F	LE	SIMC -PSC +Cortical	normal	CF 1m	SICS + PCIOL	None	CE,SK	Nil	Nil	Nil	n	CF CTF	6/60	6/18P	-1.75X70	Nil
145	Gangappa Navi	12498	65	M	RE	SIMC -Cortical	glow +	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/24	6/6	-1.50X120	Nil
146	Shivamma Anikal	12505	65	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18P	6/9	-1.50X90	Nil
147	Kasturi Baragi	12517	62	F	RE	SIMC-cortical	normal	CF 1/2m	SICS + PCIOL	None	Nil	Nil	PCO	PCO	Nil	CF 2m	6/12	6/12	-1.50X90	Nil
148	Sangappa Palled	12516	65	M	RE	SIMC-PSC +Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 4m	6/12P	6/9	-1.75X90	Nil
149	Shivappa Uppar	12506	50	M	LE	SIMC -PSC +Cortical	glow +	HM	SICS + PCIOL	DM Tear	CE	Nil	Nil	Nil	Nil	CF 4m	6/24P	6/9	-1.00X90°	Nil
150	Lakshmibai Malvadkar	12518	65	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 3m	HM	HM	Nil	Prim. optic atrophy
151	Gangawwa Galgali	12508	65	F	LE	SIMC - PSC + Cortical	glow +	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18	6/6	-2.50X90	Nil
152	Gangawwa Hitnalli	12502	65	F	RE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 2m	6/12	6/12P	-1.50X100	Nil
153	Bhagawwa Desai	12507	65	F	RE	SIMC- PSC+ Cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 3m	6/12	6/9	-1.50X90	Nil
154	Kallappa Ghosarwad	12497	80	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 3m	6/24	6/6	-1.75X90	Nil
155	Yamunaji Ranse	12501	60	M	LE	SIMC -PSC +Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 2m	6/24	6/24P	-2.25X80	Old BRVO inferior
156	Jakkawwa Uppar	12504	60	F	LE	SIMC -PSC +Cortical	glow +	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF CTF	6/12	6/6P	-1.50X90	Nil
157	Shantawwa Masabinal	12500	45	F	LE	SIMC - PSC+Cortical	glow +	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/12	+1.00X60	Nil
158	Gourawwa Janoji	12513	65	F	LE	SIMC -N3 +Cortical	glow +	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/36P	6/18	-1.75X90	Nil
159	Devaki Hattigeri	13542	55	F	RE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/60	6/36	-1.75X90	ARM changes
160	Bhagawwa Kuri	13536	80	F	LE	SIMC- PSC+ Cortical	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24	6/24	-3.50X100	Polar cataract
161	Shaywwa Mali	13538	65	F	RE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24	6/9	-1.50X90	Nil
162	Shantabai Gadyal	14669	60	F	RE	SMC	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6	6/6	+1.50X180	Nil
163	Sattewwa Solapur	14658	75	F	RE	SIMC -PSC +Cortical	normal	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/18	-2.00X80	Nil
164	Putalabai Manoor	14655	62	F	RE	SIMC-PSC	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/6P	-1.50X80	Nil
165	Kamallawwa	14654	60	F	RE	SIMC-PSC +Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18P	6/12	-1.59X90	Nil
166	Yallappa	14656	60	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/18	6/9P	-1.00X120	Nil
167	Mallappa	14670	70	M	LE	SIMC -PSC +Cortical	normal	CF 3m	SICS + PCIOL	None	SG,CE	Nil	Nil	Nil	Nil	CF CTF	6/24	6/12	-1.50X90	Nil
168	Ladamma Khiji	15804	60	F	RE	SIMC -PSC +Cortical	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12P	6/9	-1.75X90	Nil
169	Rukmabai Kambale	15809	60	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12P	6/6	-0.5X70	Nil
170	Seetawwa Madar	15802	68	F	LE	SIMC -PSC +Cortical	glow +	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/9P	6/9	-2.50X80	Nil
171	Drupadibai Manvar	15810	65	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/6P	6/6P	-1.00X90°	Nil
172	Gurubai Kamkandi	15803	45	F	RE	SIMC- PSC+ Cortical	glow +	CF CTF	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/36	6/9	6/6	-1.50X90	Nil
173	Yashubai Sharagar	15807	65	F	LE	SMC	no glow	HM	SICS + PCIOL	None	IP	Nil	Nil	Nil	Nil	6/36	6/24P	6/12P	-2.00X90	Nil
174	Vittabai Barakade	15811	60	F	LE	SMC	no glow	HM	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	CF 4m	6/18P	6/12P	-2.0X90	Nil
175	Maleppa Gudadinni	15815	70	M	LE	SIMC -PSC +Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 2m	6/12	6/9P	-1.75/90	Nil

176	Sayebi	16936	60	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/60	6/12	-0.50X100	ARM changes
177	Bhimappa	16932	61	M	RE	SIMC - PSC + Cortical	normal	CF 1/2m	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/9	6/9	6/6P	-1.00X180	Nil
178	Shantappa	16941	65	M	RE	SIMC - PSC + Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/9	6/6	-0.50X100	Nil
179	Basamma Matpati	18651	60	F	RE	SIMC - PSC + Cortical	glow +	CF 1m	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/36	CF 3m	HM	Nil	Old CNVM
180	Muttawwa Karjol	18647	70	F	LE	SIMC - PSC + Cortical	glow +	CF 2m	SICS + PCIOL	None	CE,SK	Nil	Nil	Nil	Nil	6/18P	6/12	6/9	-2.50X80	Nil
181	Hucchawwa Sunyad	18653	70	F	LE	SIMC - PSC + Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/9P	-2.00X100	Nil
182	Mahadevi Swami	18650	65	F	LE	SIMC -Cortical +NS2	glow +	CF 3m	SICS + ACIOL	PCR	Nil	Nil	Nil	Nil	Nil	6/60	6/36	6/36	Nil	RD
183	Sabu Indi	18648	65	M	RE	SIMC -PSC +Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/6	6/6	-0.50X60	Nil
184	Sabu N Indi	18651	66	M	LE	SIMC -Cortical+NS 3	glow +	CF CTF	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/18P	6/12	6/9P	+0.50X40	Nil
185	Kantawwa Devanal	19953	65	F	LE	SIMC -Cortical +NS2	glow +	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/18	6/12	+1.50X120	Nil
186	Revanasiddappa Kannur	19946	65	M	RE	SIMC-PSC +Cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/6P	6/6	-1.00X50	Nil
187	Hanamanth Kambar	19939	70	M	LE	SIMC- NS2	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	+1.50X50	Nil
188	Laxamawwa Chalvadi	19955	60	F	RE	SMC	no glow	PL PR	SICS + PCIOL	PCR	CE,SK	earlyPCO	PCO	Nil	Nil	6/24	6/18	6/24	-3.00X120	Nil
189	Bhimappa Hosamani	19969	55	M	RE	SIMC -PSC +Cortical	glow +	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/12	6/18	-2.00X100	Nil
190	Kasturi Hadapad	19948	60	F	RE	SIMC - PSC	glow +	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/12	6/9	-1.25X130	Nil
191	Shivalingawwa Math	19959	65	F	LE	SIMC - PSC+Cortical	glow +	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/12	6/6P	-0.50X110	Nil
192	Lakkawwa Honshyal	19954	60	F	LE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/24	6/18	-1.00X70	Nil
193	Laxman Biradar	19941	60	M	LE	SIMC -PSC	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/18P	6/9	-1.50X70	Nil
194	Yallawwa Kallur	19957	70	F	RE	SMC	no glow	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/24P	6/18P	-2.50X90	Nil
195	Chandabai Walikar	21529	70	F	RE	SIMC-PSC+cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	PCO	Nil	Nil	6/18	6/18	6/24	-1.75X110	Nil
196	Basalingawwa Biradar	21532	80	F	RE	SIMC- PSC+ Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/9	6/6P	-1.75X150	Nil
197	Yeshubai Jadhav	21530	70	F	LE	SIMC -PSC	normal	CF 1m	SICS + PCIOL	None	CE,SK	Nil	Nil	Nil	Nil	6/18P	6/12	6/9	-1.75X70	Nil
198	Jateppa Karajagi	21531	75	M	RE	SMC	no glow	PL PR	SICS + PCIOL	DM Tear	CE	Nil	Nil	Nil	Nil	6/24P	CF 1m	CF 1m	Nil	DM detachment
199	Gurulingappa Arakeri	21533	72	M	LE	SIMC-NS3	no glow	CF 3m	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/12P	6/12	6/12	-1.00X60	Nil
200	Satyeppa Pujari	21721	70	M	RE	SIMC-PSC	no glow	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18	6/12P	-1.50X60	Nil
201	Yamanappa Biradar	21719	68	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	CF 3m	6/12	6/12	-2.50X80	Nil
202	Shantawwa Vasanthi	21724	50	F	LE	SIMC- PSC+ Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	PCO	Nil	Nil	6/12P	6/12	6/12	+1.00X80	Nil
203	Tipawwa Kumbar	21720	60	F	LE	SIMC - PSC+Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/6P	6/6	6/6	+0.50X120	Nil
204	Sangappa Javeagi	22326	80	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Uv,SG	IR	Nil	Nil	Nil	6/24	6/18p	6/12	-1.50x90	Nil
205	Rabai Kalpe	22330	60	F	RE	SIMC- PSC+ Cortical	normal	CF 2m	SICS + PCIOL	None	DIOL,Ir	Nil	Nil	Nil	Nil	6/18	6/24	6/18	-2.00X90	Nil
206	Housabai Gotrale	22322	65	F	LE	SIMC-PSC +Cortical	glow +	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/12	6/6P	-1.00X80	Nil
207	Rukmini Kumbar	22325	58	F	RE	SIMC -CortcaL+NS2	normal	CF 2m	SICS + PCIOL	None	Ir	Nil	Nil	Nil	Nil	6/12	6/9	6/6P	-0.50X90	Nil
208	Satyawwa Hosakoti	22328	80	F	RE	SMC	no glow	PL	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36P	6/24	6/24	-3.50X80	Nil
209	Bhimappa Chalwadi	23574	55	M	RE	SMC	no glow	PL PR	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	6/24P	6/12	6/9P	-1.50X120	Nil
210	Nandeppa	23575	55	M	RE	SIMC - PSC+Cortical	glow +	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/12	6/9	-1.50X90	Nil
211	Dundawwa	23572	55	F	RE	SIMC - PSC	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6	6/6	-0.25X120	Nil
212	Yallappa Chalwadi	23581	60	M	RE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/9	6/6	-1.00X120	Nil
213	Adiveppa	23579	72	M	LE	SIMC- PSC+ Cortical	glow +	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/36	6/18	-1.50X110	myopic fundus
214	Jainamma Girini	26539	50	F	RE	SMC	no glow	PL PR	SICS + PCIOL	DM Tear	CE,SK	SK	Nil	Nil	Nil	CF 1/2m	CF 1m	HM	Nil	DM detachment
215	Basalingawwa	26558	75	F	LE	SIMC- NS+Cortical	glow +	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12P	6/12P	-1.50X90	Nil
216	Iramma Kumbar	26564	63	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	DIOL,Ir	DIOL	Nil	Nil	Nil	6/18	6/12	6/12	-2.00X70	Nil
217	Tarabai Hadapad	26541	70	F	LE	SIMC- PSC+ Cortical	glow +	CF 1/2m	SICS + PCIOL	None	Nil	Nil	PCO	Nil	Nil	6/24	6/18P	6/18	-1.75X70	Nil
218	Neelamma Pirashetti	26562	70	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24	6/24	-0.75X150	ARM changes
219	Shantabai Harijan	26530	55	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/12	6/12	-2.50X90	Nil
220	Sayabanna Havalagi	26560	70	M	LE	SIMC-NS3	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/36	6/18	-1.50X90	ARM changes
221	Bhagawwa Chalwadi	26559	65	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/18P	6/12	-1.75X100°	Nil
222	Basappa Harijan	26563	60	M	LE	SMC	no glow	HM	SICS + ACIOL	PCR,VL	Ir	Nil	Nil	Nil	Nil	6/24	6/12P	6/12	-0.75X90	Nil
223	Sharanawwa Harijan	26531	65	F	LE	SIMC -PSC+ Cortical	glow +	CF 1m	SICS + ACIOL	PCR	Nil	Nil	Nil	Nil	Nil	6/24	6/24	6/18	-2.0x70	Nil
224	Radhabai Rajaput	26549	75	F	LE	SMC	no glow	PL PR	SICS + PCIOL	ID	Nil	Nil	Nil	Nil	Nil	CF 3m	6/24	6/12	-1.50X90	Nil
225	Katunabee Nadaf	26561	60	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/12	6/12	-1.50X70	Nil
226	Hanamantaraya	26550	60	M	LE	SIMC -NS3-4	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/6	6/9	6/9	-1.00X90°	Nil
227	Siddrama Badiger	26556	70	F	RE	SIMC- PSC+ Cortical	glow +	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/12	6/9	-1.00X90°	Nil
228	Mahadevi Gotagunaki	26565	59	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/18P	6/9	+1.50X180	Nil
229	Basalingawwa	26558	75	F	LE	SIMC-PSC+ Cortical	glow +	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12P	6/12	-1.50X90	Nil
230	Parvathi Madar	26570	70	F	RE	SIMC- NS2+PSC	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/24	6/12	-1.75X90	Nil
231	Parvati Mangali	26553	70	F	LE	SIMC- PSC +NS2	glow +	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/24	6/18	-1.50X90	Nil
232	Ganagabai Hachadad	26543	60	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/12	6/12	-1.50X100	Nil
233	Chidanand Ganiyar	26545	64	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/18P	6/12	-1.00X90°	Nil
234	Neelamma Biradar	26554	60	F	RE	SMC	no glow	PL PR	SICS + PCIOL	ID	CE	Nil	PCO	Nil	Nil	HM	CF 4m	CF 2m	Nil	Dry ARMD
235	Ramachandrappa	26533	70	M	RE	SMC	no glow	PL PR	SICS + PCIOL	PCR,VL	Endoph	PrUv	Nil	Nil	Nil	CF 2m	CF 2m	6/60	+8 sph	Nil
236	Sharanamma Hiremath	26532	69	F	RE	SIMC -Cortical	normal	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18P	6/9P	-1.00X90°	Nil

237	Sidappa Jambagi	26548	75	M	RE	SIMC- PSC+ Cortical	glow +	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24	6/18	-1.00X110	Nil
238	Bhimabai Chalwadi	26555	60	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/24	6/9	-1.50X80	Nil
239	Gangabai Hadapad	26551	61	F	LE	SIMC-PSC+ Cortical	glow +	CF CTF	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/12	6/12	-2.00X90	Nil
240	Iramma Kumbar	26534	65	F	LE	SIMC -Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	IP	Nil	Nil	Nil	6/24	6/24	6/12	-2.00X80	Nil
241	Bouramma Charashetti	26567	45	F	RE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/18	6/18	-1.25X90	ARM changes
242	Gurulingawwa Padasalagi	26537	60	F	RE	SMC	no glow	HM	SICS + PCIOL	None	Nil	Ir	Nil	Nil	Nil	6/12	6/18P	6/9	-1.00X90°	Nil
243	Mahadevi Biradar	26568	55	F	LE	SIMC- PSC+ Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/24	6/18	-2.00X80	Nil
244	Chanabasappa Angadi	27560	68	M	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/36	6/36	-2.50X90	ARM changes
245	Dundappa Desai	27555	65	M	LE	SIMC- PSC+ Cortical	glow +	CF 3m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/6	6/6	-1.00X80	Nil
246	Ningappa Angadi	27562	75	M	RE	SIMC- PSC+ Cortical	glow +	CF 3m	SICS + PCIOL	None	CE,SK,Ir	CE,SK	Nil	Nil	Nil	CF 6m	6/24P	6/18P	-1.50X90	Nil
247	Allabi Chapparband	27558	58	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/24	6/18	-2.00X90	Nil
248	Basawwa Humanabad	28638	80	F	RE	SIMC -PSC+ Cortical	normal	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/9	6/12	-1.00X120	Nil
249	Laxman Guled	28423	83	M	RE	SMC	no glow	CF 1/2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/60	6/18	6/12	-2.50X80	myopic fundus
250	Pandappa Hosamani	28404	60	M	RE	SIMC- NS3	glow +	CF 1m	SICS + PCIOL	None	CE	Nil	Nil	Nil	Nil	CF 4m	6/24P	6/12	-2.00X90	Nil
251	Kallawwa Madar	28751	70	F	RE	SIMC-PSC+ Cortical	glow +	CF 1m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18	6/12	-2.00X90	Nil
252	lachappa Babaleshwar	28434	65	M	RE	SIMC-PSC+NS2	normal	CF 3m	SICS + PCIOL	PCR	Nil	Nil	Nil	Nil	Nil	6/24	6/18	6/12	-0.50X120	Nil
253	Mallappa Danagond	28611	72	M	LE	SIMC-PSC+ Cortical	normal	CF 2m	SICS + PCIOL	None	Nil	PCO	PCO	Nil	Nil	6/24	6/18	6/18	-1.00X130	Nil
254	Yamanawwa Valikar	28418	62	F	LE	SIMC -Cortical	normal	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/18	6/12	-1.00X120	Nil
255	Siddanagouda Biradar	28438	53	M	RE	SIMC -PSC	glow +	CF 2m	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/12	6/6P	-0.50X60	Nil
256	Rukmawwa Babaleshwar	28406	50	F	LE	SMC	no glow	PL PR	SICS + PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/24	6/9	-1.00X90°	Nil
257	Lacchawwa Babaleshwar	28408	60	F	RE	SIMC-PSC	glow +	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/18P	6/18P	-1.25X100	Nil
258	Susalawwa Hosamani	28405	65	F	RE	SIMC-NS4	no glow	CF CTF	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/18P	6/9P	-1.00X90°	Nil
259	Sangappa	28409	75	M	RE	SIMC-NS3	no glow	CF 1/2m	SICS+PCIOL	None	SG	Nil	Nil	Nil	Nil	6/24	6/18	6/9P	-1.00X90°	Nil
260	Hanamanth Hebbalati	28410	60	M	RE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/9P	6/9	-1.75X120	Nil
261	Bapu Patel	28412	80	M	LE	SIMC-PSC+ Cortical	glow +	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/12	6/9	-1.50X90	Nil
262	Shayawwa Sharanawwa	28414	65	F	LE	SMC	no glow	CF CTF	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/12	6/9	+1.50X10	Nil
263	Hanamanth Hebbalati	28411	65	M	LE	SMC	no glow	CF CTF	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9P	6/9	-2.50X110	Nil
264	Ganagabai Biradar	28407	65	F	LE	SMC	no glow	CF CTF	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/36	6/12	-1.50X100	Nil
265	Hanamawwa Ranoji	28435	71	F	RE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	CF 1m	CF 1/2m	HM	Nil	Post staphyloma
266	Rayasab Dange	28631	65	M	RE	SMC	no glow	PL	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/24	6/12	-1.00x100°	Nil
267	Amtewwa	28436	60	F	RE	SIMC-NS3	glow +	HM	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/18	6/12	-2.50X90	Nil
268	Shantappa Hiremath	28425	77	F	LE	SIMC-NS+cortical	glow +	CF 3m	SICS+PCIOL	None	LM,SG	Nil	Nil	Nil	Nil	6/12P	6/18	6/12	-2.50X90	Nil
269	Mahadev Tengale	28699	70	M	RE	SIMC- PSC+ Cortical	normal	CF 3m	SICS+PCIOL	None	SG	Nil	Nil	Nil	Nil	6/24	6/9	6/6P	-1.50X110	Nil
270	Lalabee	29838	45	F	LE	SMC	no glow	HM	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/18	6/12	+2.00X180	Nil
271	Jettiagaraya Ilakal	29840	60	M	LE	SIMC - cortical	normal	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6	6/6	-0.75x80	Nil
272	Roobai Ramoji	29844	45	F	RE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/18	6/12	-1.00X70	Nil
273	Neelabai Bellundagi	29839	68	F	RE	SMC	no glow	HM	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/9	6/6P	-1.00X120	Nil
274	Mallanagoud	30421	70	M	LE	SIMC -PSC+ Cortical	glow +	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	+1.50X40	myopic fundus
275	Jateppa Kambali	30420	70	M	RE	HMC	no glow	PL PR	SICS+PCIOL	None	Ir	Nil	Nil	Nil	Nil	6/12	6/12	6/12	-5.00X110	ARM changes
276	Shivabai Kodabagi	150	70	F	LE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/9	-2.25X90	Nil
277	Sundrawwa Kambar	153	60	F	RE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18P	6/12	6/9P	-1.75X80	Nil
278	Indrabai Badiger	151	55	F	RE	SIMC- PSC+ Cortical	glow +	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/6	6/6P	6/6P	-1.75X90	Nil
279	Imambee Nadaf	155	60	F	RE	SIMC -NS+ Cortical	glow +	CF 1m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36	6/9	6/9	-1.25X90	Nil
280	Murthusab Walikar	811	60	M	RE	SMC	no glow	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/9	6/9	6/9	-0.50X100	Nil
281	Beeyamma Walikar	812	50	F	LE	SIMC -PSC +NS1	glow +	CF 2M	SICS+PCIOL	None	Ir	Ir	Nil	Nil	Nil	6/12	6/12	6/12	-1.50X90	Nil
282	Shankareppa Rathod	1434	50	M	LE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/9	6/9	-1.75X110	Nil
283	Shankareppa Rathod	1434	50	M	RE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/12	-2.50X90	Nil
284	Dundawwa Pujari	1432	60	F	RE	SIMC -PSC+Cortical	glow +	HM	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	-1.00x120	Nil
285	Mallawwa Biradar	1431	65	F	RE	SIMC - NS3	glow +	CF 2m	SICS+PCIOL	PCR	Uv,SG	OC	Nil	Nil	Nil	CF 3m	6/60	6/12	-1.50X50	Nil
286	Kamalabai Bise	1430	70	F	RE	SMC	no glow	PL PR	SICS+PCIOL	None	CE,SK	Nil	Nil	Nil	Nil	6/60	6/12	6/12	-2.50X60	Nil
287	Channappa Saasare	2064	65	M	RE	SIMC -NS2-3	glow +	6/60	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/6P	-1.00X90°	Nil
288	Mhadevappa Dalawai	2083	60	M	LE	SIMC-NS4	glow +	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12P	6/9	6/9	-1.00X90°	Nil
289	Shantabai Padaganur	2083	70	F	RE	SMC	no glow	HM	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/6P	6/6P	6/6P	-1.25X70	Nil
290	Balawwa Kambale	3406	70	F	RE	SIMC-NS2-3	glow +	CF 3m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/12	-1.25X90	Nil
291	Satirawwa Kalaburgi	3390	59	F	RE	SIMC-NS3	glow +	6/60	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24P	6/9P	6/9	-1.50X110	Nil
292	Padewwa Harijan	3398	65	F	LE	SIMC-PSC+ Cortical	glow +	CF 2m	SICS+PCIOL	PCR,VL	Nil	IP	Nil	Nil	Nil	6/12	6/18	6/12P	-1.50X80	Nil
293	Sayawwa Sarenavar	3404	60	F	RE	SIMC -PSC	glow +	CF 2m	SICS+PCIOL	None	CE,SK	Nil	Nil	Nil	Nil	CF 1m	6/36	6/24P	-1.25X90	Pale disc
294	Ningawwa Kambale	3402	60	F	LE	SIMC -NS2-3	glow +	CF 1m	SICS+PCIOL	PCR	Ir	Nil	Nil	Nil	Nil	6/24	6/18	6/12	-1.00X90°	Nil
295	Husenbi Mandapur	3407	65	F	RE	SIMC-PSC +Cortical	glow +	CF 2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/9	-1.00X90°	Nil
296	Darakabai Mole	4784	70	F	LE	SIMC -PSC	glow +	CF CTF	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	-1.75X90	Dry ARMD
297	Sayabawwa Pujari	4781	65	F	RE	SIMC- PSC	glow +	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/12	6/12	-1.00X90°	Nil

298	Bhimaraya Biradar	4786	70	M	RE	SIMC -PSC	normal	6/36	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	-1.75X120	Nil
299	Ramanna Jambagi	4783	60	M	RE	SIMC -PSC+ Cortical	normal	HM	SICS+ACIOL	PCR	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/12	-1.00X80°	Nil
300	Hanamanth Jambagi	6269	60	M	RE	SIMC -PSC	normal	6/60	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	-1.00x120	Nil
301	Yalappa Madar	6267	70	M	RE	SIMC- PSC+ Cortical	glow +	CF 3m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/12	6/12	-1.00X110	Nil
302	Shantabai Gadyal	7065	60	F	LE	SIMC -PCS+Cortical	glow +	6/60	SICS+ACIOL	PCR,VL	Nil	Nil	Nil	Nil	Nil	6/60	6/60	CF 1M	Nil	RD
303	Chandrashekar	7059	60	F	RE	SIMC -PSC	glow +	CF 3m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	-1.00X90°	Nil
304	Moulasab Yadgiri	7854	55	M	LE	SMC	no glow	PL PR	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36P	6/24	6/18	-1.50X110	ARM changes
305	Siddawwa Hugar	7849	65	F	RE	SIMC-Cortical	glow +	CF 3m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	-1.50X90	Nil
306	Ganabai Maranur	7848	S	F	LE	SIMC -PSC	glow +	CF 1/2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6P	6/6P	-1.00X80°	Nil
307	Kashibai Kotyal	7843	65	F	LE	SMC	no glow	HM	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/9	6/9	-1.75X90	Nil
308	Mahadevi Biradar	7856	60	F	RE	SMC	no glow	PL PR	SICS +PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/12	6/6	6/6	-1.75X120	Nil
309	Bibijan	7842	59	F	LE	SIMC-PSC+ Cortical	glow +	CF 1m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/24	6/12	6/12	-1.75X90	Nil
310	Sangawwa Math	7851	70	F	LE	SIMC-PSC	normal	CF 2m	SICS+ PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/6	6/6	-0.50X90	Nil
311	Tarabai Kulakund	9292	50	F	RE	SIMC-PSC+ Cortical	glow +	CF 3m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/18	6/9	6/9	-1.00X70	Nil
312	Siddawwa Pujari	9266	70	F	LE	SIMC -PSC+NS2	glow +	CF 1m	SICS+PCIOL	None	IP,Ir	Nil	Nil	Nil	Nil	HM	6/18	6/9	-1.50X100	Nil
313	Parvati Bilagi	9284	60	F	LE	SIMC-PSC+NS2	glow +	CF 2m	SICS+PCIOL	None	Nil	Nil	Nil	Nil	Nil	6/36P	6/12	6/12	-1.50X80	Nil