A REVIEW AND CLINICAL EVALUATION OF PER-OPERATIVE AND POST-OPERATIVE COMPLICATIONS IN CASE OF MANUAL SMALL INCISION CATARACT SURGERY AND EXTRACAPSULAR CATARACT EXTRACTION WITH POSTERIOR CHAMBER INTRA-OCULAR LENS IMPLANTATION

By

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Dissertation submitted Rajiv Gandhi University of Health Sciences, Karnataka for Partial fulfillment of the requirement for the degree of

MS in OPHTHALMOLOGY

Under the Guidance of

Dr.T.R.MANJULA

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Mysore

2006

DECLARATION

I here by declare that this dissertation entitled **"A REVIEW AND CLINICAL EVALUATION OF PER-OPERATIVE AND POST-OPERATIVE COMPLICATIONS IN CASE OF MANUAL SMALL INCISION CATARACT SURGERY AND EXTRACAPSULAR CATARACT EXTRACTION WITH POSTERIOR CHAMBER INTRA-OCULAR LENS IMPLANTATION"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.T.R.MANJULA,** Professor and Head of the Department of Ophthalmology.

(Dr. Kushal Banerjee)

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(Dr.Kushal Banerjee)

Date :

Place: B.G.Nagara

LIST OF ABBREVIATIONS

(in alphabetical order)

AC	Anterior chamber
ACIOL	Anterior chamber intraocular lens
ADP	Adenosine diphosphate
ATP	Adenosine tri phosphate
BSS	Balanced salt solution
CME	Cystoid Macular edema
DM	Decemet's Membrane
ECCE	Extra capsular cataract extraction
HEMA	Hydroxy ethyl methacrylic acid
ICCE	Intracapsular cataract extraction
IOL	Intraocular lens
IOP	Intra ocular pressure
NAD	Nicotinine adenine dinucleotide
NADP	Nicotinine adenine dinucleotide phoshate
NADPH	Nicotinine adenine dinucleotide phosphate hydrogenase
PC	Posterior capsule
PCIOL	Posterior chamber intraocular lens
PCO	Posterior capsular opacity
PGE	Prostaglandin E
PMMA	Polymethyl methacrylic acid
PPV	Parsplana vitrectomy
PVD	Posterior Vitreous detachment
RBB	retrobulbar block
RBH	Retrobulbar haemorrhage
RD	Retnal Detachment
RRD	Rhegmatogenous retinal detachment
SICS	Small incision cataract surgery
VA	Visual acuity
VL	Vitreous Loss

ABSTRACT

Background & Objectives

Cataract by far is the commonest cause of preventable blindness in India. Surgical replacement of the natural cataractous lens with an artificial Intra-Ocular-Lens (IOL) is the best possible way to rehabilitate a cataract patient, of which Extra-Capsular Cataract Extraction (ECCE) & Manual Small Incision Cataract Surgery (SICS) with Posterior Chamber IOL implantation (PC IOL I) are the most practiced cataract surgeries in our country presently. This study aims to review and clinically evaluate the per-operative and post-operative complications of the above mentioned two surgical procedures.

Methodology

100 patients, half of whom underwent ECCE and rest Manual SICS with PC IOL I in this hospital were followed up till 6 months from the date of surgery and managed for complications, if any. The data was used to interpret results.

Results

Cases consisted of 49 males and 51 females, most of the patients were in the age group 40-80. Majority of them had advanced immature and senile mature cataracts. Few per-operative, early and late post-operative complications occurred involving both the anterior and posterior segments. Most patients were managed medically, few required surgical interventions. Patients had relatively good visual outcome, better being in patients resolving to medical management than surgical treatment.

Interpretation & Conclusion

Ocular hypotony, strict asepsis, preserving corneal endothelial health and intact posterior capsule, proper iris tissue handling and ‘in-the-bag’ IOL I are of utmost importance to prevent complications. Prognosis depends on severity of complications, early diagnosis and prompt institution of appropriate management.

Key words : Cataract; surgery; ECCE; Manual SICS; PC IOL I; complication; prognosis.

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INTRODUCTION

Cataract can be defined in many ways, on an *anatomic basis* it can be defined broadly as any lens opacity on or inside lens, or *functionally* as only those lens opacities that interfere with vision.

According to some authors cataract is a structural, physical, bio-chemical and optical change in the crystalline lens of the eye that interfere with the normal transmission and refraction of light rays. This interference affects the overall sharpness of definition of the retinal image.

Cataract is the commonest cause of blindness in India. Blind population in our country is nearly 12 million, of which blindness due to cataract is 80.9%. In order to restore normal visual acuity the visual axis must be cleared.[1]

So far the only means known to clear the visual axis, is the surgical removal of the defective lens. The refractive power should be replaced and so far the IOL has been proved to be the best option.

It is well documented that the replacement of the natural cataractous lens with an artificial one is one of the best way to rehabilitate the cataract operation. [2,3,4]

The traditional cataract operation has many disadvantages such as aphakic correction by spectacles produces[5,6,7] :-

i. image magnification of about 25 to 30%,
ii. constriction of visual field,
iii. spherical aberration
iv. “jack in the box’ phenomenon,
v. ring scotoma,

vi. prismatic errors,

vii. lack of physical co-ordination,

viii. physical inconvenience and cosmetic deficiency of heavy spectacle lens.

A significant improvement for the aphakia came with introduction of contact lens because its :-

i) image magnifying effect is lesser,

ii) spherical aberration and distortion of images are eliminated.

However the contact lens have their drawbacks including :-

i) corneal vascularization,

ii) endothelial decompensation, corneal ulcer and

iii) even loss of eye may occur if sterilization of the lens is not maintained properly.

Contact lens is not the answer for all aphakias.[8] These disadvantages of aphakias are preventable if an artificial intraocular lens is implanted into the capsular bag following extra capsular cataract extraction & Manual small incision cataract surgery.

Though the history of IOL goes back to 18th century, the modern practice of IOL implant surgery started when Sir Harold Ridley inserted the first Posterior Chamber IOL in the left eye of a 45 years old lady after an ECCE at St. Thomas hospital London on 29th November, 1949. Ridley first got the idea when a medical student while observing him operating a cataract case asked him why he did not replace the cataractous lens which he had removed with a new one.

From Ridley's first lens implantation to the present day, the evolution of IOL can be arbitrarily divided into six generations :-

Table 1 : ***Evolution of Intra-Ocular Lenses***

Generation	Date	Description
I	1949-1954	Original Ridley PC IOLs
II	1952-1962	Early AC IOLs
III	1953-1975	Iris-supported lenses
IV	1963-1990	Intermediate AC IOLs
V	1975-1990	Improved PC IOLs
VI	1990 to present	Modern capsular PC IOLs & modern AC IOLs

In March, 1977 Shearing began with compressible lens which stretched out completely in the ciliary sulcus.[9] The posterior chamber lens offers several advantages. These advantages are optical, anatomical and physiological in nature. Patient acceptance is high, visual rehabilitation is rapid.

The extra capsular cataract extraction & Manual small incision cataract surgery with posterior chamber lens implantation are one of the best ways to rehabilitate the cataract patient, but has some pre-operative, per-operative & post-operative complications too, viz :-

i) posterior capsule rupture,
ii) zonule rupture with vitreous loss,
iii) hyphaema,
iv) cortical remnants,
v) iridodialysis,
vi) corneal oedema,
vii) Iridocyclitis,
viii) secondary glaucoma,
ix) dislocation of IOL,
x) cystoid macular oedema,
xi) retinal detachment and

xii) endophthalmitis etc.

The present work was conducted to study the common per-operative and post-operative complication and evaluation of extra capsular cataract extraction and Manual small incision cataract surgery with posterior chamber intraocular lens implantation at Adichunchanagiri Institute of Medical Sciences, B.G.Nagara in between 2004-2005.

AIMS AND OBJECTIVES

- To study the preoperative and postoperative complications of posterior chamber intraocular lens implantation.
- To evaluate the complications and thereby highlighting the causes of complications.
- To diagnose and manage the complications.
- Prevention of complications following evaluation of causes to achieve operative success.

REVIEW OF LITERATURE

All the different generations of IOLs have their own drawbacks & complications, viz:

First generation[10]- was experimented by Ridley in 1948 biconvex lens with diameter of 8.35 mm. wt. 17.4 mg in water refractive power + 24 .D.

The disadvantages were severe postoperative reactions high incidence of dislocation (13%). Glaucoma in 10%, Iris atrophy. The implant had to be removed in approximately 15 percent of the cases.

Second generation – lenses were anterior chamber angle fixated lenses. They had few advantages over first generation lenses like, minimal lens dislocation, secondary implantation could be performed: Implantation could be performed after an ICCE or ECCE. However two major complications were corneal decompensation and Glaucoma. Barraquer modified Dannheim's lens and created the first J loop IOL.

3rd Generation IOLs- Binkhorst in 1957 developed his Iris clip lens to circumvent the major problem of the preceding lenses. The lens was clipped to the iris which two anterior loops and two posterior loops extending behind the iris through the pupil. Complication like iris atrophy and papillary block glaucoma were more with these lenses. Fyodorov Sputnik, Worst Medallion lens, Worst Platina lens were the modifications of third generation lenses. [11,12,13.]

Fourth generation lens Choyce's anterior chamber one piece lens emerged as the fourth generation IOL in 1956. These lenses were excellent for ease of insertion and stability of fixation.

The Fifth generation- These are posterior chamber lenses initially modified by removing the posterior two loops of Binkhorst's four loop iris clip lens and placing retro-iridially after ECCE. But the real posterior chamber implant was made by Shearing (1977) who modified Barraquers anterior chamber lens by designing his flexible J- loop lens to rest against cilliary sulcus, the recess between the root of the iris and the anterior row of ciliary processes. Subsequently PC IOL, primarily intended for placement in the capsular bag have been designed- advantages are, it lies close to nodal point thereby reducing image magnification and anisokeinia, glare is eliminated as it is covered by iris, provides good pupil mobility, fundus view is superior, damage to corneal endothelium, trabecular meshwork erosion as well as large dislocation are practically nil .

Basically there are two types of IOLS, there piece- optic and haptics are made of different materials and single piece- optic and haptics made of the same material.

Materials for optics are PMMA, glass etc. Foldable IOLs are made of silicones, acrylics and hydrogels.

Materials for haptics are polyamides, polypropylenes, terepthelate polyethylene, glycolterepthelate polypropylene and PMMA.

John Graether was the first to design one piece all PMMA lens of 12.0 mm length designed for capsular fixation. Basic features of capsular IOL are one piece all PMMA IOLs having superior loop memory and modified C loop configuration is easy to insert with optimal loop conformity to capsular bag.

Lens is manufactured by three methods- lathe cutting, compression moulding and injection moulding. The optical quality is excellent by lathe cut method, it is time consuming.

Ridley first introduced the process of sterilization of IOL of immersion of IOL in 1 percent of quarternary ammonium base with tetra decyl trimethyl ammonium bromide. Gas sterilization method is now only permitted in United States. The gas used for sterilization (ethylene oxide) causes postoperative inflammation. Thermal sterilization (160^0C for 1 hr.) can only be used for glass lenses and silicone lenses can be exposed upto 2.5M rad without any significant alteration of its optical chemical or physical properties.

Before deciding the date of surgery with PCIOL, every patient should undergo a biometry examination to give calculation of implant power and corneal astigmatism in order to attain emmetropia after surgery.The simplest, most commonly used and most dependable formula used is SRK formula (Sanders DA, Retzleff J, Kraff MC).

$$P = A – 2.5 \times L – 0.9 \times K$$

Where P = IOL power to produce emmetropia in D

L = Axial length of the eye in mm

K = Average horizontal and vertical curvature of cornea in D measured by keratometry.

A = Specific constant for each lens type and manufacturer.

Another technique is based on basic refraction the refractive power prior to onset cataract where p = 19D+ (Rx1.25) p= Implant power R=basic refractive power.

Perfect predictability of IOL power is probably unattainable due to inherent limitations either in the instrument used or in its operations. The error may be in the determination of axial length of eye, in keratometry or due to A constant error. Some A constant published by one manufacturer show a total variation from 111.7 to 118.4 for the complete range of lens type and position.

ANATOMY OF LENS

The lens is a transparent, biconvex, crystalline structure placed in between iris and vitreous in a saucer shaped depression called *patellar fossa.*

The diameter of lens measures 9-10 mm and thickness 4-5m.m. The rim of the lens separating the anterior and posterior surfaces is called *equator.* Centre of anterior surface is known as the *anterior pole* and centre of the posterior pole is known as the *posterior pole.*

The anterior surface is less convex (radius of curvature 10mm) .The anterior surface is related to the posterior chamber and iris. The posterior surface is more convex (radius of curvature 6mm) and is related to the vitreous in hyaloid fossa and is attached in a circular area with *ligamentum hyaloideocapsulare (Weigner's ligament)*

Inside this circle, between the hyaloid face and the lens capsule is a small cavity or potential space called *retrolental space or Berger's space.*

Refractive index of human lens is 1.39 & Refractive power of human lens is about 16-17 Dioptres.

STRUCTURE OF THE LENS

1. **Lens Capsule**:- It is a thin, homogenous, transparent, highly elastic, hyaline collagenous membrane which is surrounds the lens completely. It is secreted at the embryonic stage as a basement membrane of lens epithelium (thickest basement membrane of the body). It is thicker anteriorly and at the equators than posteriorly.

2. **Lens Epithelium** : Anterior lens epithelium is single layer of cuboidal nucleated epithelial cells which lies deep to the anterior capsule. Almost all the metabolic, synthetic and transport processes of lens occur in this layer.

In *equatorial region* cells become *columner* and actively dividing and elongating to form new lens fibres throughout life.

There is no posterior epithelium, as these cells are used up in filling the central cavity of the lens vesicle during development of the lens.

The anterior lens epithelium is divided into 3 zones-

a. **Central zone**: consists of cuboidal cells, which are polygonal in flat section. Nuclei are round and located slightly apically.

b. **Intermediate zone** : comparatively smaller and more cylindrical cells, located peripheral to central zone. Nuclei are round and central.

c. **Germinative zone** : columner cells, most peripheral fibres. and located just pre-equatorial. Nuclei of these lens are flattened and lie in the plane of cell axis. These cells actively divide to form the new cells which migrate posteriorly to form new lens fibres.

3. **Cement Substance :** This is the intracellular amorphous substance in the lens.

4. **Lens Fibres** : Lens fibres are elongated, prismatic bands. They are composed of albuminoid material enclosed in a pseudo-membrane.

Formation : Epithelial cells divide, elongate and differentiate to produce long , thin, regularly arranged lens fibres. The superficial new fibres are nucleated and elongation of cells. Nuclei assume a relatively more anterior position. Thus anteriorly shifted nucleus forms a line convex forward at the equator, called *lens or nuclear bow.*

Structure : On cross section, lens fibres are almost hexagonal in shape and are bound together by the ground substance.

There are inter-locking process between cells (*ball & socket* and *tongue & groove interdigitation*) with *zonulae occludentis*.

Arrangement : Embryonic nucleus are arranged as they terminate with two 'Y'shaped sutures, anterior- upright Y and posterior- inverted Y shaped on the surface of the lens.

Zonular arrangement

a) **Nucleus** : Central and oldest part.

   i) Embryonic nucleus- innermost part, formed at 1-3 months of gestation.

   ii) Fetal nucleus - 3 months of gestation till birth

   iii) Infantile nucleus – birth to puberty

   iv) Adult – lens in adult life.

b) **Cortex** : Youngest and most recently formed lens fibres.

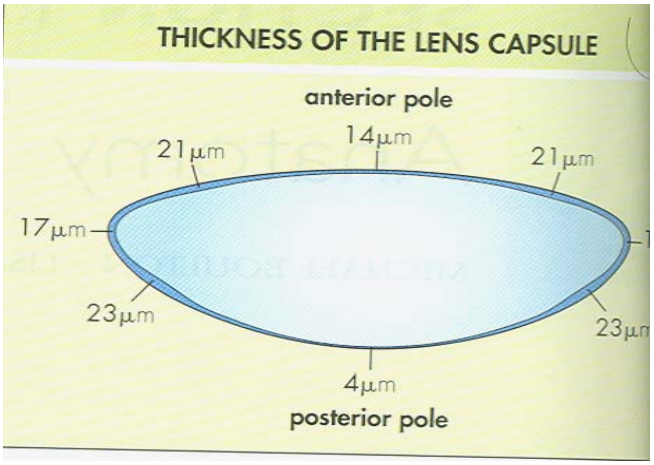

Fig 1: Lens capsule

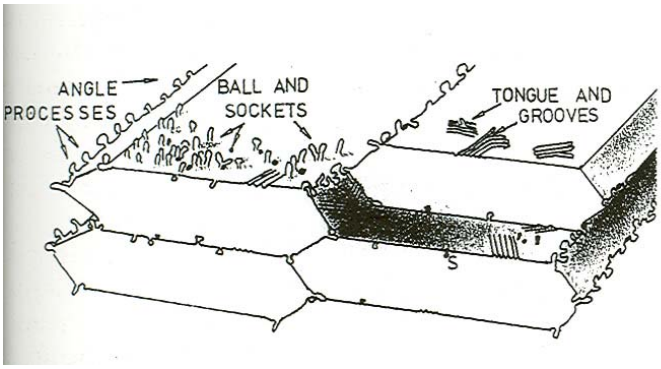

Fig 2 : Ball & socket and tongue & groove interdigitation

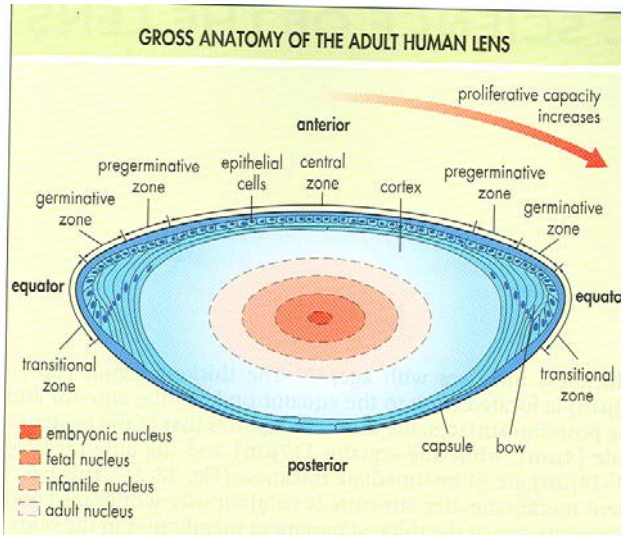

Fig 3 : Anatomy of Lens

PHYSIOLOGY AND BIOCHEMISTRY

Main constituents of lens are water and proteins. Water constitutes about 65% of lens wet weight.[14] Proteins constitute 34% of total weight of adult lens.

Lens water

- lens is a relatively dehydrated organ.
- cortex being more hydrated than nucleus.
- lens dehydration is maintained by an active Sodium pump that resides within the membrane of the cell of lens epithelium.

Proteins

- protein content is higher than any other organ in the body.
- Physical state of protein is important factor for maintainance of transparency of the crystalline lens.

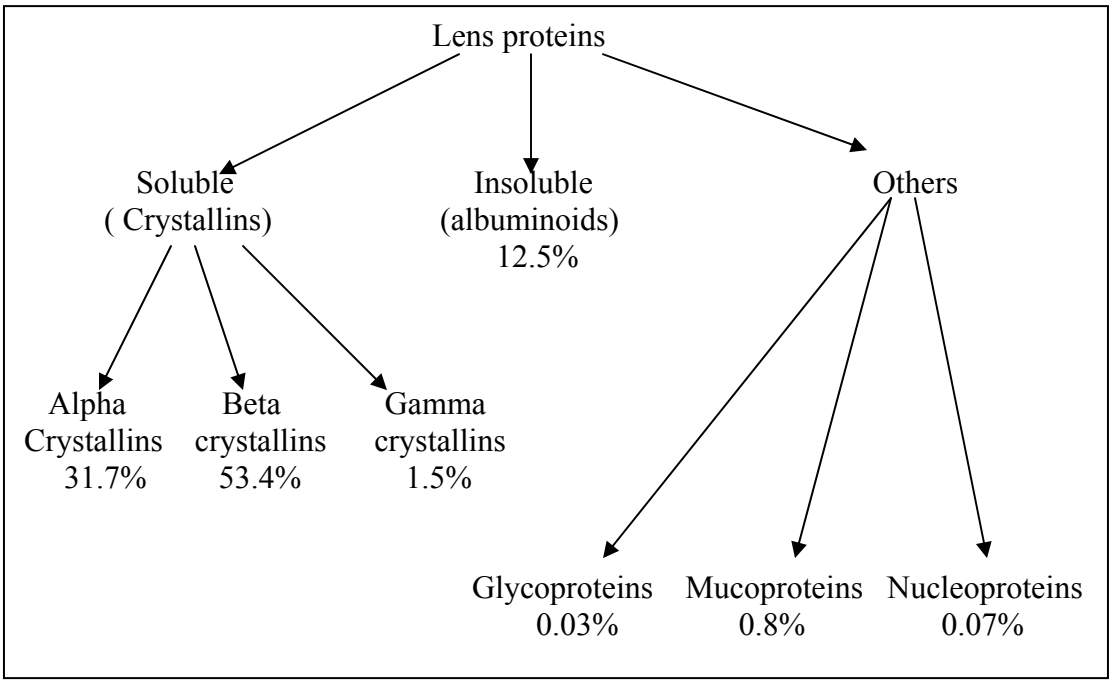

Fig 4 :- ***Lens Protiens***[15] -

Amino acids

Proteogenic	**Non proteogenic**
Alanine	taurine
Leucine	alpha amino butyric acid
Glutamic acid	ornithine
Aspartic acid	1-methyl-histidine
Glycine	3-methyl-histidine
Valine	homocarnosine
Phenyalanine	
Tyrosine	
Serine	
Isoleucione	
Lysine	
Histidine	
Methionine	
Proline	
Threonine	
Arinine	

Carbohydrates

Glucose	20 – 120 mg%
Fructose	
Glycogen	
Sorbitol	
Inositol	

Lipids

- Total lipids of human lens is 2.5% of the wet weight
- Cholesterol, phospholipids, cephalin, isolecithin, sphingomyelin, glycerides and lipoproteins.

Electrolytes

a. Potassium – predominant cation of the lens,114 – 130 mEq/kg of lens matter.
b. Sodium – 14 – 25 mEq/kg of lens water
c. Calcium - .14µg/mg dry weight of human lens
d. Anions – Chloride, bicarbonate, Phosphate – predominant, Sulphates

Glutathione – 200 – 450 mg/ 100 gm of lens gamma glutamyl cysteinyl glycine

Ascorbic acid - 30mg/ 100 gm wet weight of lens .

METABOLIC ACTIVITES OF LENS[16]:-

Glucose metabolism

- Anaerobic glycolysis
- Kreb's cycle
- HMP shunt
- Sorbitol pathway

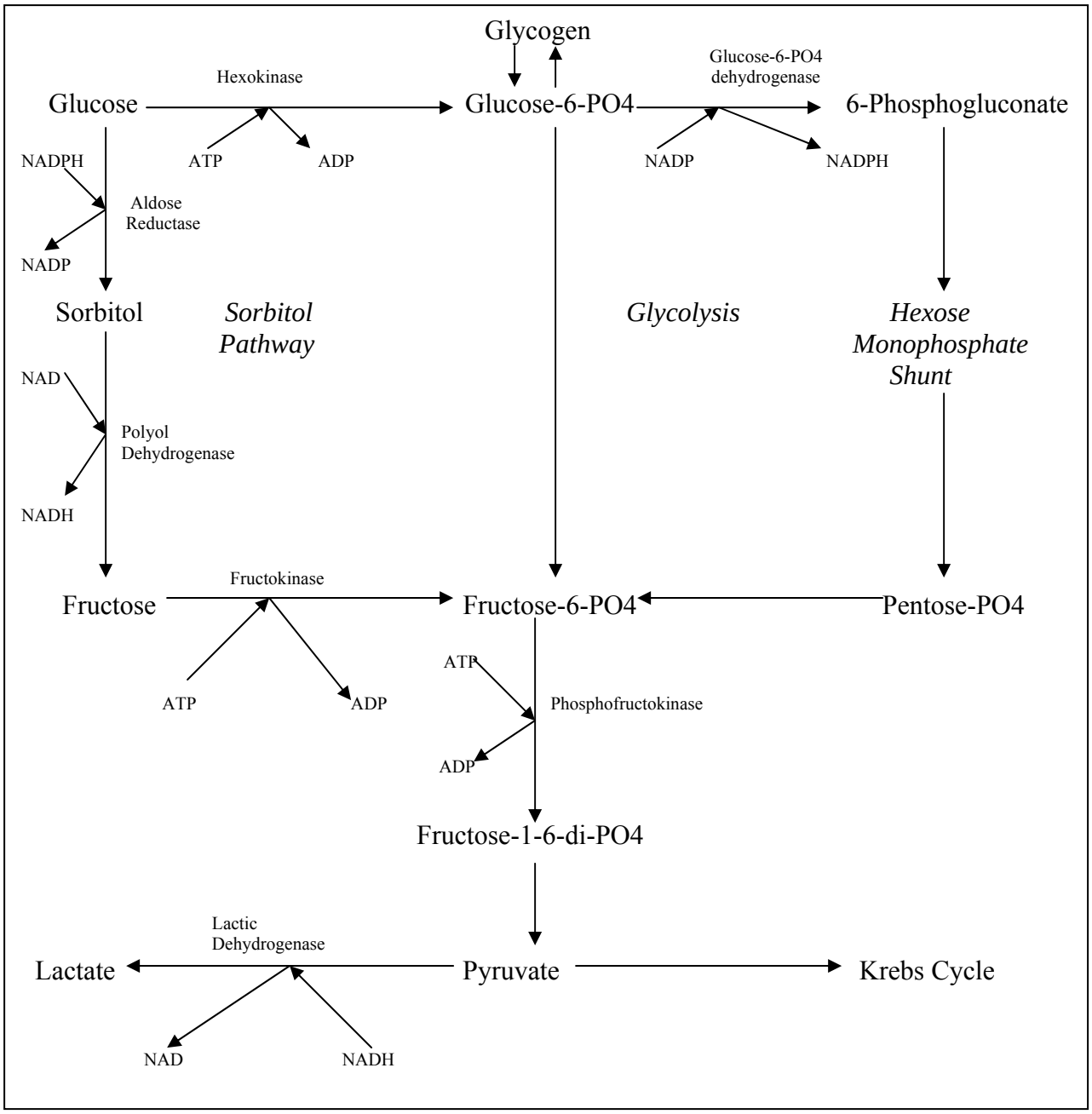

Fig 5 :- Glucose Metabolism

Protein metabolism

- Protein synthesis
- Protein breakdown

PERMEABILITY AND TRANPORT MECHANISM OF LENS[17]:

Active and Passive Transport (permeability dependent)

- provide nutrients for metabolism
- dispose of waste products of metabolism
- regulate water and cation balance in the lens.

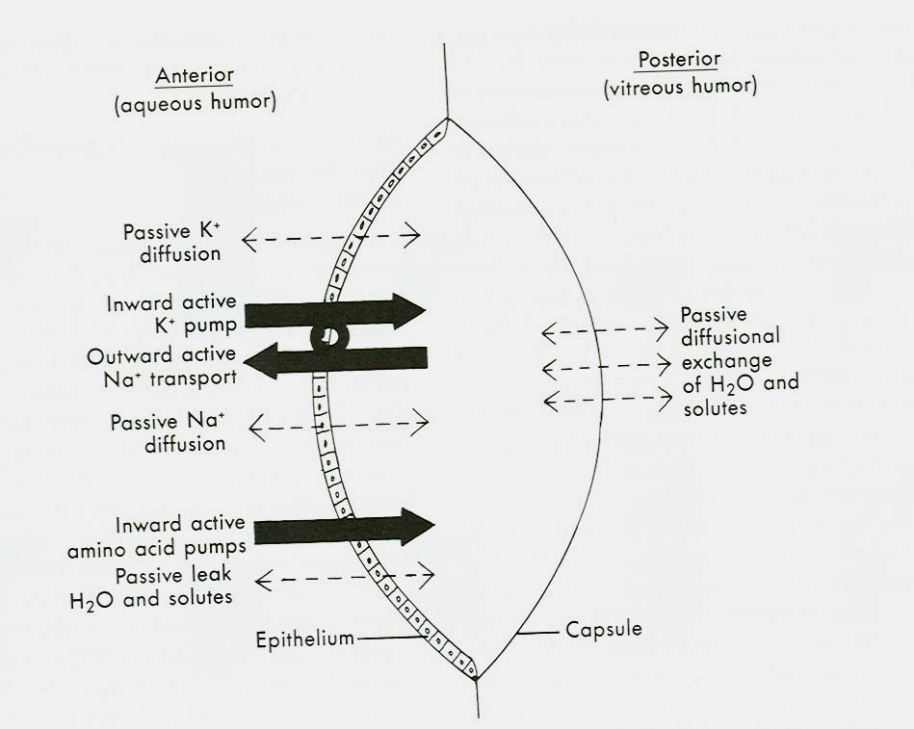

Fig 6 :- Transport mechanism of Lens -

1. Water and electrolytes transport:

- Energy dependent cation pump
- at the level of anterior lens epithelium
- active extrusion of Sodium coupled with uptake of K^+ mediated by membrane bound enzyme Na^+-K^+-ATPase which degrades ATP into ADP, inorganic phosphate and energy.

- As a result- chemical gradient is generated, which stimulates diffusion of Na^+ into the lens and K^+ out of the lens, mainly through the posterior surface and little through anterior surface.
- This process of active transport (cation pump) stimulating passive diffusion (leak)- is termed "*pump-and-leak*" theory of cation transport.

2. Transport of amino acids and inositol:

- working in "pump-and-leak concept
- 3 different pumps one each for acidic, basic and neutral amino-acid.

3. Glucose transport

Simple diffusion and facilitated diffusion through both anterior and posterior surface of lens.

LENS TRANSPARENCY

- Transparency of lens presumably depends on the avoidance of large transitions of refractive index between cells and surrounding cement substance.
- Torkel [18] proposed that lens transparency is due to the regular arrangement of lens fibres and uniform distribution and paracrystalline state of proteins with in the cell.
- Jones and Lerman[19] reported that the lamellar conformation of lens proteins rather than helical structure also contribute to transparency.
- Factors maintaining transparency of lens

a) tightly packed nature of the lens cells.

b) arrangements of lens protein.

c) regulation if electrolyte and water balance in the lens.

CHANGES IN AGEING LENS

1) Physical changes

a) lens weight and thickness- increases steadily

b) light transmission- decreases at lower wavelength.

c) Light absorbance- increases with age.

d) Light scattering- increases with age, because of ***SYNERISM***- conformational changes to protein release bound water enhancing the difference in refractive index between the 'driver ' protein region and its surroundings.

e) Fluorescence- increases with age.

f) Refractive index- increases with age.

2) Metabolic changes

a) proliferative capacity- declines in adult life.

b) Enzyme activities- declines with age.

c) Increased urea soluble proteins at the expense of soluble proteins.

d) Glutathione and ascorbate levels decreases with age.

e) Super-oxide dismutase and G-6-P-D activity- lost with age.

3) Changes in Crystallins

a) by using recent fast high performance gel chromatography.

b) Alpha-crystallins- almost disappears.

c) Beta-crystallins- more polydisperse.

d) Gamma-crystallins- increased in di-sulphide bonds with age.

e) Non-tryptophan fluorescence- increases with age.

4) Changes of plasma membrane and cytoskeleton:-

a) loss of hexagonal cross-section of fibres and their inter-locking devices.

b) Age related loss of membrane proteins and lipids

c) Loss of membrane potential and increase in lens Na^{+} & Ca^{++} with age .

d) All large membrane polypeptides decreases with age.

e) Main junctional polypeptides-converted to smaller variants with age.

f) Changes in membrane rigidity.

CATARACTOGENESIS

SENILE CATARACT

Risk Factors Affecting Onset, Type And Maturation Of Senile Cataract-

1. Heredity

2. Exposure to ultra-violet irradiation- more exposure- early onset and maturation of senile cataract. U-V radiation between 290-320 nm induce lens opacification.

Activated O2 or H2O2 causes crystalline aggregation and pigmentary changes.

1) Dietary factors

- Jacques et-al (1988) found
- apparent protective effect, associated with high blood levels of carotenoids, precursors of Vit-A, but not for Vit-A itself, more powerfully for Vit-D.
- Selenium- risk factor.

4. Severe diarrhea

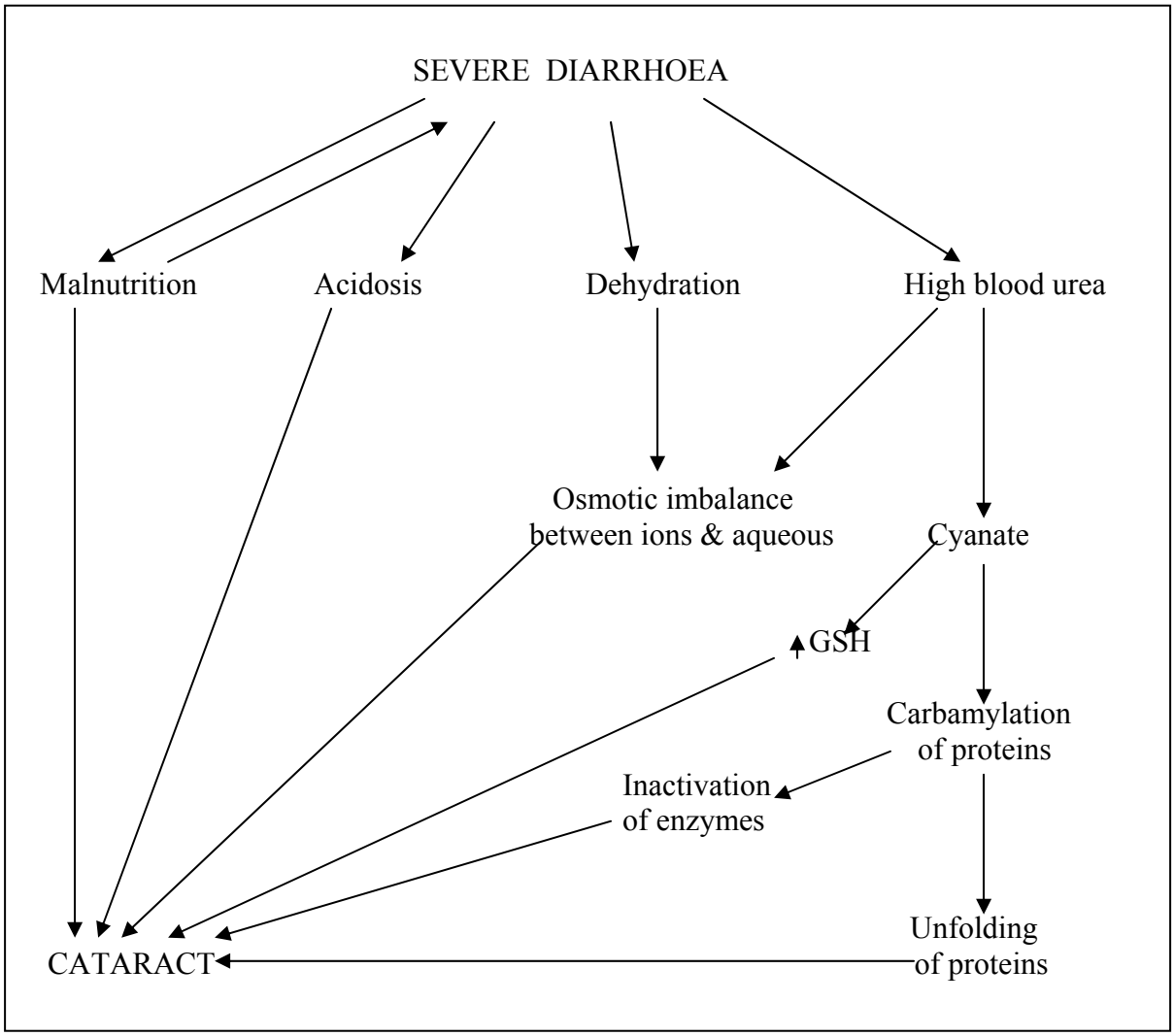

Fig 7 :- Catarataractogenesis in severe diarrhea

5. Diabetes- causes more rapid maturation of cataract, but may not affect initiation of cataract.
6. Renal failure- powerful risk factor
7. Hypertension and diuretics
8. Myopia
9. Miscellaneous- smoking; alcohol use; glaucoma; steroid use.

Table 2 : Changes observed in Age-related Cataracts with Progression

	Changes
Structural Proteins	
1. Protein fluorescence	↑
2. Water-insoluble protein fraction	↑
3. Urea-insoluble protein fraction	↑
4. Disulphide-bonded high molecular weight protein aggregates	↑
5. Cross-linked by non-disulphide bonds	↑
6. Soluble gamma fraction	↓
7. Protein-bound glutathione	↑
8. Oxidation of methionine	↑
9. Oxidation of cysteine	↑
10. Protein thiol	↓
11. Aspartyl racemization	↑
12. Covalent changes in membrane proteins	↑
Enzymes	
1. Glutathione S-transferase	↓
2. Superoxide dismutase	↓
3. Glutathione peroxidase	↓
4. Proteolytic enzymes activities	↑
Low Molecular Weight Components	
1. Ca^{++}	↑
2. Na^{+}	↑
3. Inositol	↓
4. Glutathione	↓

B) Biochemical Changes and possible mechanism of loss of lens transparency

1) CORTICAL CATARACT

a. Water content of lens- Normal is 65%; it increases with maturation of cortical cataract from immature (68-70%) to hyper mature (78-80%).

b. Protein content of lens- Normal is 34%; decreases with maturation of cortical senile cataract, while there is an increase in the water insoluble fraction of protein.

Possible mechanisms

- Leakage of low molecular weight proteins from lens to the surrounding tissue.
- Conversion of soluble protein into insoluble proteins.
- Decreased synthesis of lens proteins.
- Increased protein catabolism.

c. **Free amino acids** : A progressive decrease in the level of total free amino acids occur with the maturation of cortical cataract, attributed to leakage from disrupted lens membrane.

d. **Sodium and Potassium**: Sodium content of lens progressively increases and Potassium levels are decreased.

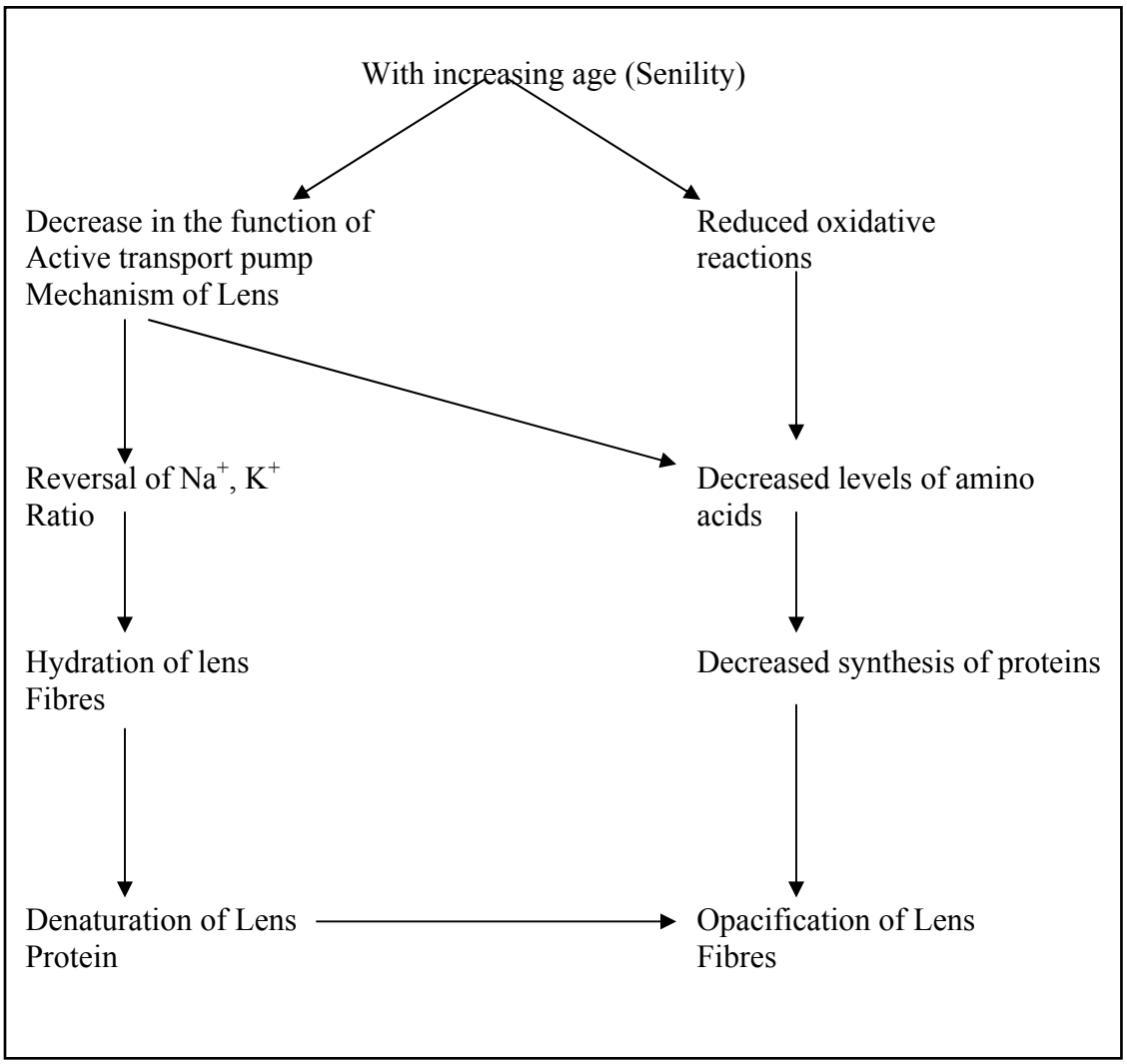

Fig 8 : Mechanism of development of Senile Cataract

e. Calcium- Calcium content increases with maturation of cataract.

Stages		**Calcium content**
Normal lens	-	1mg%
Incipient	-	<10mg%
Immature	-	10-35mg%
Mature	-	>35mg%

2) NUCLEAR CATARACT

- degenerative changes are intensified with age.
- related nuclear sclerosis associated with dehydration and compaction of nucleus, resulting in hard cataract.

C) Role of glutathione and acorbic acid in cataractogenesis :

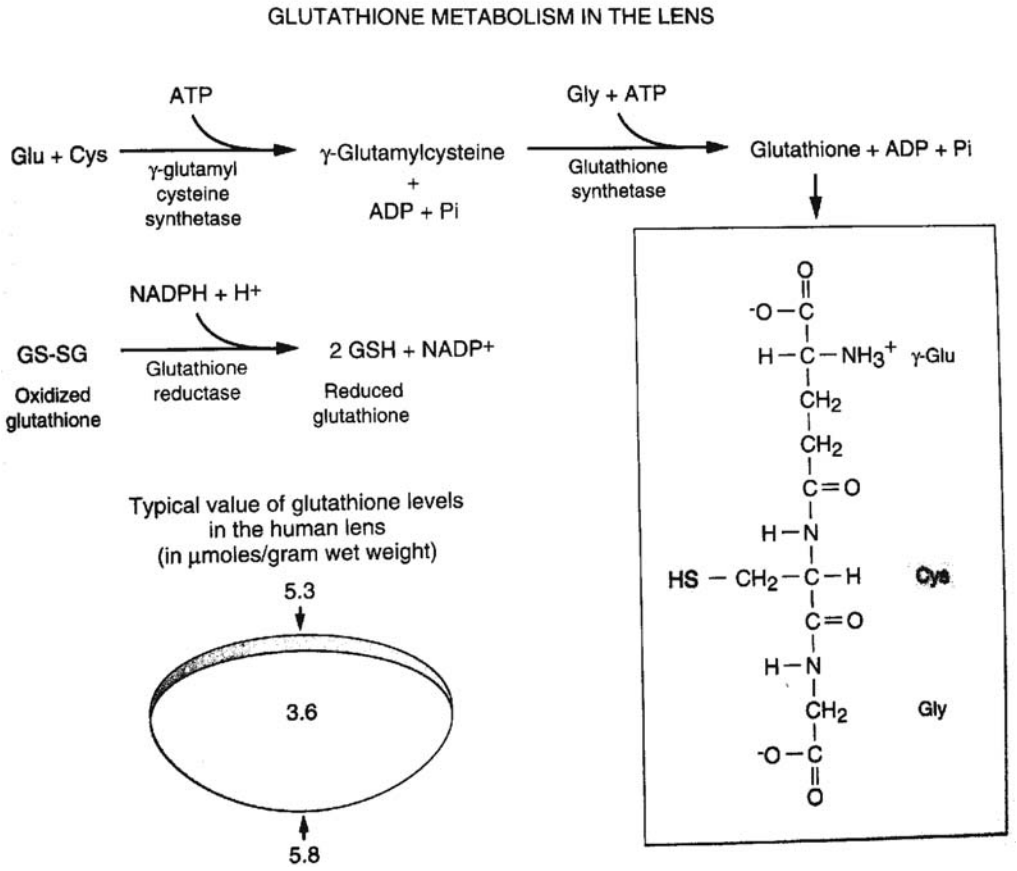

Fig 9 : Glutathione Metabolism in Lens

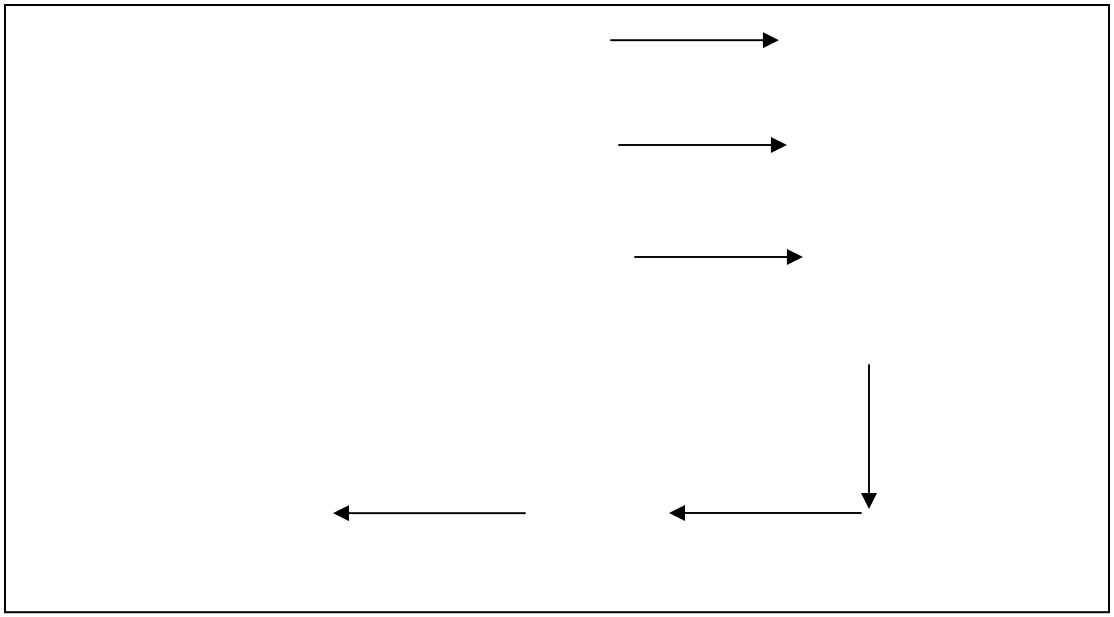

Fig.10 - Key enzymes active in defence against oxidative insults and xenobiotics

Oxidants viz:- H2O2, super-oxide anions, hydroxyl radicals, if not disposed, because of decreased concentration of Ascorbic acid and Glutathione, causes oxidation of protein hydrals viz- enzymes, crystallins and membrane proteins.

- Super-oxide radicals are most toxic to lens protein.
- Glutathione, Ascorbic acid and Vit-E prevents this damage.
- decreased Glutathione, Ascorbic acid and Vit-E concentration leads to cataractogenesis.
- Factors contributing to fall in the level of Glutathione in catarctous lens-

i. decrease in synthesis- reduced Glutathione is non-permeable and aqueous has very little Glutathione.

ii. increased permeability of lens membrane to reduced glutathione.

iii. relative deficiency of enzyme *glutathione reductase* and increased *mixed di-sulphide* formation with protein sulphydryls.

SUGAR CATARACT

- Cataracts associated with galactosemia, juvenile diabetes mellitus and adult diabetes mellitus.
- Galactosemic cataract
    a) Classical Galactosemia- deficiency of GUPT.
    b) Related- deficiency of Galactokinase (GK)
- bilateral cataract => ***OIL DROPLET CENTRAL LENS OPACITIES***.
- True Diabetic cataract- ***SNOW FLAKE / SNOW STORM CATARACT.***

PATHOGENESIS

1. Sorbitol, Aldose reductase and osmotic hypothesis Mainly due to *ALDOSE REDUCTASE* deficiency.
2. Autoxidation of sugar hypothesis
3. Theory of non-enzymatic glycosylation (glycation)

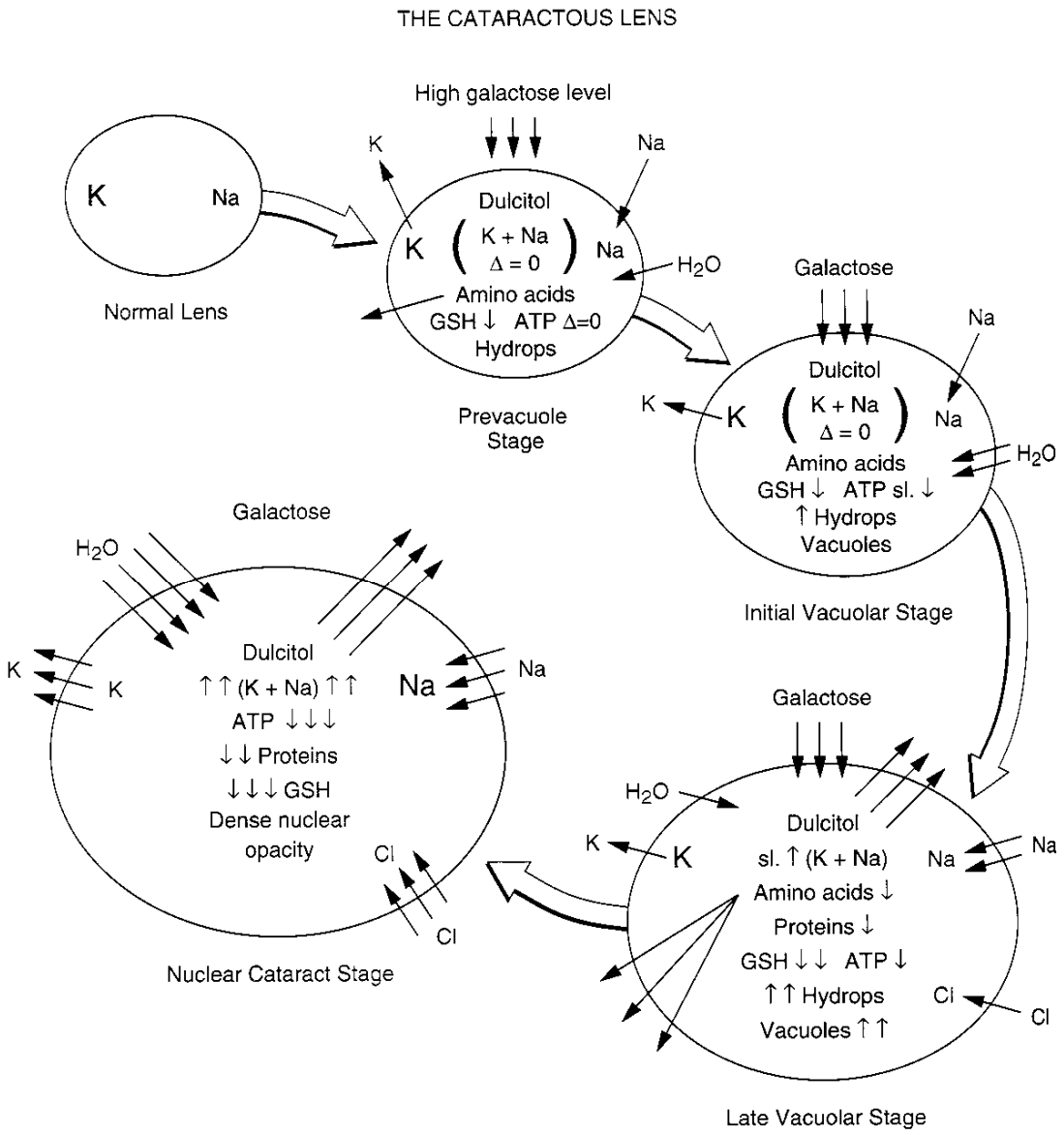

Fig 11 :- Mechanism of development of Sugar Cataract

RADIATION CATARACT

Radiant energy produce cataract by causing damage to the germinative zone of lens epithelium.

Infra-Red (Heat) Cataract

- prolonged exposure - discoid posterior subcapsular opacity.
- ***glass-blowers and glass-workers cataract.***

IRRADIATION CATARACT

Prolonged exposure to ionizing radiation viz- X-rays, gamma-rays or neutrons.

MECHANISM OF IRRADIATION CATARACT [20] :

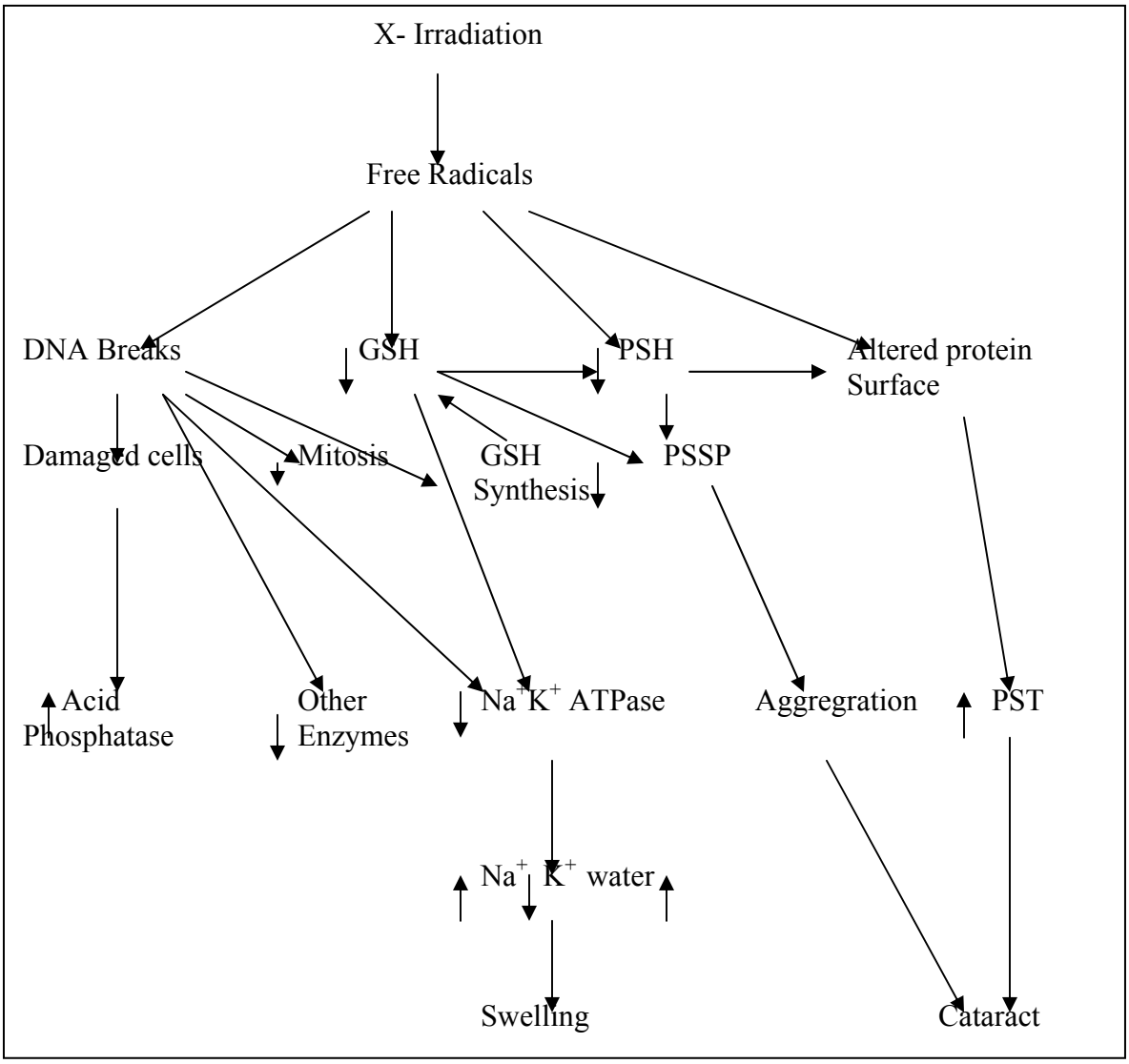

Fig 12 : Mechanism of Radiation Cataract

CORTICOSTEROID INDUCED CATARACT

- Posterior sub-capsular opacities with prolonged use of topical and systemic steroids
- Steroids may induce aberrant differentiation and migration of epithelial cells leading to the posterior opacification.
- Cataract is induced by any of the following mechanisms.

    i. elevation of glucose

    ii. inhibition of Na^{+}-K^{+}-ATPase

    iii. increased cation permeability

    iv. inhibition of G-6-P-D

    v. inhibition of RNA synthesis

    vi. loss of ATP

    vii. covalent binding of steroids to lens protein.

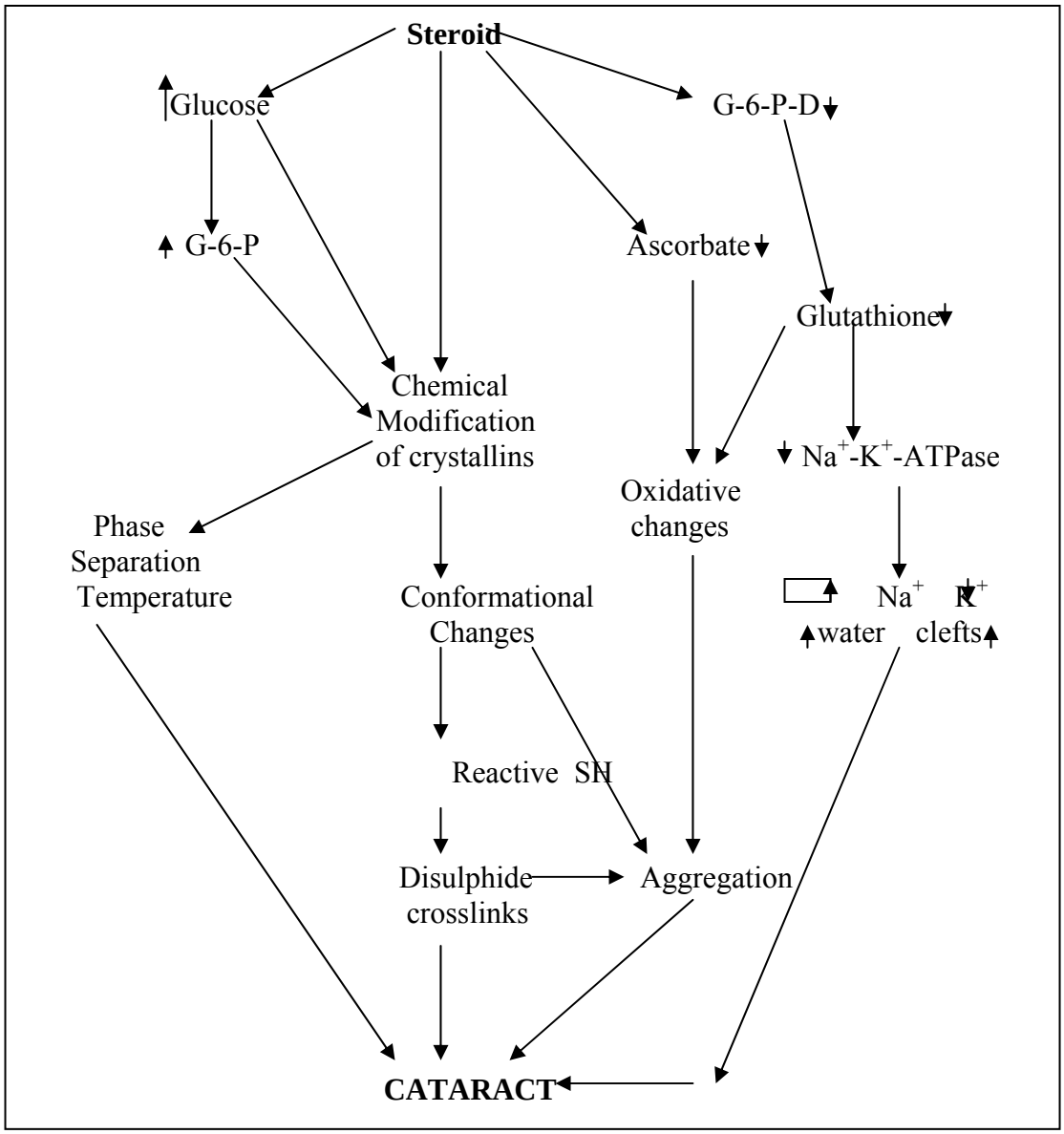

Fig 13 :- Mechanism of Steroid induced Cataract -

CLASSIFICATION OF CATARACT

a) ETIOLOGICAL CLASSIFICATION

i) Congenital and Developmental Cataract

ii) Acquired Cataract

1. Senile Cataract
2. Traumatic Cataract
3. Complicated Cataract
4. Metabolic Cataract
5. Electric Cataract
6. Radiational Cataract
7. Toxic Cataract
    - i) Corticosteroid induced
    - ii) Miotics induced
    - iii) Copper (Chalcosis) and Iron (Siderosis) induced
8. Dermatogenic Cataract
9. Cataract associated with osseous diseases
10. Cataract with miscellaneous syndromes
    - i) Dystrophica myotonica
    - ii) Down's Syndrome

b) MORPHOLOGICAL CLASSIFICATION:-

1. Capsular Cataract

i) Anterior capsular

ii) Posterior capsular

2. Subcapsular Cataract
    - i) Anterior subcapsular
    - ii) Posterior subcapsular
3. Cortical Cataract
4. Supranuclear Cataract
5. Nuclear Cataract
6. Polar Cataract
    - i) Anterior polar
    - ii) Posterior polar

Best mode of treating cataract is by surgical removal of cataractous lens and replacement by artificial Intra-ocular-lens.

Table 3 : 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	United States	Kelman J.Shock
1975	Iris-pupil supported IOLs	Netherlands	Binkhorst Worst
1984	Foldable IOLs	United States South Africa	Mazzocco Epstein

of these ECCE & Manual SICS are the most commonly practiced cataract surgeries in India at present.

Like any other surgical procedure, the above mentioned two varieties of cataract surgeries also have some complications.

Common Pre-operative, Per-operative & Post-operative complications encountered after ECCE & Manual SICS cataract surgery are:-

I. PRE-OPERATIVE COMPLICATIONS

1. Anxiety
2. Nausea & gastritis
3. Irritative or allergic conjunctivitis

Anaesthesia Related

4. Retro-bulbar Haemorrhage (RBH)
5. Globe trauma
6. Injury to optic nerve
7. Corneal abrasion
8. Sub conjunctival haematoma & chemosis
9. Spontaneous dislocation of lens
10. Acute Anaphylactic reaction

II. PER-OPERATIVE COMPLICATIONS

1. Patient movement
2. Superior rectus muscle laceration
3. Thermal burn
4. Irregular incision
5. Tunnel perforation
6. Corneal complications
7. Hyphaema
8. Iridodialysis

9. Iris sphincter damage
10. Iris tuck
11. Extension of Anterior Capsulotomy
12. Posterior Capsule tear
13. Positive vitreous pressure (up- thrust)
14. Subluxation or Dislocation of lens
15. Zonular dialysis
16. Vitreous loss
17. Expulsive haemorrhage

III. POST-OPERATIVE COMPLICATIONS

A) EARLY (with in 1 week)

1. Striate keratopathy & Corneal oedema
2. Flat & shallow Anterior chamber
3. Hyphaema
4. Iris prolapse
5. Retained lens matter
6. Post-operative Uveitis
7. Post-operative Glaucoma
8. Bacterial Endophthalmitis
9. Pupillary block
10. Haptic incarceration
11. Lens precipitate
12. Fibrinoid reaction
13. Malposition

B) LATE (weeks to months)

1. Posterior Capsule Opacification (PCO)
2. Cystoid Macular Edema (CME)
3. Epithelial downgrowth
4. Fibrous ingrowth
5. Filtering bleb formation

6. Unpredictable Astigmatism
7. Persistant chronic Uveitis
8. IOL related complications
9. Retinal Detachment
10. Bullous Keratopathy
11. Late post-operative Endophthalmitis
12. Late onset Hyphaema
13. Pupillary membrane

I. PRE-OPERATIVE COMPLICATION

1. ANXIETY

- Some patient develop anxiety on the eve of operation due fear & apprehension of operation.
- Anxiolytics & Sedatives given.

2. NAUSEA & GASTRITIS :-

- due to pre-operative medications as Acetazolamide, glycerol.
- oral antacids are given.

3. IRRITATIVE OR ALLERGIC CONJUNCTIVITIS

- due to pre-operative topical antibiotics & its preservative.
- operation postponed till allergy subsides.

4. RETRO-BULBAR HAEMORRHAGE (RBH)

- more common after ***RETRO-BULBAR BLOCK (RBB)***
- earliest signs
  - tense immobile globe that gives firm resistance to ***retropulsion.***
  - taut eyelids

- diffuse sub-conjunctival haemorrhage
- ecchymosis of lids

- ***direct & in-diret ophthalmoscopy*** performed to asses the status of ***CENTRLRETINAL ARTERY.***

- Treatment
- goal is to maintain patency of Central Retinal Artery during increased pressure
- postpone the surgery.
- immediate 2% pilocarpine + pressure bandage.
- topical Beta-blocker , IV Carbonic Anhydrase inhibitor, Hyperosmotics
- lateral canthotomy
- anterior chamber paracentesis
- direct orbital pressure to stop active bleeding.

GLOBE TRAUMA

- sudden softening of the eye & collapse of posterior chamber -- Hallmarks of globe trauma.

Table 4 : Risk factors to Perforation of the eyeball-

a) Long eye, axial length > 26 mm
b) patients with axial myopia have 30 times greater risk
c) Posterior staphyloma
d) Enophthalmos
e) Faulty technique
f) Un-cooperative patient
g) Unnecessary long needles
h) No appreciation of risk factors

Treatment

- Surgery postponed
- refer to vitreo-retinal specialist.

6. INJURY TO OPTIC NERVE

- more common with long needles & RBB
- sudden loss of vision (black out) & oculo-cardiac reflex.

Treatment

- surgery postponed.
- refer to vitreo-retinal surgeon.

7. CORNEAL ABRASION

- care should be taken while doing Schiotz tonometry, pre-operative preparation of eye with Betadine, lash cutting, post-anaesthetic massaging, draping, & putting lid speculum.
- postpone surgery with antibiotic ointment & patching for 2 days.

8. SUB-CONJUNCTIVAL HAEMATOMA & CHEMOSIS

- observed after excess & repeat aneasthetic solution injection.

9. SPONTANEOUS DISLOCATION OF LENS

- patient with
  - weak & degenerated zonules
  - hypermature cataract &
  - pseudo-exfoliation syndrome , are more prone
- mainly due to vigorous massaging after giving anaesthesia.
- Treatment
  - surgery postponed
  - refer to vitreo- retinal surgeon.

10. ACUTE ANAPHYLACTIC REACTION :-

Table 5 : Neuro- Ophthalmic Reflexes

Reflex	Afferent Pathway	Efferent Pathway	Precipitating Factors	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 center & 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	

II. PER-OPERATIVE COMPLICATION

1. PATIENT MOVEMENT

common in 1st time operable patient

apprehensive & anxious patient

patient who sleep off on the operation theatre (OT) table

Prevention Anxiolytics & sedatives before surgery

taping patients forehead with the OT table

2. SUPERIOR RECTUS MUSCLE LACERATION

- due to direct damage to the muscle while putting the ***Bridal suture***
- very rarely causes post-operative Ptosis if Levator muscle complex also gets damaged.

3. THERMAL BURNS

- due to excessive cauterization to prevent bleeding while making conjunctival flaps.
- inadvertent corneal burns might occur.
- post-operative contracture of burnt tissue & excessive conjunctival cutting may hinder future glaucoma surgery.

4. IRREGULAR INCISION

Placement

- supero-temporal & temporal incisions are better considering future filtrating glaucoma surgery.
- should be exactly sclero-corneal, at the limbus in ECCE,

- more corneal incision -- more astigmatism
- more scleral incision -- more iris prolapse
- smile incision in Manual SICS -- best wound healing & water sealing in nature.

Depth

- too deep incision will lead to entry into suprachoroidal space or premature entry into anterior chamber.
- former causes bleeding & hypotony and the latter leads to iris prolapse.
- if suprachoroidal space is entered, deep Radial sutures are put.

Inadequate

- during IOL insertion, leading haptic often planes upwards towards the cornea, thus - difficult to reach inferior fornix of capsular bag
- corneal endothelial damage & Descemets membrane stripping
- increased posterior tear vitreous loss.

5. TUNNEL PROBLEM

- if too thin flap bucket handle tear occurs.
- if too deep premature entry into anterior chamber , leads to
- shallow anterior chamber
- hypotony
- increased iris prolapse
- increased chances of corneal endothelial damage & Descemets detachment.
- increased chances of posterior capsule tear & vitreous loss (VL)
- wound doesn't become self-sealing.
- Temporary sutures are put in outer lip of the tunnel.

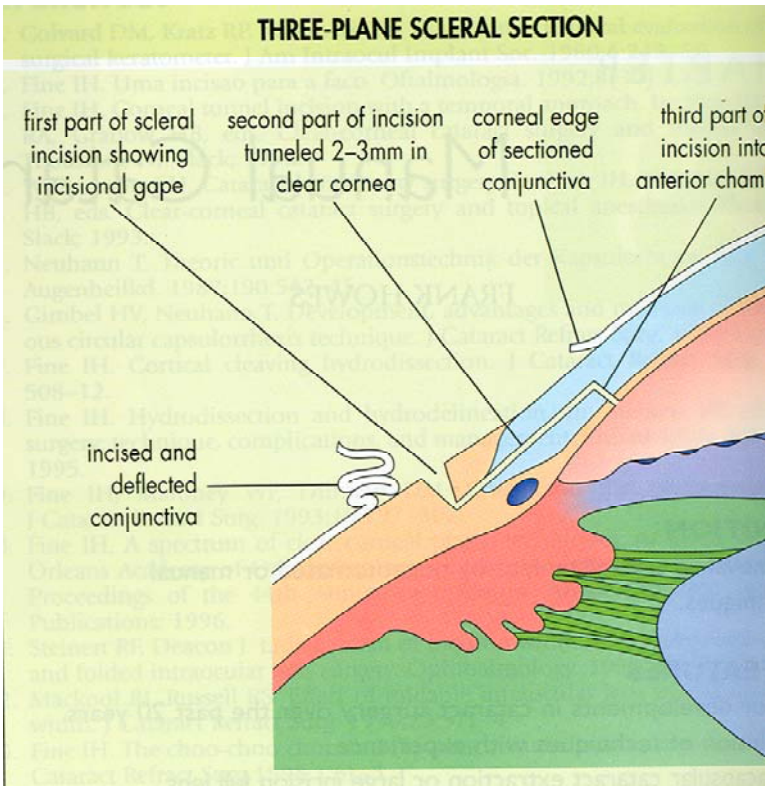

Fig 14 :- Three plane tunnel for Manual SICS

- if too small inability to deliver nucleus & difficulty in IOL insertion, leading to compromised corneal & posterior capsule (PC) conditions.

6. CORNEAL COMPLICATIONS

A) STRIPPING OF DESCEMET'S MEMBRANE

- occurs while entering sharp instrument in Anterior Chamber, like Keratome, Simcoe's irrigation-aspiration cannula or during IOL insertion through inadequate incision.

- Pathological findings as Descemet's Membrane contains contractile myofilaments, it curls on itself after getting detached.[21] So detachment looks like transparent sheet curling inwards from corneal lip of incision.

- may cause corneal oedma with cellular & fibrous infiltration and anterior synaechia by adhering to iris.

- If < 20% of Descemet's membrane (DM) is detached, tamponade of air bubble is given beyond the detached inwardly curled tip of DM.[22,23]

- if >20% of DM is detached -- 10-0 nylon suture is placed to adhere back DM to position

- Penetrating Keratoplasty (PK)

B) ENDOTHELIAL TRAUMA

- normal endothelial cell count in adults - 2400-3000 cells / mm^2
- increased proneness if endothelial cell count < 500 cells / mm^2
- by direct contact with nucleus or IOL if maneuvered in shallow AC
- ample use of visco-elastic material to keep AC formed & deep during surgery.

7. HYPHAEMA

- serious complication, as it impairs surgeons view.
- Potential complication of blood in AC
    - premature miosis intra-operatively.
    - delayed or incomplete miosis at the end of surgery.
    - increased risk of correctopia.
    - pupillary capture
    - IOL decentration

- PCO post-operatively
- increased duration of post-operative inflammation

Treatment

- avoid deep, temporal & posterior incision
- direct wet field coagulation by point cauterization.
- adding epinephrine in Balanced Salt Solution (BSS)+irrigation solution
- epinephrine injection intracamerally.
- increasing height of infusion bottle.
- washing out of retained blood.
- air bubble tamponade.

8. IRIDODIALYSIS

- occurs during enlargement of incision or during IOL implantation.
- Problems with large iridodialysis
  - small pupil
  - diplopia
  - pupil not aligned with IOL optic
  - IOL haptic loop prolapse
  - Iridodonesis
  - excessive pseudophakodonesis with corneal touch.
  - increased CME
  - ***Iris Retraction Syndrome***.

Treatment

- small iridodialysis -- left alone

- large iridodialysis -- Mc-Cannel's suture [24]
- suturing the border of dialyzed iris to the posterior margin of incision wound.

9. IRIS SPHINCTER DAMAGE

- most commonly caused during delivery of hard nucleus or IOL implantation through poorly dilated pupil, resulting in iris sphincter rupture & atonic pupil.
- Causes of small pupil
  - long term use of miotic therapy
  - pseudo-capsular exfoliation
  - senile miosis.
- Managed by
  - pre-operative topical NSAIDs viz, flurbiprofen or intra-operative epinephrine in infusion flow maintains pupillary dilatation.
  - Other methods
  - Radial iridotomy through peripheral iridectomy or through pupil.
  - Multiple sphincterotomies.
  - sector iridectomy
  - sphincter stretching
  - iris retractors.

10. IRIS TUCK

- entrapment of peripheral iris tissue in the angle of haptic feet or loop of IOL, occurring when the IOL is inserted across AC.
- More common with AC IOL
- oval pupil with vertical axis corresponding to vertical axis of IOL is pathognomic of iris tuck

Treatment

- IOL should be retracted & re-inserted correctly.
- use of adequate visco-elastic substance to keep AC deep.

- release of tucked iris tissue by iris repository.

11. EXTENSION OF ANTERIOR CAPSULOTOMY

a) Radial tear & peripheral extension

Due to - Anterior bowing of lens iris diaphragm.

- shallow AC
- increased elastic forces of zonules.
- increased intralenticular pressure.
- decreased scleral rigidity.
- insufficient incision
- significant posterior pressure (up-thrust)
- high pressure in irrigation - aspiration bottle
- widely dilated pupil.

Managed by

Deepening AC by injecting ample visco-elastic material.

Followed by two options

i. return to origin of capsulorrhexis to create a second continuous circumferential capsulorrhexis in opposite direction of original tear.

ii. use ***multiple puncture, can opener technique*** to connect the remaining un-connected portions of the capsulotomy.

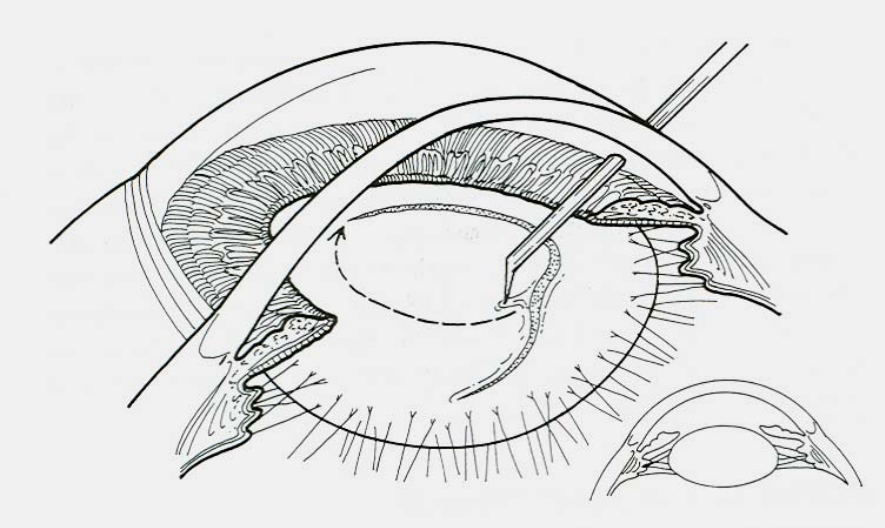

Fig 15 : Extension of Anterior Capsulotomy

b) Small capsulectomy

- more common in young patient as superior portion of capsule is most difficult to enlarge.
- new tear is started by making an oblique cut with vannus scissors in desired direction & make a larger capsulorrhexis.

12. POSTERIOR CAPSULE (PC) TEAR

- can occur during nucleus delivery
- cortical clean-up
- IOL insertion
- more common in
- posterior polar [25] & subcapsular cataract.
- young patient with congenital cataract
- brunescent cataract
- morgagnian cataract
- small pupil
- pseudo-exfoliation syndrome

if associated with vitreous loss, it increases the chances of

- vitreous induced pupillary block
- CME
- Retinal Detachment (RD)

Prevented by

- use of ample visco-elastic to maintain the AC deep during the surgery.
- limited hydrodeliniation & no hydrodissection.

Managed by

- if VL is there
- removal of all the vitreous from the AC by anterior vitrectomy followed by ***'dry tap'***[26]
- converting linear PC tear to continuous posterior capsulorrhexis, thus reducing chances of extension.
- minimal & gentle maneuvering inside AC
- placing IOL with haptic orienting at 90 degrees away from axis of tear or placing 'in-the-bag' IOL in the sulcus by reducing the power by 0.5D[27]

13. POSITIVE VITREOUS PRESSURE (UP-THRUST)

occurs with

- external compression of globe
- excessive volume of retro-bulbar & peri-bulbar anaesthesia
- poorly designed & improperly placed lid speculum
- obese patient
- any valsalva maneuver by patient
- excess traction by Bridal suture of superior rectus muscle.

14. SUBLUXATION OR DISLOCATION OF LENS

- either the lens nucleus or IOL can subluxate or dislocate posteriorly into the vitreous through an undiagnosed PC tear or zonular dialysis.

Managed by [28]

- liberal extension of incision & removed bi-manually
- rapid creation of sclerotomy 3mm posterior to limbus & passage of supporting spatula to prevent further dropping , followed by removal.
- refer to vitreo-retinal specialist for pars-plana removal.

AC IOL optic dislocating posteriorly, passes behind iris & causes '*Internal Iris Prolapse*'

15. ZONULAR DIALYSIS

Risk factors

- Marfan's Syndrome
- Homocysteinuria
- Weill- Marchesani Syndrome
- Microspherophakia
- Pseudo-exfoliative syndrome [29]
- phacodoneis
- iridodonesis
- visible vitreous strands in AC
- lens subluxation
- unequal AC depth

Acquired zonular dialysis - occurs during

- traumatic extension of capsulotomy
- excessive maneuvering of nucleus

- aspiration of anterior or posterior capsule with irrigation-aspiration cannula
- excess hydrodissection or hydrodeliniation.
- IOL insertion

Treatment

- minimal & gentle maneuvering inside AC
- IOL insertion in capsular bag as long as anterior capsular ring is intact & zonular dialysis is < 4-5 clock hours.
- perpendicular IOL placement prevents post-operative capsular contracture leading to IOL decentration.
- IOL stability & centration is checked by ***'bounce-back-test'***
- PMMA or Silicone made ***endocapsular ring*** may be used.
- if zonular dialysis is large-- alpha-chymotrypsin is instilled to detach the entire capsular bag & its contents, followed by scleral fixated or AC IOL implantation.

16. VITREOUS LOSS

- occurs following accidental PC rupture or zonular dialysis.

Prevention

- Decreased vitreous volume - by 20% mannitol
- Decreased aqueous volume - 500mg of oral Acetazolamide
- Decreased orbital volume -- proper post- anaesthetic massage
- Better ocular akinesia & anaesthesia
- Minimizing external pressure on eyeball by not using speculum or releasing Bridal suture of superior rectus muscle
- Fleiringa ring to prevent scleral collapse
- planned posterior sclerotomy with vitreous drainage from pars-plana.

Detected by

- gentle pulling of dry cellular sponge after placing at scleral incision, demonstrates viscous strands of vitreous adhering to it because of ***' spinbarkeit effect'***
- dilatation of pupil

Treatment

- all remnant cortical vitreous should be removed from AC & scleral incision wound
- partial anterior vitrectomy followed by dry tap
- complete vitrectomy by using automated vitrector
- previously distorted pupil will restore back to normal , round & aspiration through vitrectomy probe easens up.

Complication

- vitreous incarceration
- impaired wound healing
- epithelial downgrowth
- fibrous ingrowth
- corneal stromal oedema secondary to vitreo-corneal touch
- iritis
- glaucoma
- vitreous haemorrhage
- CME
- Retinal Detachment
- ***proliferative vitreo-retinopathy***.

17. EXPULSIVE HAEMORRHAGE

- expulsive choroidal haemorrhage is most catastrophic complication of cataract surgery occurring due to rupture of posterior ciliary arteries [30]

Pathogenesis

- Sudden hypotony secondary to surgical decompression of globe , commonly after delivery of lens leads to rupture of blood vessels.

Risk factors

- advanced age
- brunescent cataract
- pre-existing uveitis
- glaucoma
- high myopia
- systemic hypertension
- patient under anti-coagulant therapy
- atherosclerotic heart disease
- acute rise of systemic BP intra-operatively
- intrinsic vascular fragility
- H/o expulsive haemorrhage in fellow eye.

Signs

- chamber shallowing with positive pressure is the 1st sign
- surgeon sees gradual loss of red reflex through pupil as a dark mass steadily increasing in size
- patient complains of pain despite good anaesthesia
- globe suddenly becomes firm
- spontaneous gaping of wound followed by expulsion of intra-ocular contents, initially iris, then lens, vitreous eventually retina & finally a gush of bright red blood.

Management

- immediate cancellation of surgery & closure of wound
- digitally closing the wound while giving bolus of IV mannitol
- suturing of wound with 7-0 suture, followed by reposition of prolapsed uveal tissue
- deepening of AC with air tamponade
- drain choroidal haemorrhage via posterior sclerotomy 3.5-4mm posteror to the limbus
- repeated B-scan & indirect ophthalmoscopy to rule out large, choroidal detachment & ***'kissing choroiditis'***

Prognosis

- very poor, most of the times the eye is lost & evisceration is to be done.

III POST-OPERATIVE COMPLICATION

A. Early Post-Operative Complication:-

1. STRIATE KERATOPATHY & CORNEAL OEDEMA

Mainly due to Descemet's Membrane folds.

Table 6 ***:Causes of Corneal oedema after Cataract Surgery***

1. Surgical trauma -
- Instrumentation
- IOL
- Irrigating solution
- Ultrasonic vibrations
- Nuclear fragments
- Prior surgery

2. Primary corneal endothelial disease -
- Fuch's dystrophy
- Low endothelial cell density without guttae

3. Chemical injury -
- Preservatives in solution
- Residual toxic chemicals on instruments (e.g , detergents, dried solutions)
- Improper concentrations of solutions (e.g, antibiotics)
- Osmotic damage
- Direct toxicity
- Mistakenly used toxic chemicals, expired agents or incorrect solutions (e.g, normal saline instead of balanced salt solution)

4. IOL Syndromes -
- Direct endothelial touch
- Long term toxicity (inflammatory)

5. Contact with other ocular tissues -

- Flat anterior chambe
- Iris bombe
- Suprachoroidal effusion & haemorrhage

6. Detachment of Descemet's Membrane -

7. Trauma from retained foreign material -

- Nuclear chips
- Particulate matter

8. Post-operative Glaucoma -

9. Inflammation -

10. Membranous ingrowth or downgrowth -

- Epithelial downgrowth
- Fibrous ingrowth
- Endothelial proliferation

11. Vitreous touch adherence -

- absence of IOL & capsule

12. Brown- McLean Syndrome

Late onset corneal oedema associated with AC IOL often preceeded or accompanied by CME => **Cornea-Retina-Inflammatory Syndrome (CRIS)**

Table 7 : Treatment of Corneal Oedema

1. Eliminate causes

- Treat inflammation
- Lower intra-ocular pressure
- Remove tissue-IOL contact
- Reattach Descemet's membrane

2. Enhance surface dehydration -

- Evaporate
- Hypertonic agents

3. Treat pain

- Lubricants
- Soft contact lenses
- Cautery of Bowman layer
- Conjunctival flap

4. Restore anatomy -

- Penetrating Keratoplasty

2. FLAT & SHALLOW ANTERIOR CHAMBER:-

- Surgical emergency, as irreversible corneal damage may result.

Table 8 : - **Causes of Flat or Shallow Anterior Chamber**

	Malignant Glaucoma	Serous Choroidal Detachment	Pupillary Block	Suprachoroidal Haemorrhage	Wound Leak
Onset	Intra-operatively or any time thereafter	Within 1st post-operative week	Early or late post-operatively	Intra-operatively or within the 1st week	Within the 1st post-operative week
Anterior chamber	Shallow or Flat	Shallow or flat	Shallow or flat	Shallow or flat	Shallow or flat
Intra-ocular pressure	Normal or elevated	Low	Normal or elevated	Normal or elevated	Low
Fundus	No choroidal detacthment	Smooth, light brown choroidal elevations	Normal	Dark brown or red choroidal elevation	Choroidal detachment may or may not be present
Patent iridectomy present	Yes	Yes	No	Yes	Yes
Relief by iridectomy	No	No	Yes	No	No
Relief by suprachoroidal fluid drainage & anterior chamber reformation	No	Yes	No	Yes	No

Wound leak is tested by ***Siedel;s test***.

Treatment is by

a. Immediate pressure bandage for 2 days.

b. Medical treatment to reduce intra-ocular pressure (IOP)

c. Repair of wound leak

d. Argon Laser treatment for cyclodialysis.

e. *Mc-Cannel suture* to close cyclodialysis cleft.

3. HYPHAEMA (EARLY)

Fresh blood in Anterior chamber (AC) originates from

- cataract incision
- iridectomy
- pupillary sphincter tear.
- If associated with Uveitis & Glaucoma, it is called ***Uveitis-Glaucoma-Hyphaema (UGH) Syndrome.***
- it is usually self-limiting & absorbs by 1st week.
- Medical treatment of decreased IOP, if –IOP > 40mm of Hg
- IOP > 30mm of Hg for 2 wks
- pre-existing glaucomatous optic nerve damage or sickle-cell-disease.

Indications for Surgical removal of blood from AC

- Corneal blood staining
- IOL blood staining
- clot staying for prolonged duration >10 days.

- IOP- > 50 mm of Hg for 5 days
- > 35 mm of Hg for 7 days
- total hyphaema for > 5 days

Surgical treatment

- AC washout with/without co-axial irrigation aspiration.
- Automated cutting-aspiration of clot material
- Clot expression.

4. IRIS PROLAPSE

- Prolonged iris prolapse is one of the commonest source of Acute Endophthalmitis.
- Mainly due to faulty suturing in ECCE or improper wound construction in Manual SICS.[31]

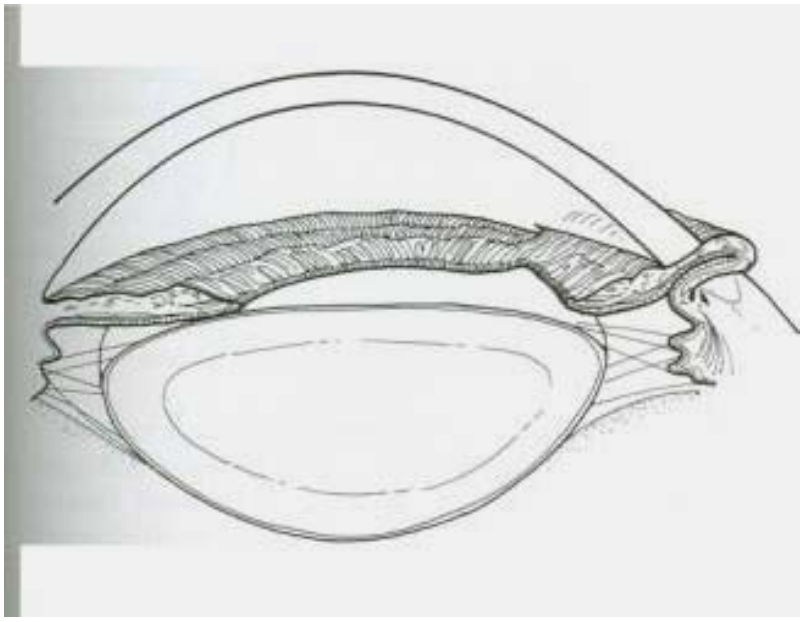

Fig 16 : Mechanism of Iris Prolapse

5. RETAINED LENS CORTICAL MATTER

- Due to improper washing during cataract surgery, there is retained lens cortical matter.
- It causes- decreased Visual Cavity (VA)
- Pupillary block glaucoma
- Uveitis
- Increased PCO formation

Treatment

- Steroids
- AC wash by paracentesis

6. POST-OPERATIVE UVEITIS:

- Contributing factors
- Delicacy of surgery
- Type of cataract surgery
- IOL location
- method of IOL sterilization
- Lens impurities
- ***AC IOL + UGH Syndrome*** => ***<u>ELLINGSON'S SYNDROME</u>***.
- Plastic IOL if dry, drags impurities and predisposes uveitis.
- *Ethylene Oxide* used for steilization also causes uveitis.
- large breach in *blood- aqueous- barrier* occurs.

Treatment

- frequent use of corticosterois
- cycloplegics & sympathomimetics

Contraindication

- Miotics.

7. POST-OPERATIVE GLAUCOMA: [32]

Table 9 : Causes of Glaucoma after cataract surgery -

Open - Angle Glaucomas	
- Primary open-angle glaucoma	- Dislocated nuclear fragments
- Blood induced glaucomas	- Corticosteroids
Hyphaema	- Viscoelastics
Ghost cell glaucoma	- Nd: YAG laser capsulotomy
- Uveitis	- Vitreous in anterior chamber
- UGH syndrome	- Cyclodialysis cleft closure
- Lens particle	- alpha-Chymotrypsin
Closed - Angled Glaucomas	
- Preexisting angle closure glaucoma	
- Pupillary block	
- Malignant glaucoma	
- Neovascular glaucoma	
- Epithelial / fibrovascular ingrowth	

Blood induced - **Ghost Cell Glaucoma**.

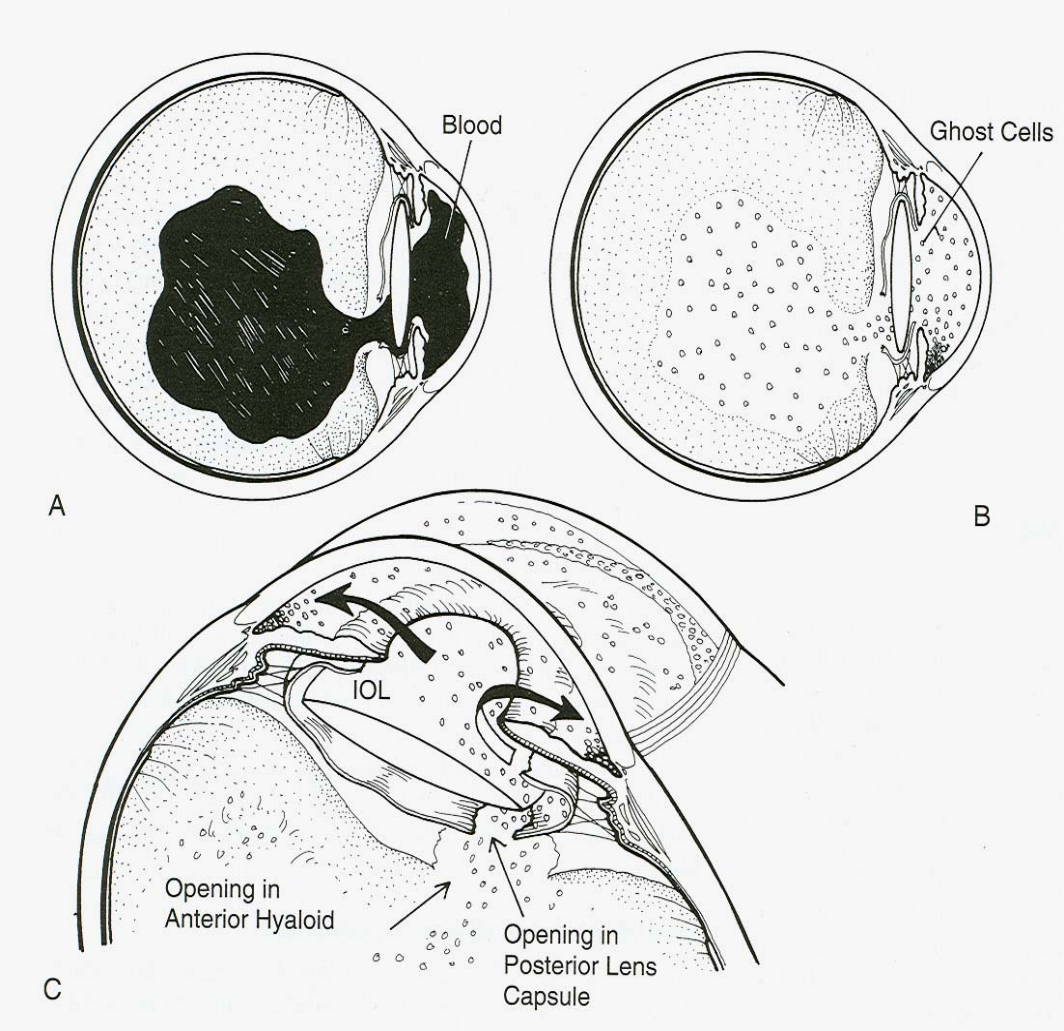

Fig 17 :- Mechanism of Ghost cell Glaucoma -

***Uveitic glaucoma*, due to**

- swelling of Trabecular matrix
- endothelial cell dysfunction
- accumulation of inflammatory cells & debris

Lens particle glaucoma

- lens matter causes severe obstruction of trabecular outflow channels.

Malignant Glaucoma or **Posterior Aqueous Diversion-**

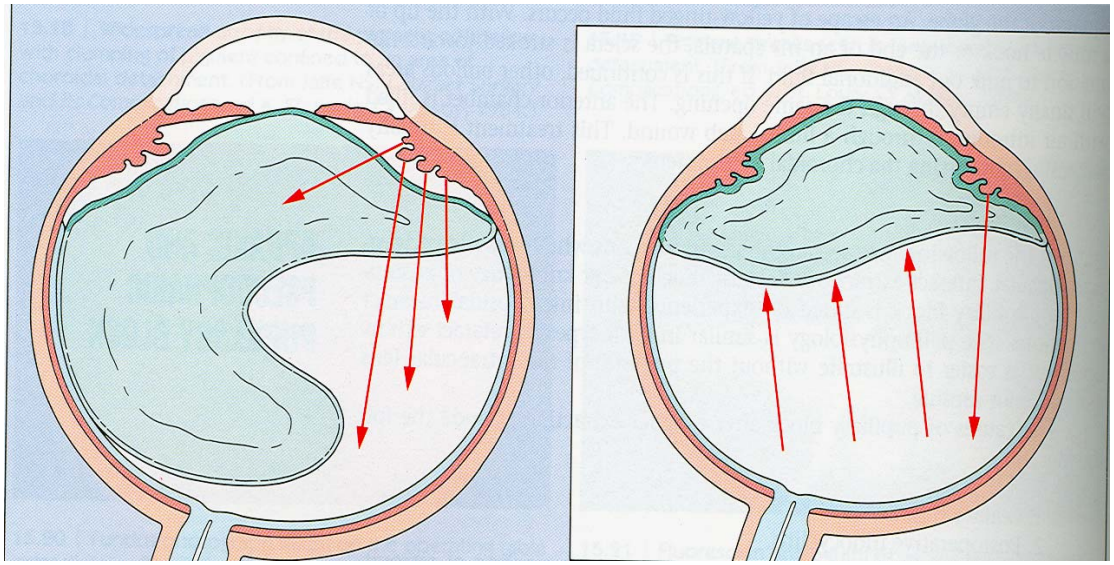

Fig 18 :- Mechanism of Malignant Glaucoma

Treatment

- Medical therapy
- Nd:YAG Laser therapy
- Surgical intervention.

8. BACTERIAL ENDOPHTHALMITIS

Causative organism [33]

- *Staphylococcus epidermidis*
- *Staphylococcus aureus*
- *Pseudomonas* sp.
- *Proteus* sp.

Clinical feature

- pain out of proportion
- severe diminution of V.A
- swollen lids
- corneal oedema

- hypopyon

***Staphylococcus aureus* – 1st – 3rd post-operative day**

***Staphylococcus epidermidis* – 4th – 10th post-operative day.**

Differential diagnosis

- retained lens material
- toxic reaction
- prolonged / difficult surgery => corneal oedema

Table 10 : - ***Methods of Culture Specimen collection in Endophthalmitis***

Anterior Chamber
1. Make keratotomy in the peripheral cornea with a Wheeler knife 2. Insert a 25-gauge needle (attached to a tuberculin syringe) into the anterior chamber 3. Withdraw 0.1 ml of aqueous
Vitreous Cavity
Pars Plana Approach 1. Insert a 22-gauge needle (attached to 3 ml syringe) 3.5 to 4 mm posterior to the limbus into the mid-vitreous cavity 2. Withdraw 0.2 to 0.3 ml liquid vitreous 3. If the yield is poor, consider a vitrectomy. ***In the Absence of Posterior Chamber IOL and Intact Posterior Lens Capsule*** 1. Widen the initial keratotomy from the anterior chamber tap 2. Insert a 22-gauge needle (attached to 3ml syringe) through the pupil into the vitreous cavity 3. Aspirate gently to yield 0.2 to 0.3 ml of liquid vitreous 4. If the yield is poor, consider a vitrectomy

Table 11 : *Initial Antibiotic Regimen in Endophthalmitis*

Intravitreal injections :	- Vancomycin hydrochloride 1mg in 0.1 ml
	- Amikacin sulfate 0.4 mg in 0.1 ml *or*
	- Ceftazidime 2 mg in 0.1 ml
Subconjunctival :	- Vancomycin 25 mg/0.5 ml
	- Ceftazidime 100mg/0.5 ml
	- Dexamethasone 6mg/0.25ml
Topical :	- Vancomycin 50mg/ml hourly
	- Amikacin 20mg/ml hourly
	- Scopolamine 0.25% *or*
	- Atropine 1% twice daily
	- Prednisolone acetate 1% hourly
Systemic :	- Prednisolone 30 mg bid for 5-10 days

Table 12 : *Dilution for Intravitreal Injection*

Vancomycin (500-mg vial)

1. Add 10 ml of normal saline solution to the vial to obtain an initial concentration of 50 mg/ml
2. Take 2 ml aliquot and add 8 ml of normal saline solution to obtain a concentration of 10 mg/ml
3. Inject 0.1 ml (containing 1 mg)

Amikacin (100-mg vial)

1. Amikacin comes in a vial with a concentration of 50 mg/ml
2. Take 1 ml aliquot and add 11.5 ml of normal saline solution to obtain a concentration of 0.4 mg/ml
3. Inject 0.1 ml (containing 400 ug)

Ceftazidime (1-g vial) :

1. Add 10 ml of sterile water to the vial to obtain an initial concentration of 100 mg/ml
2. Take 2 ml aliquot and to 8 ml of normal saline to obtain a final concentration of 20 mg /ml
3. Inject 0.1 ml (containing 2 mg)

Table 13 *: Endophthalmitis Vitrectomy Study Results*

Presenting Vision	**Action**
Better than light perception :	- Tap/biopsy for culture - Intravitreal antibiotics - Subconjuntival antibiotics &steroids -Topical antibiotics - Systemic steroids
Light perception only :	- Three-port vitrectomy - Intravitreal antibiotics - Subconjuntival antibiotics & steroids - Topical antibiotics - Systemic steroids

9. PUPILLARY BLOCK

- Pupillary space & iridectomies are occluded by - vitreous,
- gas,
- blood,
- inflammatory cells,
- lens capsule,
- retained cortical material
- IOL,
- silicone oil, etc.
- It is the most common cause of post-operative glaucoma.

Possible mechanism

- entities force IOL & capsular bag anteriorly (wound leak & choroidal detachment)
- entities that physically block pupillary aperture (inflammatory debris, blood or fibrin)

REVERSE PUPILLARY BLOCK or **STICKY PUPIL SYNDROME-**

- blockage of communication between AC & PC due to sealing of visco-elastic agent between iris & IOL.

CAPSULAR BLOCK

edge of small anterior capsulorrhexis opening adheres to PC IOL optic.

Table 14 : ***Treatment sequence for Pupillary Block*** -

- Laser iridectomy
- Pupillary dilatation
- Reduce IOP medically
- Argon laser gonioplasty -- residual synechiae
- Surgical goniosynechialysis -- synechiae with ↑ IOP
- Filtration / seton surgery

10. HAPTIC INCARCERATION

- Proximal feet of IOL haptic might be discovered in the wound within 3rd post-operative day.
- it should be replaced surgically by passing a spatula through a stab incision at the limbus to dislodge the incarcerated haptic.
- if discovered late, it may cause severe haemorrhage.
- if maneuver to dislodge incarcerated haptic & replaced it is not successful, wound should be opened & haptic replaced.
- protruding haptic may lead to- endophthalmitis
- wound leak

- endothelial touch.
- if haptic cannot be replaced leading to destabilization of IOL & subluxation
- IOL should be removed.

11. LENS PRECIPITATE

These are of three types, viz

1. Pigment deposits- on both surfaces of IOL. Arises from pigment epithelium of iris because of dispersion by the loop supports behind the iris.
2. Greyish white precipitate – these are residues of lens material after an ECCE.
3. Precipitate resembling KPs- these are seen after recurrent uveitis.

Treatment

- topical steroids
- sub-tenons steroids
- faulty IOL removal

12. FIBRINOID REACTION

- A transient deposition of fibrin like material is sometimes seen in pupillary area mainly following ECCE & PC IOL.
- there is little or no cellular uveitis, usually seen between 2^{nd} and 5^{th} post-operative day. When reaction is severe the fibrin coalesces & becomes adherent to the anterior surface of the lens.

Pathogenesis

- pseudometaplasia of lens epithelium which is enhanced by contact with PMMA.

Risk factors

- Exfoliation Syndrome
- Diabetes Mellitus
- Glaucoma

Treatment

- steroid drops hourly
- intensive mydriasis
- if reaction doesn't subside by 24 hours, 4mg Betamethasone injected into the floor of orbit.
- usually resolves with in 2 weeks of treatment.
- rarely requires Nd: YAG membranotomy

13. MALPOSITION

Causes

- Too much air in AC
- Too wide pupil
- Premature discontinuation of Pilocarpine
- Accidental use of mydriatics
- Loop length of IOL is too short.
- Implant size - too small.
- Operative rupture of PC, zonular dialysis.
- Ocular trauma.

DECENTRATION

- edge of optic reaching pupillary area.
- most commonly results from, one haptic (inferiorly) in capsular bag & other (superiorly) with in ciliary sulcus.
- if centration is good- ***bounce-back-test*** is +ve.
- minor decentration occur if optic becomes adherent to mid-stromal iris in post-operative period => ***Reverse iris tuck***.

PUPIL CAPTURE

- part of PC IOL optic anterior to iris plane & rest in posterior chamber, below iris plane.

Complications

- Capsule synechiae
- low grade uveitis
- pupillary block glaucoma [34]
- Cystoid Macular Edema (CME)
- pigment deposition on IOL.

Treatment

- Dilatation of pupil, then constriction.
- pressure over haptic with glass rod.

WIND SHIELD WIPER SYNDROME

- when IOL is too small for eye.
- when 'in-the-bag' IOL placed in 'ciliary sulcus'.
- upper portion of IOL has pendular movement with ocular movement.
- Treatment – fixing IOL by Mc-Cannel's suture.

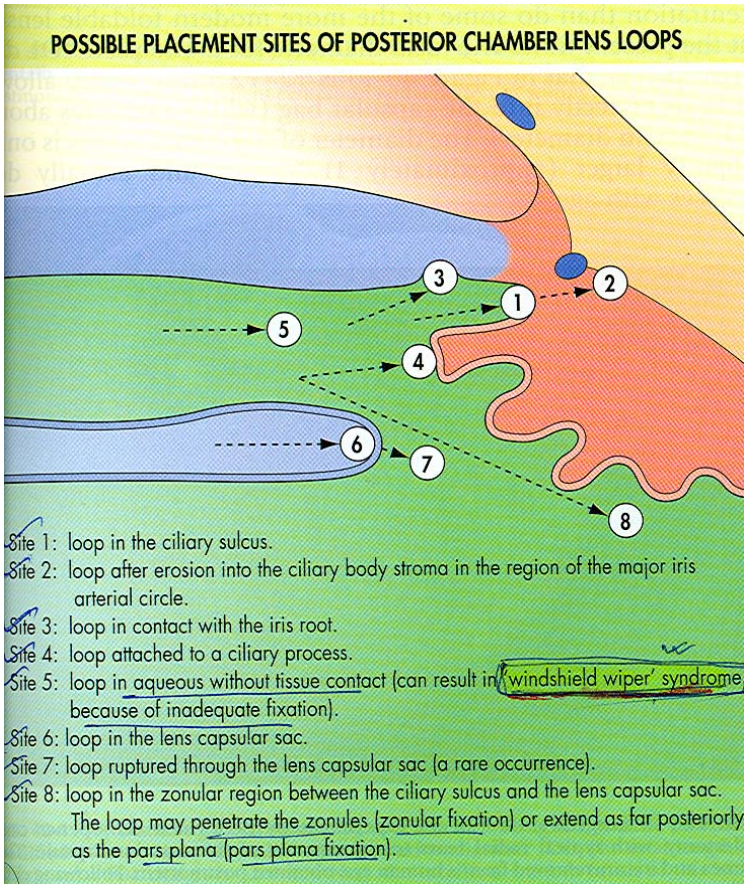

Fig 19 :- Possible sites of PC IOL haptic placement -

SUN SET SYNDROME

- IOL gradually sinks down towards 6'o clock position.
- due to inferior zonular dialysis in early cases.
- late cases due to globe trauma.
- optic pulled towards 12'o clock & fixed by Mc-Cannel's suture.

SUN RISE SYNDROME

- vertical displacement of optic superiorly.
- eccentric capsular bag fibrosis.
- inferior zonular rupture with horizontally placed IOL in the bag.
- IOL exchange or fixation by Mc-Cannels suture.

Table 15 : **Advantage of placing both haptics 'in the bag'**

1. proper IOL positioning
2. both loops are symmetrically placed in the bag
3. avoiding intra-operative stretching & tearing of posterior capsule by loop manipulation
4. decreased IOL decentration & dislocation
5. decreased spontaneous loop dislocation
6. maximum working distance from cornea
7. maximum distance from iris tissues
8. iris chaffing is reduced
9. decreased blood-aqueous barrier break down
10. least surface alteration of loop material
11. safer for children & young adults
12. incidence of posterior capsule opacification is reduced
13. it's easier to explant
14. decreased incidence of post-operative springing out of loops from capsular bag => ***PEA-PODDING EFFECT***.

B. Late Post-Operative Complications

1. POSTERIOR CAPSULAR OPACITY (PCO)

- Misnomer, as posterior capsule doesn't opacify, but it is covered by opaque material.
- Opacity results from opaque secondary membranes formed by active lens epithelial proliferation & transformation of lens epithelial cells into fibroblasts with contractile elements & collagen deposition.
- The anterior lens epithelial cells proliferate onto the posterior capsule at the site of apposition of the anterior capsule flaps to the posterior capsule.
- Contraction caused by myoblastic fibres of lens epithelial cells produces wrinkling of posterior capsule.
- Collagen deposition results in white fibrotic opacities.
- Mitotic inhibitors instilled into the AC after cataract surgery reduces capsular opacification.[35]

Types

a) CAPSULAR FIBROSIS

- gray - white band or plaque like opacity
- fibrosis present in 1st post-operative week, represents cortical lamella left at the time of surgery.
- fibrosis which appear months to years post-operatively are caused by migration of anterior lens epithelium, fibroblastic metaplasia & collagen production.
- commoner in young individuals with more vigorous proliferative potential.

b) SOEMMERINGS RING

- Thick range of after cataract formed behind the iris, enclosed between the two layers of lens capsule.

c) CAPSULAR WRINKLING[36]

- *Broad undulations*- early post-operative period, before capsule becomes tense, due to fibrosis of wrinkles caused by PC IOL along the axis of the haptic orientation.

- *Fine wrinkles or folds* – by myoblastic differentiation & results in marked optical disturbances.

d) ELSCHNIG'S PEARLS and BLADDER CELLS

- Caused by proliferating lens epithelial cells at he sites of apposition between anterior capsule remnants and posterior capsule.

- Vacuolated subcapsular epithelial cells are clustered like soap bubbles along posterior capsule.

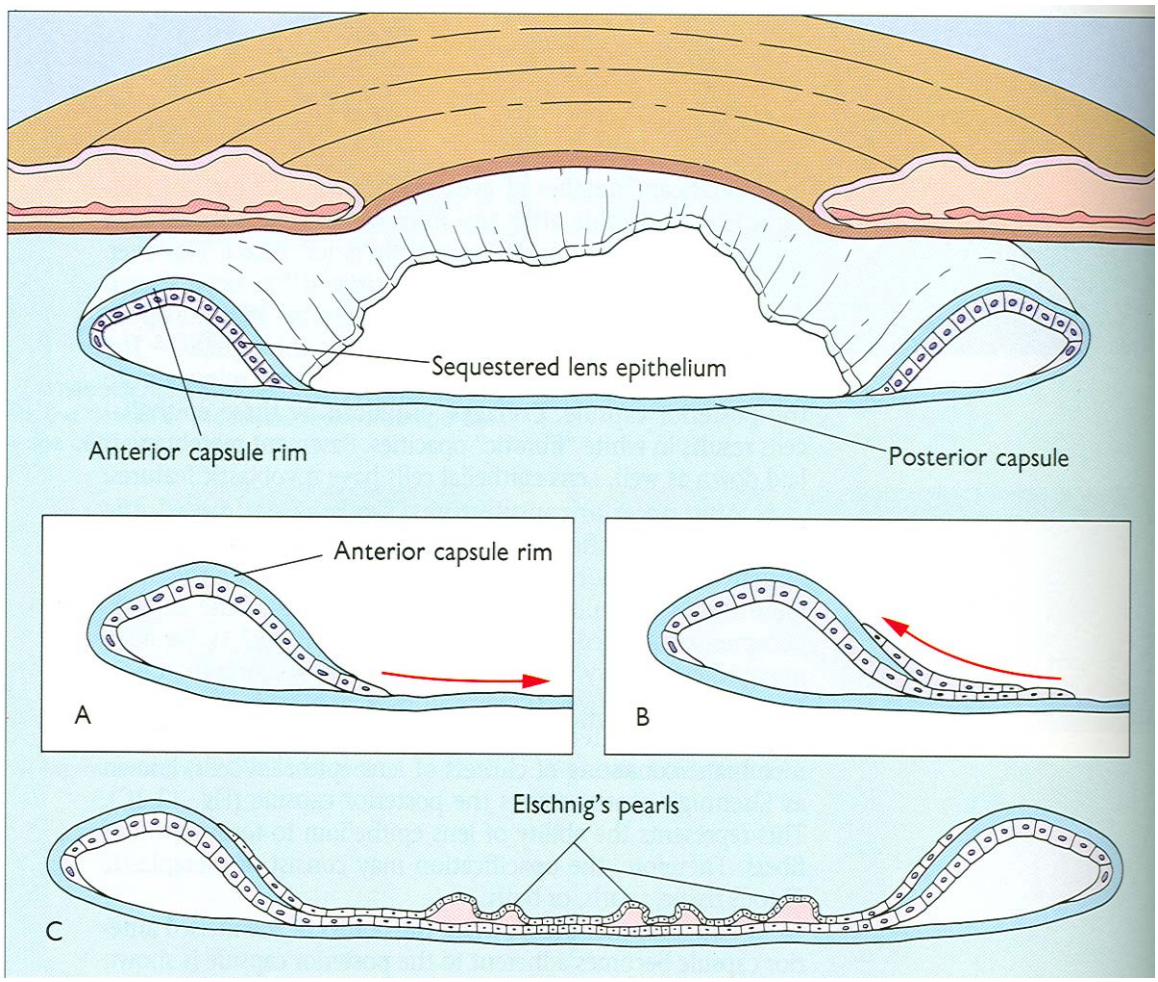

Fig 20 : Elschnig's Pearl

INDICATION FOR TREATMENT

- Decreased visual acuity
- Impaired visualization of fundus for diagnostic and therapeutic purposes
- Severe glare by capsular wrinkling

Treatment

a) Surgical

- Needle discussion in thin posterior lental membrane.
- Membranectomy with vitrectomy for thick membrane.

b) Nd- YAg LASER capsulotomy

Table 16 : Laser Posterior Capsulotomy Technique

Use minimum energy : 1mJ if possible
Identify and cut across tension lines
Perform a cruciate opening -
- Begin at the 12 o'clock position in the periphery
- Progress towards the 6 o'clock position
- Cut across at the 3 & 9 o'clock positions
- Clean up any residual tags
- Avoid freely floating fragments

2) CYSTOID MACULAR EDEMA (CME)

Fluid accumulation in the outer plexiform layer of central macula, resulting in visible cystoid spaces.

Pathophysiology- *breakdown of blood retinal barrier* by

1. Mechanical Disturbance

- iris incarceration in the wound
- anterior vitreous adhesions and incarcerations
- vitreous traction at the macula

- hypotension
- turbulence
- endophthalmodonesis

2. Inflammation

- Iris retraction by vitreous adhesions
- Iris tuck by IOL
- Pupillary capture by IOL
- Pupil irregularity
- Anterior chamber inflammation

All increases release of prostaglandin mainly PGE1 and PGE2 as inflammatory mediators which increases the permeability of parafoveal capillaries leading to disruption of inner blood retinal barrier and eventually CME

3. Toxic Effect

Aqueous humour contains biochemically active principles manifesting biotoxic effect when it leaves it natural reservoir, called ***Aqueous biotoxic complex factors.*** [37]

Intact PC and anterior hyaloid face walls off posterior segment of eye from effects of theses ABC factors.

Clinical features

- decreased central vision with VA, 6/60
- Photophobia
- metamorphosia
- micropsia
- scotomata

Triad of

- Vitreous touch
- Bullous keratopathy
- CME => is called **Irvine- Gass- Syndrome**

Diagnosis

a) Ophthalmoscopy - altered foveal light reflex (FLR)

honey-comb appearance of macula.

b) Slit-lamp bio-microscopy altered volume, thickness & cystic spaces in macula

c) Fundus Fluorescine Angiography (FFA)-

- mild CME - para-foveal hyper fluorescence.
- CSME - pin point spots of fluorescence leakage in blood-retinal-barrier breaches
- these coalesces into ***'flower petalloid 'staining*** pattern, with dark or bright central cyst in fovea

d) Vitreous Fluoro Photometry

- leakage at blood- retinal barrier can be measured quantitatively.

Treatment

- Medical

Table 17 : **Therapeutic Agents for Cystoid Macular Edema**

Corticosteroids
Topical
- Prednisolone acetate 1% four times daily
- Prednisolone sodium phosphate 1% four times daily
- Dexamethasone 0.1 % four times daily
Peribulbar

- Triamcinolone (Kenalog) 20 mg (o.5 ml) every 3-6 wk - Methylprednisolone (Depo-Medrol) 20 mg every 3-6 wk ***Oral*** - Prednisolone 1-1.5 mg/kg/day ***Intra-vitreal*** : - Triamcinolone (TRICORT)
Nonsteroidal Antiinflammatory ***Topical*** : - Diclofenac sodium 0.1 % 4 times daily - Flurbiprofen sodium 0.03 % (Ocufen) 4 times daily - Suprofen 1% 4 times daily - Ketorolac trimethamine 0.5% 4 times daily - Indomethacin
Carbonic Anhydrase Inhibitors ***Oral*** -Acetazolamide (Diamox) 500 mg once daily
- Hyperbaric Oxygen

Surgical

- release of vitreo- macular traction surgically or by Nd:YAG laser.
- Persistant & unremitting CME – Vitectomy.

3. EPITHELIAL DOWNGROWTH [38]:

- Migration of conjunctival epithelial cells onto the corneal endothelium through fistulous wound or needle track.

Clinical features

- decreased VA

- Pain
- photophobia
- tearing
- conjunctival injection
- corneal decompensation
- severe glaucoma with or with out obvious angle closure
- chronic anterior uveitis
- presence of retro-corneal membrane with a demarcated leading edge.
- Siedel's test is + ve

Diagnosis confirmed by

- ***Argon Laser Photocoagulation*** pf iris of laser setting 300-700 mV & 500um spot size.
- blanching seen in presence of epithelial cells, but standard brown burns in normal iris tissue.
- ***Specular Endothelial Microscopy***- shows a definitive demarcated line of heaped up epithelial cells presenting as the leading edge of the epithelial membrane.

EPITHELIAL CYST - formed due implantation of conjunctival epithelial cells in the AC.

Definitive treatment

- Complete destruction of all intra-ocular epithelial tissue by
- cryotherapy of involved cornea,
- iridectomy
- pars-plana viterectomy with removal of involved iris, ciliary body, lens, by endolaser & filling AC with air.
- Prognosis - poor.

4. FIBROUS INGROWTH [39]

- Abnormal invasion of AC by connective tissue by fibroblastic proliferation originating from sub-epithelial connective tissue.
- corneal & limbal stroma & - metaplastic endothelium
- Retro-corneal membrane has an irregular border of fibrous tissue.
- Secondary complications are-- corneal stromal oedema
- Glaucoma
- Retinal Detachment (RD)

5. FILTERING BLEB FORMATION

- Wound leak under sealed conjunctiva, results in formation of filtering bleb.
- blebs developing after 1st few post-operative days are mainly caused by wound leak due to trauma
- suture breakage or loosening
- scleral melting.
- these cases may be treated by surgical repair of wound.
- long standing bleb are complicated by epithelization of fistula.
- Treatment by - careful exploration of fistula
- scraping of fistulous tract
- filling of fistula with scleral graft or pericardial graft
- followed by meticulous resuturing of wound.

- bleb is shrunk by
- cryotherapy
- chemical cauterization with Tri-chloro-acetic acid,
- Argon Laser Photo coagulation followed by application of Methylene blue or Rose Bengal dye,
- Diathermy
- *thin walled* ***‘ weeping ‘ blebs*** are more prone to develop endophthalmitis.

6. UNPREDICTABLE ASTIGMATISM -

can be due to

i) Corneal Steepening

- tight suturing
- excess cautery

ii) Corneal Deepening

- loose knot
- vertical wound mis-alignment.

iii) Irregular astigmatism

- mainly against the rule astigmatism
- irregular suture tension
- horizontal wound mis-alignment

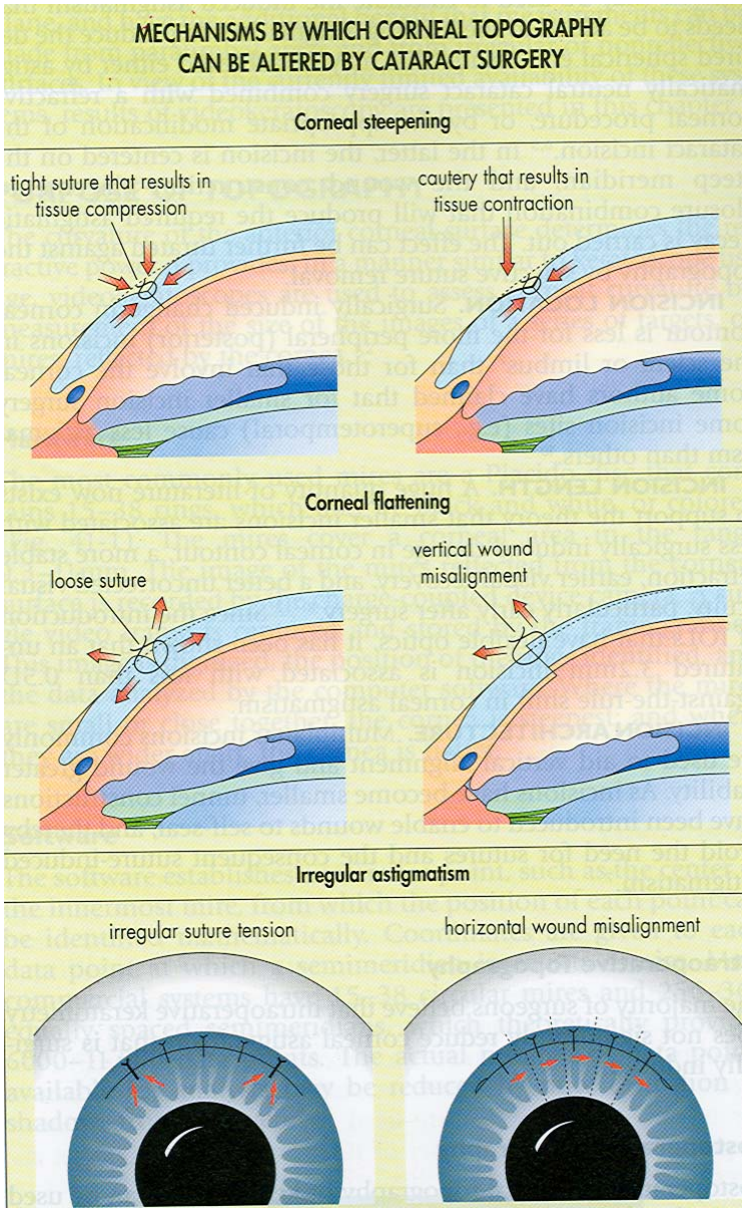

Fig 21 : Mechanism of Irregular Astigmatism after cataract surgery

Treatment by

- spectacles
- contact lenses
- removal of abnormal sutures.
- Astigmatic keratometry.

7. PERSISTANT CHRONIC UVEITIS

Causes

- intra cameral distortion of IOL haptic feet
- imperfectly finished IOL surface with small saw like projections rubbing against iris
- micro movements of IOL haptics
- iris chafing
- all causes low grade uveitis, pigment dispersion & progressive anterior synaechie.

treatment by

- sterioids, followed by implant removal.

8. IOL RELATED COMPLICATIONS: -

- improper power calculations –Astigmatism.
- improper finishing – post-operative uveitis
- improper sterilization – toxic reaction & endophthalmitis.
- malpositions – glare & vision problems
- haptic compressing angle – glaucoma

9 RETINAL DETACHMENT

- mainly ***Rhegmatogenous Retinal Detachment (RRD)*** .

Patho-physiology

a) Cataract extraction increases the sagittal length of vitreous cavity, allowing vitreous to project further anteriorly into AC, exert greater traction on the vitreous base & other areas of vitreo-retinal adhesions, leading to ***posterior vitreous detachment (PVD)*** , which in turn creates retinal holes, thus allowing accumulation of sub-retinal fluid & eventual RRD.

b) ***Endophthalmodonesis*** – relative mobility of structures with in the eye as iris, IOL, vitreous compared to immobile parts as sclera & cornea along with saccadic movements of the eye leads to increased vitreo-retinal traction.

c) ***Loss of hyaluronic acid*** from vitreous after cataract surgery makes eye more prone to trauma during ocular movements, as hyaluronic acids serve the function of shock absorber of vitreous.

Risk factors [40]

- Axial Myopia
- Retinal Detachment in the other eye
- vitreous loss in other eye.

Table 18 : Prevention & Prophylaxis of Postcataract Retinal Detachment

Keep posterior capsule intact
Perform careful preoperative dilated fundus examination prior to both cataract surgery & Nd:YAG laser capsulotomy
Perform frequent dilated fundus examination in the 1st year after surgery
Educate the patient

- Pseudo-phakic RRD occur with in 1yr of cataract removal.
- Following Nd:YAG capsulotomy RRD appears with in 6 months of procedure.

Diagnosed by

- Indirect ophthalmoscopy – pigment granules in anterior vitreous after PVD appears as ***" tobacco dust".***

Table 19 : **Indications for Treatment of Suspicious Retinal Lesions**

Asymptomatic
- Any tear with greater than one disc diameter of fluid (subclinical RRD) - Horseshoe or flap tears with fluid - Horseshoe or flap tear in contralateral eye of patient with previous RRD
Symptomatic
- Any tear with greater than one disc diameter of fluid (subclinical RRD) - All horseshoe or flap tears - Most operculated tears - Atrophic holes in contralateral eye of patients with previous RRD

- bed rest & patching
- Laser & cryotherapy
- Scleral buckling
- Premature retinopexy
- ***Pars-plana-vitrectomy (PPV***) with Laser, gas injection & internal drainage of sub retinal fluid with out scleral buckling.

10. BULLOUS KERATOPATHY [41]

Mechanisms contributing to corneal decompensation-

- Surgical trauma
- intra-ocular inflammation with loss of endothelial cells
- Pseudophakodonesis

- inflammation from lens design & lens material.

Vitreous Touch Syndrome - persistant corneal oedema due vitreous touch leading to decreased endothelial cells.

- Symptoms
- variable vision, worse in the morning with subsequent improvement as the day progresses & the cornea deturgesces from surface evaporation.
- Signs examination reveals guttata.

Treatment

- Medical
- mild cases with reasonable vision- hypertonic saline & lubricating eye drops.
- reduction of IOP decreases corneal oedema.
- topical steroids.
- bandage contact lens
- Surgical
- penetrating keratoplasty

11. LATE POST-OPERATIVE ENDOPHTHALMITIS

seen when low virulent organisms are trapped in capsular bag, viz-

  - Propionibacterium acnes
  - Staphylococcus epidermidis

- C/F- late onset, persistant, low grade inflammation.
- sometimes granulomatous features such as Mutton fat keratic precipitates (KP)s are seen.
- inflammation responds well to steroids
- after cessation of treatment inflammation soon recurs.

- enlarging white plaques on posterior capsule is sign if Propionibacterium acne infection.
- diagnosis confirmed by - vitreous culture.

Treatment

- topical & periocular steroid & antibiotics effect is transient.
- intra-vitreal injection of Vancomycin.
- IOL removal along with remnant cortex & the entire capsular bag.

12. LATE ONSET HYPHAEMA

Anterior segment haemorrhage an occur months to years after cataract surgery due to bleeding from Neovascularization of wound site or vascular tufts in contact with AC IOL haptic or PC IOL haptic in sulcus due to iris chafing, called ***Swan Syndrome / Sputtering Hyphaema Syndrome.*** [42]

- Red Blood Cells (RBC) 'dusting' might be seen on corneal endothelium.
- On exposure to bright light - rapid dilatation of pupil leads to bleeding from adhesion sites -> blurring of vision (**WHITE OUT**) -- ***WHITE OUT SYNDROME.*** [43]
- Treatment by
- Nd:YAG Laser photo coagulation of noevascularization
- limbal cryopexy
- rotation of IOL haptic by 90 degrees.

13. PUPILLARY MEMBRANE

- this membrane bridges the pupillary aperture & may obstruct the visual axis partially or completely.

- after cataract surgery interaction between pupillary margin & anterior hyaloid face results in the formation of pupillary membrane which appear like secondary cataract & is amenable to discussion.

- when these membranes recur they obliterate the pupillary space & simple discussion is not sufficient , here pars plana vitrectomy & removal of vitreous & pupillary membrane is required.

This study is mainly concerned with Per-operative & Post-operative complications of ECCE & Manual SICS cataract surgeries.

In 1953, Ridley implanted PCIOL in about 750 eyes. He found severe postoperative complication e.g. severe postoperative iritis, inflammatory pupillary membrane formation, thickening of posterior capsule, secondary glaucoma and dislocation of IOL into the vitreous. Around 1959 he gave up IOL implantation. [13,44,45]

Binkhorst successfully implanted 4 loop iris clip lens in 1958 which produced pupillary block glaucoma and lens dislocation.[46,47]

In 1968 Fydorov and Zakorov introduced three posterior loops Sputnik or Fydorov II lens . [48]

The results of implantations of the Fydorov II lens in 6995 cases of non traumatic cataract indicated few complication during surgery and some post operative complication in 3016 cases

Preoperative complication	Incidence
1. Vitreous prolapse	2.7%
2. Incomplete removal of lens matter	2.6%
3. Haemorrhage	0.6%
Post Operative complication	**Incidence**
1. Corneal oedema	3.3%
2. Iridocyclitis	2.2%
3. Pupil block	0.6%
4. Macular oedema	3.4%
5. Lens dislocations	3.2%
6. RD	0.4%

Reviewing 929 cases of Pearce PCIOL from 1975 to 1980 showed the posterior capsule opacification as follows, Pearce J.L. developed light weight parpex CQ tipped posterior chamber lens.[49,50]

Year	Incidence of PCO
1975 - 76	11%
1977	12%
1978	11%
1979	6%
1980	5.5%

Simcoe CW [51] showed the following results and complications of 1523 cases of "C" loop PCIOL performed by four different surgeons. Results of 1523 cases with Simcoe PCIOL

Complication	Incidence
Zonule or capsule routine by lens	0%
CME	1.2%
RD	0.2%
Corneal decompensation	0

Shearing S.P. first implanted compressible non sutured ("J" loop PCIOL) in 1997. According to Dr. Shearing two complications were common with this type of lens – pupil capture and sunset syndrome. [52]

There is some evidence to suggest that certain irrigating solutions or durgs used at the time of lens implantation may have a toxic effect on the endothelial cell population.[53]

Alpar described some common complication which occur during surgery with PCIOL e.g. bleeding from corneo-scleral wound, incarceration of haptic into the wound, descemets membrane detachement , zonule rupture, post capsule rupture with vitreous loss, small rigid pupil, intra operative shallowing of the anterior chamber and expulsive haemorrhage. [54,55]

Alpar suggested that one should be generous with the use of cushion material during the removal of nucleus of the lens and during introduction of artificial lens to protect corneal endothelium. [56]

Alpar described the multifactorial entity of post operative glaucoma in PCIOL. He mentioned strong water tight wound closure, intensive use of corticosteroids,

cauterization of the emissary and aqueous veins, post operative swelling of the trabecular meshwork in corneo-scleral incision, plugging of the meshwork with retained cortical material, intracameral Methylcellulose, sodium hyaluronate are the possible contributory factor of rise of post operative intraocular pressure. [57]

Summers and Lindstrom (1988) described two cases of ghost cell glaucoma resulting from large intraoperative haemorrhage during lens insertion or due to erosion of cilliary body by lens loop.[58]

Bartholonew reported that 5 to 50% of patients of ECCE with PC IOL developed posterior capsule opacification and therefore required a secondary capsulotomy between 12-24 months. [59,60]

Jafee reported the incidence of clinically significant cystoid macular oedema is about 10% after ICCE with an Iris clip lens and 4% after ECCE with sulcus fixed posterior chamber lens [61,62].

Kern et al studied 200 cases of extra capsular cataract extraction with posterior chamber lens implantation and found 1% clinical cystoid macular oedema in those cases.[63]

A prospective study of 100 cases of extra capsular procedures and IOL insertions in Saudi Arabia by Dr. Olson[64] reported the following operative and post operative complications- PC tear without vitreous loss in 2 cases (2%) , torn iris sphincter in case (41%), iris prolapse (1%), early rise of IOP in 6 cases (6%), cystoid macular oedema in 1 case (1%).

A review of worldwide IOGEL (poly HEMA) intraocular lens implantation from May , 1986 to December 1988 highlighted the following per and postoperative complications e.g. posterior capsule rupture 2.4%, descement's membrane detachment

0.3%, significant anterior chamber bleeding 0.9%, iris damage 4.7%, vitreous bulge 4.4%, vitreous loss 1.2%, cortical remnants and zonule rupture 3.5%, postoperative hyphaema 1.9%, pupillary block glaucoma 0.2%, persistant secondary glaucoma 0.7%, persistent iritis 0.5%, persistant vitritis 0.3%, hypopyon 0.2%, lens dislocation 0.7%, cystoid macular oedema 2.9%, endophthalmitis 0% and retinal detachment 0% [65,66,67]

Occasionally rupture are unavoidable.Surgeons are expected not to have more than 3% posterior capsule rupture and where the rupture rate approaches 5% the techniques of the surgeon has to be reviewed critically.

MATERIALS AND METHODS

In this study total 100 patients, irrespective of sex and age varying from 40-80 years were studied, half of them had undergone ECCE + PC IOL I & rest had undergone Manual Small Incision Cataract Surgery + PC IOL I. .

The patients with high myopia, previous retinal detachment in other eye, microphthalmos, extensive congenital eye anomalies, proliferative diabetic retionopathy, uncontrolled glaucoma, recurrent uveitis, corneal dystrophy were excluded from this study. Children, one eyed patient and traumatic cataract were also not included in the present study

History, name age, sex, religion were noted, general history of chronic illness e.g.heart diseases, respiratory diseases, diabetes and hypertension were taken.

Details ocular history including trauma to the eye, redness, pain, photophobia and family history of glaucoma and diabetes were also recorded.

Local Examination of the eyes

Visual acuity of all the patients were tested by Snellens test types. Where finger counting was not present, hand movement and projection of rays and perception of light were noted. The detailed examination of the eyes and adnexa were performed with the help of torch light, loupe and slit-lamp bio-microscope. Intra-ocular pressure was recorded with Schiotz and Goldman’s Applanation tonometer pre-operatively. After pre operative check up and a complete medical evaluation with appropriate laboratory work-up e.g. blood sugar estimation & conjunctival swab culture was done.Lens power was calculated with the help of keratometry and A-scan ultrasonography. Then these patients were admitted for operation.

Operated eyes were reopened on the next morning after the operation, steroid and antibiotic eye drops were instilled for about 3 wks, short acting mydriatic was advised & patients were also instructed to use temporary dark glasses.

Patients were kept in hospital for 2-3 days. Routinely local antibiotic and topical corticosteroid combination drop along with a cycloplegic were instilled in all cases excepting those who developed complications, they required extra medications.

Careful examination is important during the first post operative days with particular attention to the intraocular pressure and evidence of inflammation.All patients were examined under slit-lamp bio-microscope.The patient can be discharged 1-3 days after surgery if the postoperative course is smooth & follow up examinations are arranged.

Post operative complications were divided into early (upto 7 days) and late complications (after 8 days). All cases have been followed up till 6 month from the date of surgery.

Post operative follow up examination was done weekly for 1 month and for monthly for next 5 months. Post operative evaluation included-

i. detailed clinical examination

ii. Gonioscopic and & slit lamp bio-microscopic examination

iii. Schiotz & Applanation tonometry

iv. Direct & indirect ophthalmoscopy

v. Bacteriological and fungal investigations of aqueous and vitreous was done properly.

All the datas regarding per operative and post operative complications were properly compiled, evaluated, analysed and discussed.

RESULTS AND OBSERVATION

In this study, total 100 patients irrespective of sex and age varying from 40-80 years and mean age being 56.4 yr., who had, mature and immature cataract, were operated at A.I.M.S B.G.Nagara in between 2004-2005, half of them had undergone ECCE + PC IOL I & rest half underwent Manual SICS + PC IOL I, all cases were followed up for at least 6 months from the date of surgery.

Table 20 : Age and Sex distribution of cases

Age in years	Male	Female
40 – 50	14	18
51 – 60	8	12
61 – 70	13	11
71 – 80	14	10
TOTAL	**49**	**51**

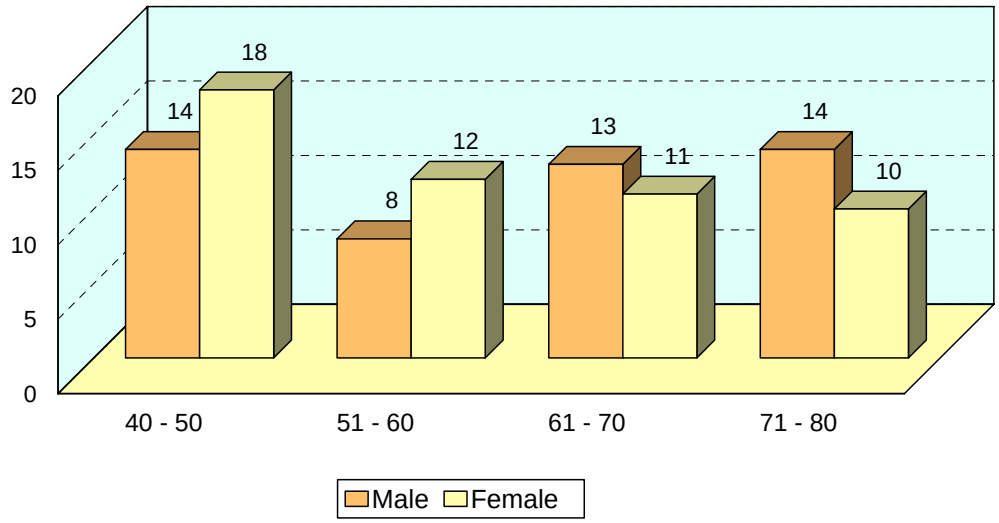

Fig 22 : Age & Sex distribution of cases

Table 21 : Sex distribution of cases

Sex	Male	Female
Male	49	49
Female	51	51
TOTAL	**100**	**100**

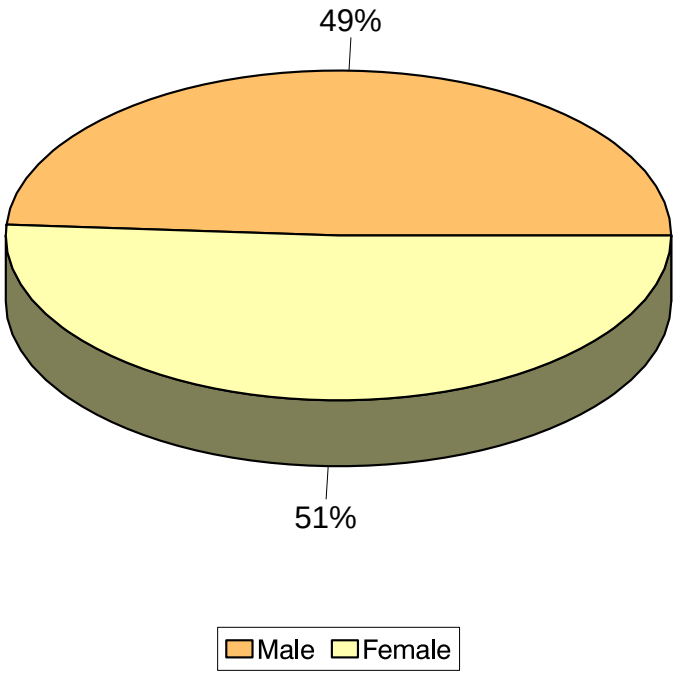

Fig 23 : Sex distribution of cases

Table 22 : Distribution of types of cataracts in cases

Type of Cataract	No. of Cases	Percentage
Advance Immature Cataract	54	54
Senile Mature Cataract	43	43
Hypermature Cataract	3	3
TOTAL	100	100

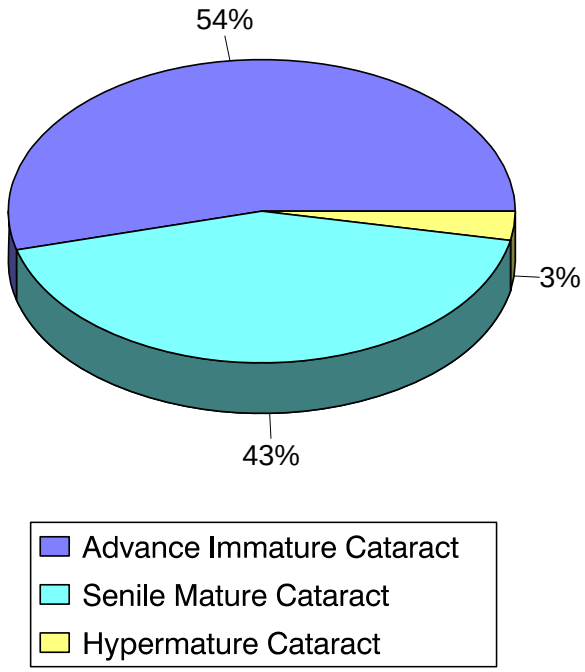

Fig 24 : Distribution of types of Cataracts in cases

Table 23 : Per-operative complications of cases

Sl. No.	Complications	No. of Cases	Percentage
1	Descemet's membrane stripping	2	2
2	Hyphaema	1	1
3	Iris prolapse	7	7
4	Iridodialysis	1	1
5	Posterior Capsular rupture with Vitreous loss	2	2

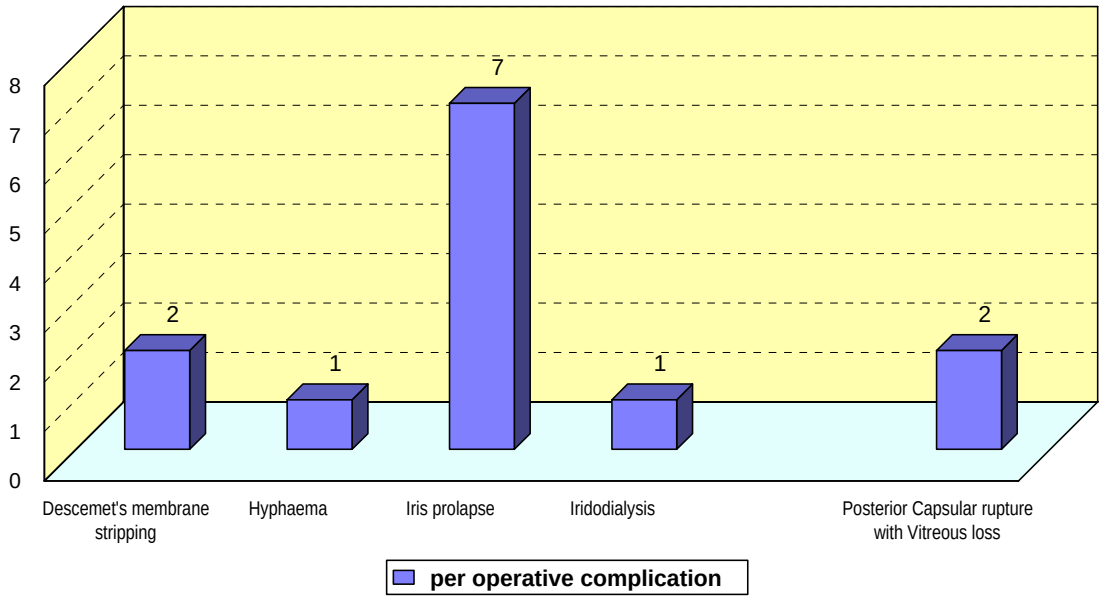

Fig. 25 - Per-operative complications of cases

Table 24 : Early Post-operative complications of cases

Sl. No.	Complications	No. of Cases	Percentage
1	Wound leakage	1	1
2	Significant corneal oedema & Striate Keratopathy	3	3
3	Secondary glaucoma	9	9
4	Iridocyclitis	6	6
5	Bacterial Endophthalmitis	1	1
6	Malposition	1	1

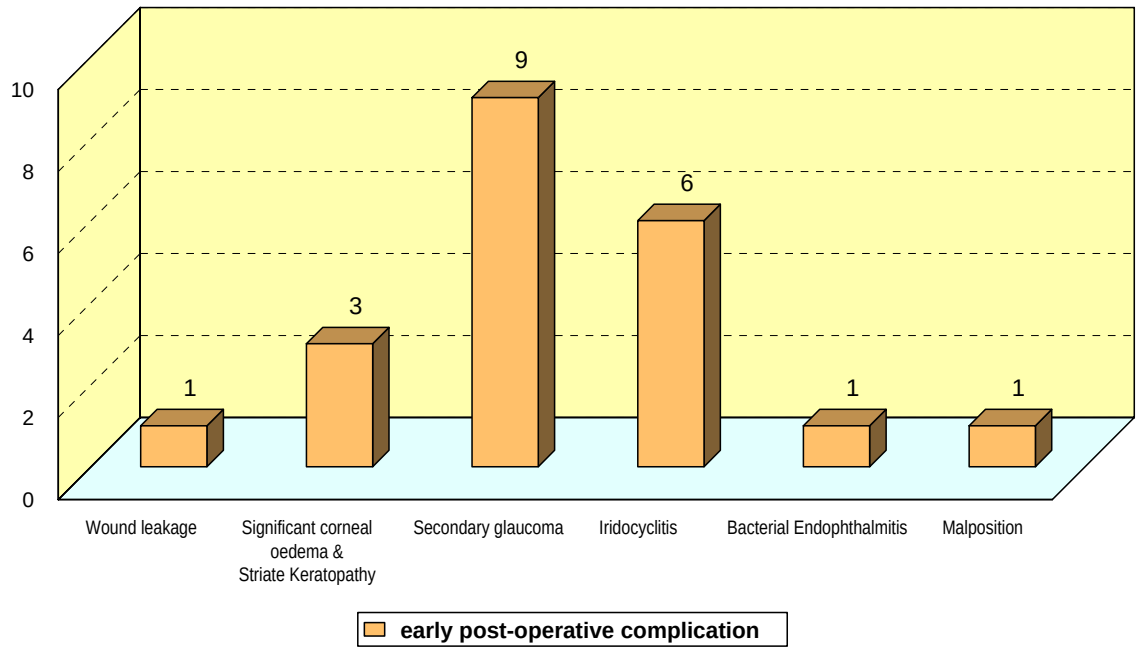

Fig26 : Early Post-operative complications of cases

Table 25 : Late Post-operative complications of cases

Sl. No.	Complications	No. of Cases	Percentage
1	Recurrent iridocyclitis	1	1
2	Persistant corneal oedema & Bullous Keratopathy	1	1
3	Posterior capsule opacification	8	8
4	Cystoid macular oedema	4	4
5	Late Endophthalmitis	0	0

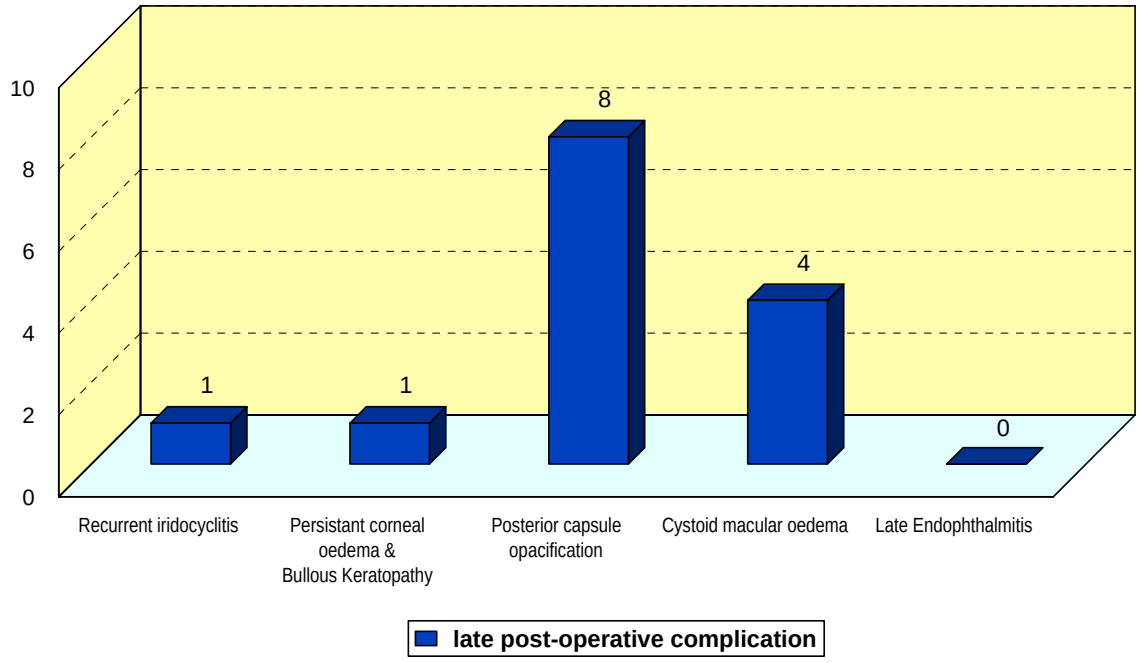

Fig 27 : Late Post-operative complications of cases

Table 26 : Best Corrected Visual Acuity with required glasses

Sl. No.	Visual Acuity	No. of Cases	Percentage
1	6/5 to 6/6	71	71
2	6/6(p) to 6/9	14	14
3	6/9 (P) to 6/12	8	8
4	6/12 (P) to 6/18	7	7

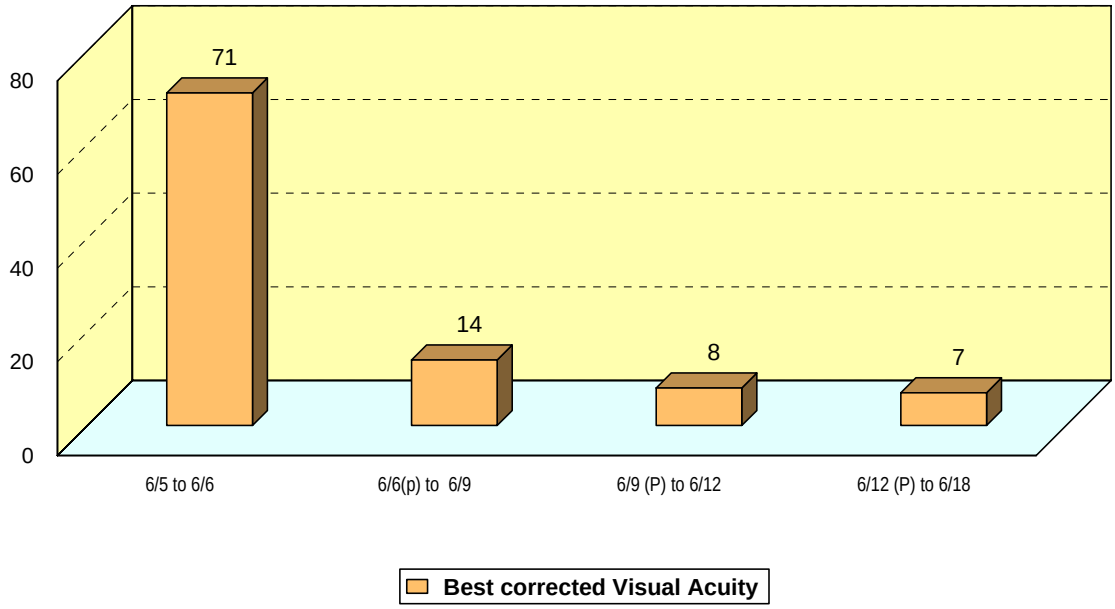

Fig 28 - Best possible corrected visual acuity of cases with required glasses

CASE PHOTOS

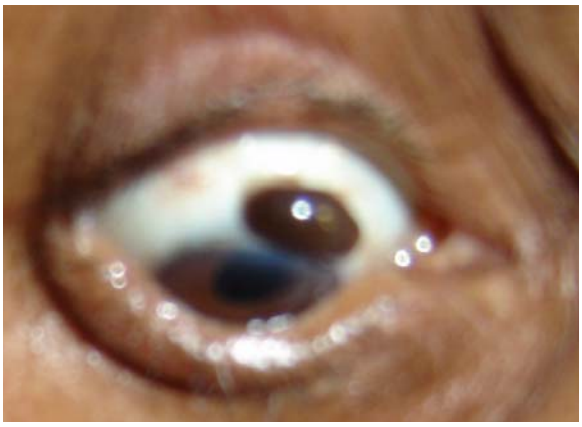

Fig. 29. Iris Prolapse

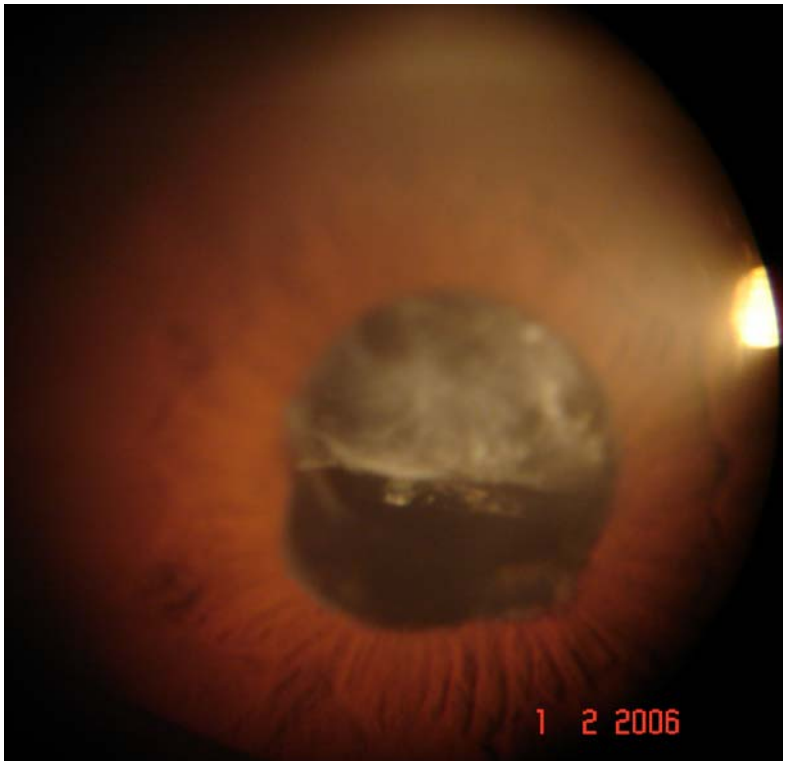

Fig. 30. Posterior capsule opacity

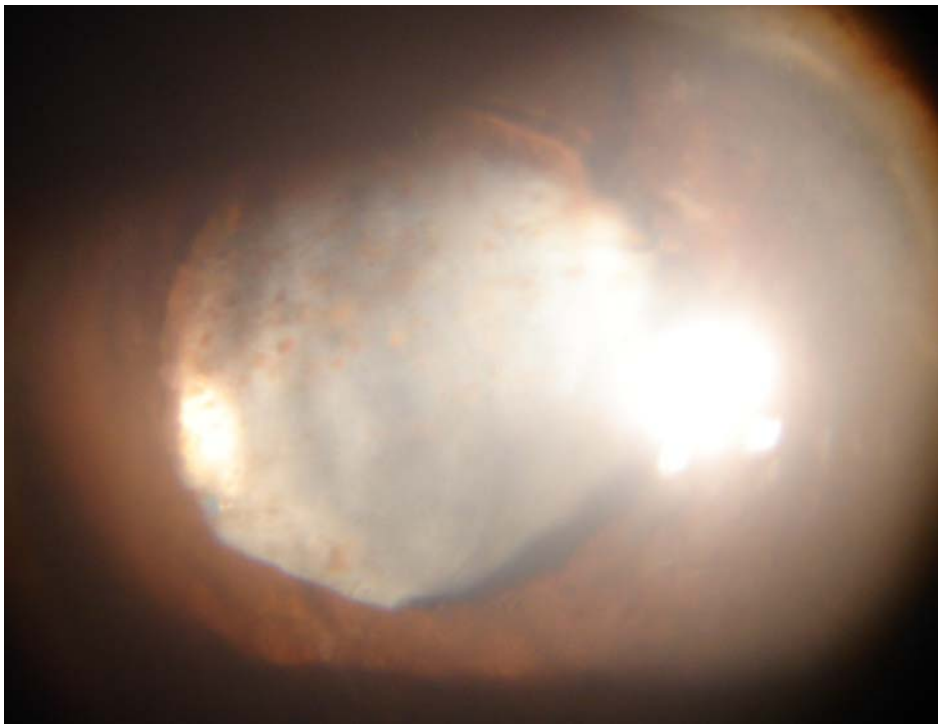

Fig. 31. Pupillary capture

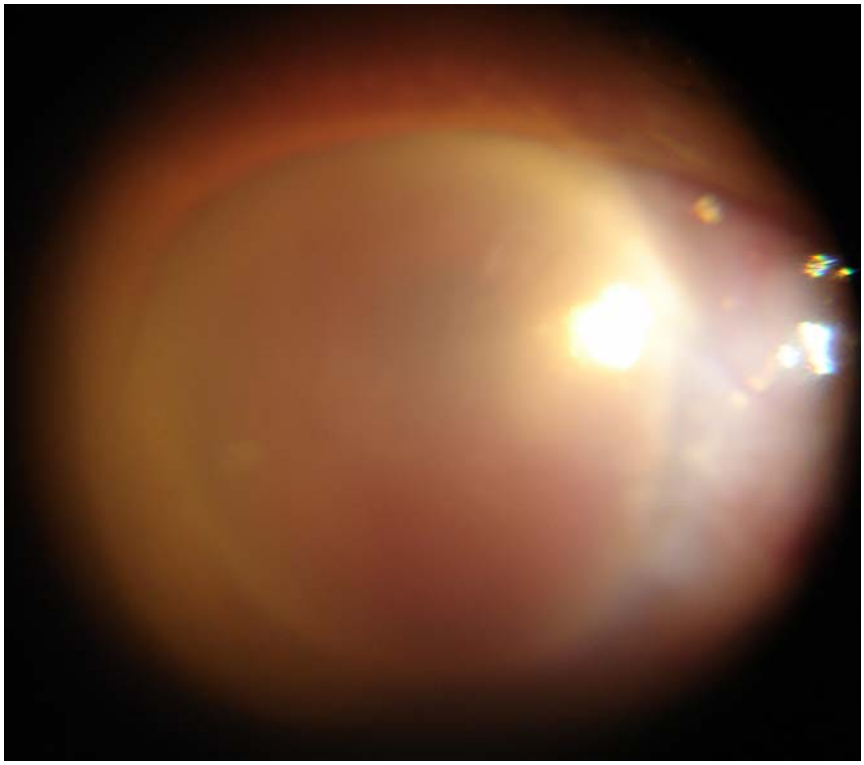

Fig. 32. Corneal oedema & Striate keratopathy

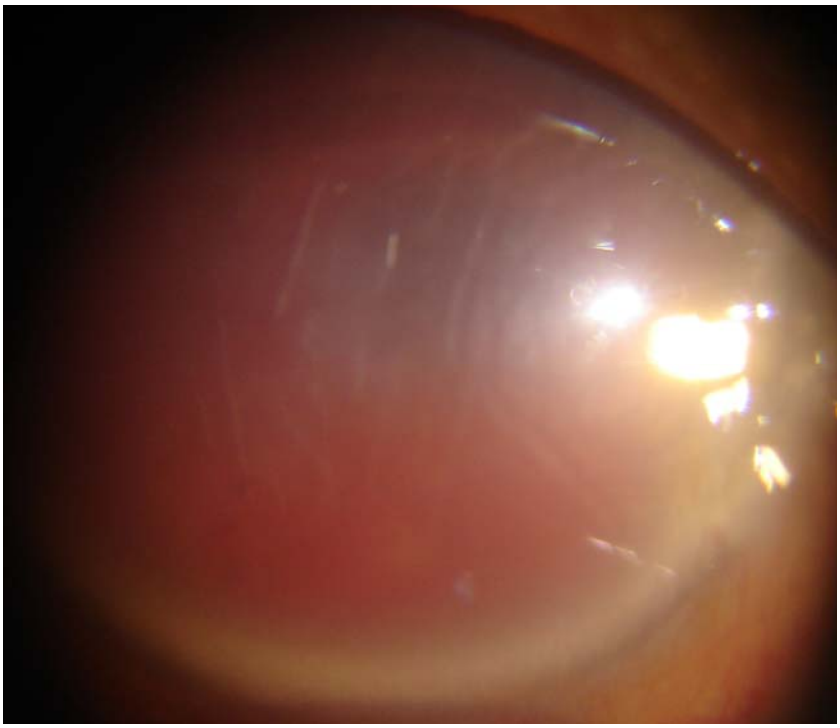

Fig. 33. Hyphaema

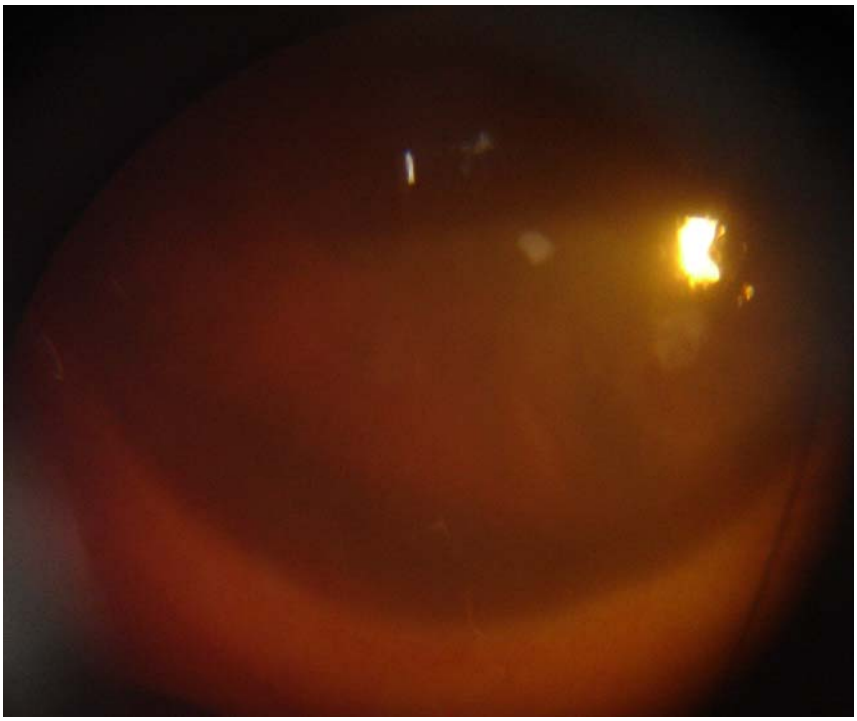

Fig. 34. Inferior iridodialysis

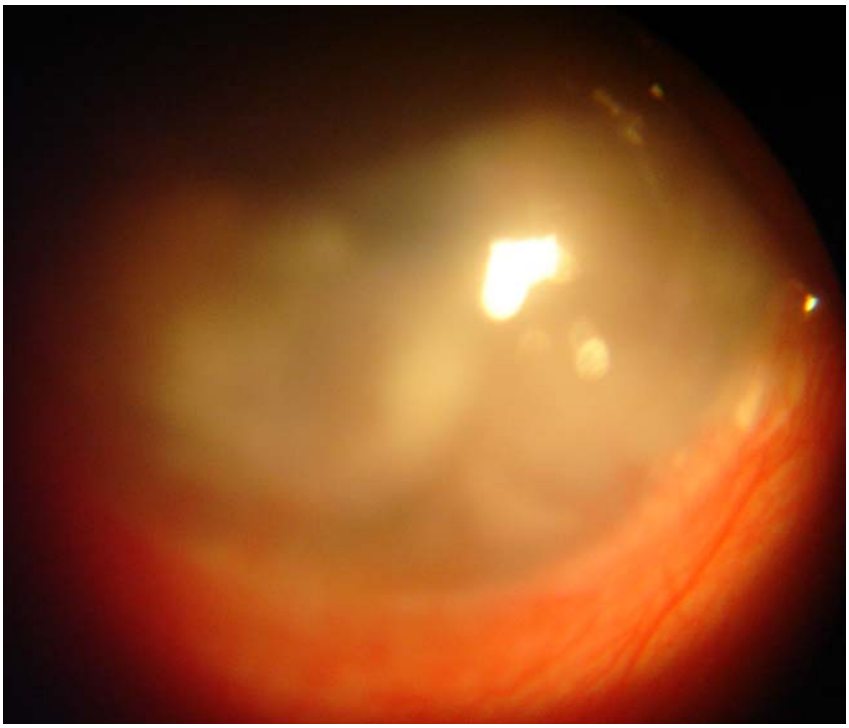

Fig. 35. Corneal oedema & Bullous keratopathy

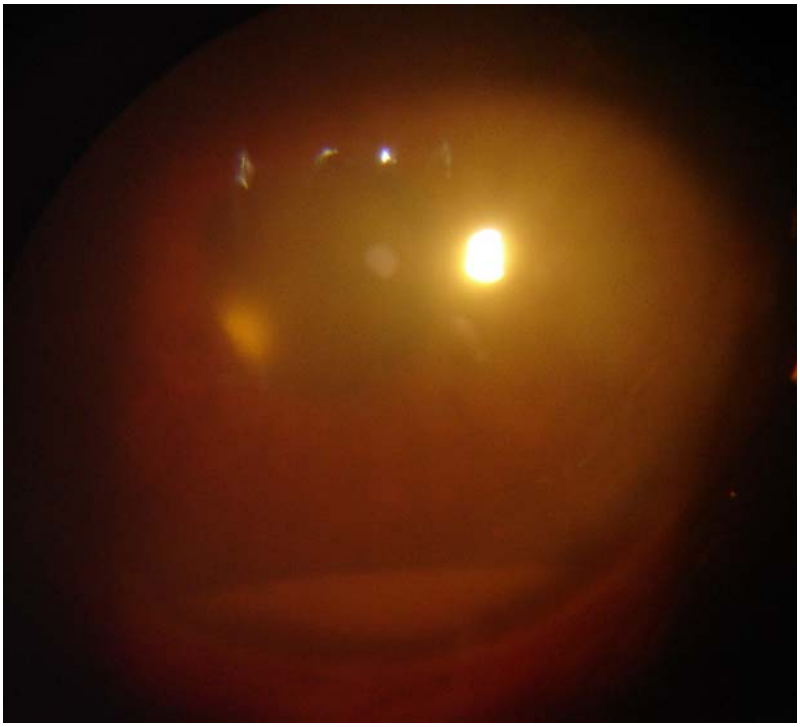

Fig. 36. Endophthalmitis

DISCUSSION

In the present study preoperative complications were seen in 12 cases. The following complication were observed

Stripping of Descemet's membrane

Descemet's membrane stripping occurred in 2 cases (2%), during introduction of 26 Gauge Cystitome in AC. The cause was that the limbal incision was too anterior.

Descemet's detachment was very small in both the cases. In order to prevent more possible damage during introduction of IOL, AC was filled up with Air.

Very large and extensive detachment is managed by resuturing with 10-0 nylon. According to study of Rosenthal [67] incidence of Descemet's membrance detachment is 4%. In 1964 Sugar[68] conducted a survey & found incidence of Descemet's detachment as 11%. Sparks[69] operated on 92 eyes in 1967 & found Descemet's detachment in 5 cases.

Hyphaema

Significant AC bleeding occurred in 1 case. Iris vessels might have been traumatized in the AC by injecting viscoelastic substance.

K Barry Mills (1977) suggested that it can be minimized by the avoidance of the limbal vessels at the 3 and 9o' clock position. A scleral tunnel initiated too far posteriorly increases the risk of transecting limbal blood vessels. According to Alpar[55] study, incidence of hyphaema is 0.7%. Flaxel & Swan[70] found 34 cases of hyphaema after operating 487 cases.

Iris prolapse

Iris prolapse found in 7 cases of which 4 cases were due to vitreous bulge, patients were heavy set individuals with thick necks, bulky and apprehensive and more or less restless. Stay sutures were released or loosened, patients were made comfortable. Iris was repositioned successfully.

In 2 cases it was due to improper insertion of upper haptic which goes behind the 12 o'clock iris .

In 1 case, patient strained during surgery which lead to sudden prolapse after implantation of IOL, which was repositioned properly.

In 1 case inferior iridodialysis was found while delivering the lens nucleus.

Posterior capsular rupture

In this study posterior capsule rupture with vitreous loss occurred in 2 cases (2%) which was higher than comparable to other reports viz, UCLA cataract surgery and Olson[64] reported 0.7% and 1 % respectively. In one case posterior capsular rupture was central and small sponge vitrectomy was done and PCIOL was placed successfully in that case . In the other one, anterior vitrectomy was done and anterior chamber IOL was implanted. Vail[71] reported 12.7% incidence of vitreous loss in 1601 eyes undergoing cataract surgery in his study. Rutllan & Barraquer[72] reported the incidence of vitreous loss of 3.7% in their study. Wilson[73] & colleagues found posterior capsular rupture in 4% cases. Skuta[74] & associates reported Posterior capsule rupture in all 5 cases they studied who had pseudo-exfoliation syndrome. Jaffe[75] & associates reported 3% incidence of vitreous loss. Guzek[76] & colleagues reported 4.9% incidence of vitreous loss.

Wound leakage

Wound leakage occurred in 1 case (1%), slightly less than the reports of UCLA cataract surgery group (1.4%) . It was successfully managed by putting another single suture and subsequent formation of AC with normal saline with 26 G needle through a separate route. Welsh[77] found 3% of cases to have wound leak after cataract surgery.

Significant corneal oedema & Sriate keratopathy

Significant corneal oedema & striate keratopathy was found in 3 cases (3%), slightly higher then the report of UCLA cataract surgery group (2.2%). Kraff[78] & associates and Sugar[68] & colleagues reported incidence of corneal oedema to be as high as 29% & 34% respectively.

Iridocyclitis

In this present study iridocyclitis found as 6% which was higher than the report of UCLA and Pretorious[66] cataract surgery study, of 1.3% and 4% respectively. Patients were treated with topical and systemic corticosteroids and cyloplegics. According to International Uveitis Study Group (IUSC)[79] incidence of iridocyclitis in their study was 3.2%. Miyake[80] & colleagues found incidence of uveitis more in cases with operative complications. Galin[81] & associates found iridocyclitis to be commonly iniciated by hypersensitivity or foreign body response.

Malposition

Post-operatively 1 case (1%) was reported to have pupil capture, it was managed by repositioning of the IOL after dilating the pupil. Lindstrom and Herman[82] reported the incidence of pupil capture to vary between 0.6% to 2.6% in their study. Rosenthal[67] found Malposition of IOL as 0.7%

Endophthalmitis

Bacterial Endophthalmitis was found in 1 case, and was treated by intra-vitreal antibiotics injection. Rosenthal[67] reported the incidence of hypopyon 0.2% in his study and Drews[83] reported endophthalmitis as 0.2-0.5%. Allen & Mangiaracine[84] found 9 out of 16,000 cases to have infection during their study in 1964-1973. Christy & Lall[85] reported 385 cases to have hypopyon out of 77,093 case studied by them in 1957-1972.

Secondary Glaucoma

Transient Hypertony was encountered in 9 cases with IOP ranging from 28-32 mmHg, & were all successfully controlled with antiglaucoma drugs. According to V lorusio et al (1990), there was 12.5% cases of transient hypertony which disappeared in 3-4 days. Kaufman[86] found increase in intra-ocular pressure by 22mm of Hg or more in 11 out 25 eyes during 1st post-operative week. Arkfeld & Jaffe[87] studied 145 cases of cataract extraction & found 41% of ECCE with rise in IOP by 25 mm of Hg or more, in 30% of Manual small incision cases IOP was increased.

Cystoid Macular Oedema

CMO was encountered in 4 cases (4%) slightly lower then the incidence of 3.4-6.7% studied by Jaffe and Claymen[88] (1978). Patients were treated with oral acetazolamide and topical flurbiprofen and topical corticosteroids drops. In all cases final visual acuity were 6/12 to 6/18 (p). Gass & Norton[89] found 2 cases to have CME after following up 48 eyes for 1year. Wright et al[90] reported 16% patient to develop CME 6weeks after surgery.

Posterior capsular opacification

The incidence of PCO in this study is 8% - developed on 5th postoperative month. Balaunowicz, Lazarczyk, Stankiewicz, Antosiok, & Mrugacz (1996) evaluated. PCO after ECCE with and without implantation of IOLS and showed PCO occurred in 37 eyes out of 107 eyes in ECCE and in 25 out of 75 eyes in patients with implantation of IOLS. Sinskey & Cain[91] reported 43% of their study patients required discission for PCO after around 26 months of surgery. Emery[92] & colleagues found PCO in 28% of their patients in 2-3 years follow up.

Recurrent Iridocyclitis and persistant corneal oedema

Both these complications were found in 1 cases (1%) in the same patient who had early postoperative iridocyclitis, this incidence is slightly higher than previous study of 0.5%. As endothelial cell count was not assessed the course could not be established.

Visual results

In this study 71% attained acuity between 6/5- 6/6 after correction, 14% attained 6/9. 8% attained 6/12 after correction, 7% attained visual acuity 6/18.

In a series of 248 cases of planned ECCE with IOL, Arrugha reported that the final visual outcome upto or better than 6/12 in 93% cases which is slightly higher than the present study.

CONCLUSION

After evaluating the incidences of various preoperative & postoperative complications of intraocular lens implantation the following conclusions were drawn:

- Some complications were iatrogenic and could be prevented.
- Surgeon has to be confident and good at both Extra capsular cataract surgery & Manual small incision cataract surgery.
- Ocular hypotony is utmost important.
- Being a wet surgery a strict asepsis is absolutely essential.
- Preserving corneal endothelial health should always be the key word.
- It is not important to clear cortex and posterior capsule over enthusiastically, instead saving posterior capsule from rupture is more important.
- Follow up records help better.
- Results improve with practice and experience.

Apart from monocular and binocular optical advantages lens implantation also helps to prevent the late pathological conditions such as vitreous contact corneal dystrophy and possibly retinal detachment in aphakics.

So it should be emphasized that 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 results of posterior chamber intraocular lens implantation in 100 eyes have been evaluated. Half of them had undergone ECCE & rest had undergone Manual SICS.

All the selected patients were examined, investigated treated and followed up for evaluating the relative merits and demerits on the intraocular lens implants.

History of evaluation of IOL implantation was briefly summarized and literature on complication of IOL implantation was reviewed.

All the facts described earlier are summarized as under:

- Age of the patients varies from 40 to 80.
- Male, female ratio is -1:1.2 approximately (total- 100)
- Majority of cases are advanced immature cataract and senile mature cataract
- The anaesthetic agents used were Bupivacaine 0.5% and Lignocaine 2% mixed with Adrenaline and Hyalase.
- Method of operation- Extracapsular cataract extraction with posterior chamber intraocular lens implantation in half of the studied patients & rest half had undergone Manual small incision cataract surgery with posterior chamber intraocular lens implantation
- Postoperative topical antibiotics, cycloplegies and corticosteriods were given to all patients.
- All cases were followed up weekly for the 1st post-op month & then monthly for at least 6 months from the date of surgery.

Peroperative complications were

Descemet's membrane detachment – 2%

Hyphaema – 1%

Iris prolapse – 7%

Iridodialysis -- 1%

Posterior capsule rupture with vitreous loss - 2%

Early postoperative complications were

Wound leakage --1%,

Significant corneal oedema with Striate keratopathy 3%,

Transient hypertony -- 9%,

Iridocyclitis -- 6%.

Bacterial Endophthalmitis -- 1%

Malposition -- 1%

Late postoperative complications were-

Recurrent iridocyclitis -- 1%

Persistant corneal oedema with Bullous keratopathy --1%

Posterior capsular opacification -- 8%,

Cystoid macular oedema -- 4%,

Late Endophthalmitis 0%.

Final visual acuity

71% achieved visual acuity 6/5- 6/6,

14% attained 6/9,

8% attained - 6/12,

7% attained - 6/18.

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PROFORMA OF THE CASE SHEET

CASE NO.:

I. PATIENT INFORMATION

Name: Age: Sex: IP No:

Religion: Occupation:

Address :

Date of Admission : Date of Operation:

Date of Discharge: Date of Examination :

Surgeon's Name: Operative Notes :

II. HISTORY

1. Chief complains:
2. History of present illness :

   Dimness of Vision Duration :

   Pain :

   Glare :

   Watering :

   Redness :

   Photophobia :

   Any treatment received before coming to the hospital :
3. Past History

   History of any previous surgery in particular eye or the other eye

   Trauma to the eye
1. Family history :

   Glaucoma, Diabetes, Hypertension, Bronchial asthma

III. GENERAL EXAMINATION

Build - Thin / average / obese

Systemic diseases - HT/DM/ Resp .

IV LOCAL EXAMINATION

Head posture

Facial symmetry

Proptosis

Enophthalmos

Orbital margins

		RE	LE
Eye Lids	**:**		
Lid edema	:		
Black eye	:		
Burn wounds	:		
Ptosis	:		
Conjunctiva	**:**		
Haemorrhage	:		
Chemosis	:		
Bruising	:		
Laceration	:		
Sclera	**:**		
Rupture of globe	:		

with prolapse of uveal tissue:

without prolapse of uveal tissue:

Incarceration of lens :

with vitreous prolapse :

without vitreous prolapse :

Cornea :

Strength of incision wound - self sealing / leaking => Siedel's test

Epithelial oedema :

Endothelial oedema :

Erosions :

Epithelial opacities :

Blood staining :

Pigmentary deposits :

Corneal lacerations :

Striate keratopathy :

Descemet's membrane detachment :

Bullous keratopathy :

Anterior Chamber :

Depth : Normal/shallow/deep :

Hyphaema : Primary / secondary Partial / Total

Hypopyon :

Abnormal contents : lens matter / IOL / Pigments / KPs

Iris :

Pattern :

Hemorrhages :

Iridodialysis :

Cyclodialysis :

Iris prolapse :

Pupil :

Size, Shape, Position :

Reaction to light : direct, indirect :

Sphincter tear :

Pupillary capture :

Pupillary membrane :

Lens :

Tears in posterior capsule:

Condition of IOL : in position / decentered :

Remnant cortical matter :

Posterior capsular opacity :

Dislocation of IOL : Anterior / posterior :

Visual Acuity :

Distant vision :

Through pin-hole :

Near vision :

Refraction :

Lacrimal Passages :

Canaliculi :

Sac :

Extraocular Movements

Intraocular Pressure :

Normal / increased / decreased :

FUNDUS EXAMINATION

a) Direct ophthalmoscopy

Media

Optic disc

Blood vessels

Macula

foveal reflex

cystoid macular edema

Background retina

Retinal detachment

b) Indirect ophthalmoscopy

V. SYSTEMIC EXAMINATION

- Repiratory system
- C.V.S.
- Per abdomen
- C.N.S.

VI INVESTIGATIONS

- Slit lamp examination - details anterior segment examination
- Gonioscopy - details angle of anterior chamber status
- Posterior segment examination using +90 D Volk lens
- Laboratory investigations
  - Conjuntival swab culture
  - Blood sugar estimation
  - Aqueous & Vitreous humour culture in Endophthalmitis
- USG B-scan-in RD/Endophthalmitis / Posterior dislocation of lens or IOL

VII. TREATMENT

- Medical
- Surgical

VIII FOLLOW UP

- Weekly for 1st post-operative month
- then Monthly till 6 months from the date of surgery

1. VISUAL ACUITY

Status quo / Improved / worsened

2. INTRAOCULAR PRESSURE

Normal / Increased / Decreased

3. REMARKS

PER OPERATIVE COMPLICATIONS IN PATIENT WITH ECCE + PCIOL I

SL. No.	NAME	AGE	SEX	I.P.No.	EYE OPERATED	DATE OF OPERATION	STRIPPING OF D M	SIGNIFICANT A C BLEEDING	IRIS PROLAPSE	IRIDODIALYSIS	P C TEAR WITH V L
1	G.B.Puttaswamy	72	M	46731	RE	06/12/2004	-	-	-	-	-
2	Kalamma	45	F	46407	LE	06/12/2004	-	-	-	-	-
3	Subbamma	40	F	47540	LE	30/6/2004	-	-	-	-	-
4	Kempamma	58	F	47765	RE	14/7/2004	-	+	+	-	-
5	K.M.Mallikarjuna	75	M	48082	LE	14/7/2005	-	-	-	-	-
6	Nagamma	42	F	48762	RE	30/7/2004	-	-	-	-	-
7	Shivvannegowda	55	M	48742	RE	30/7/2004	-	-	-	-	-
8	Devegowda	50	M	48761	RE	08/03/2004	-	-	-	-	-
9	Doddalingegowda	58	M	48891	RE	08/03/2004	-	-	-	-	
10	Bommamma	60	F	49993	LE	15/9/2004	-	-	-	-	-
11	Javaramma	66	F	50707	LE	15/9/2004	-	-	-	-	-
12	Javaregowda	67	M	57347	LE	15/9/2004	-	-	-	-	+
13	Ramegowda	79	M	57654	RE	28/9/2004	-	-	-	-	-
14	Rangappa	65	M	57363	RE	28/9/2004	-	-	-	-	-
15	Puttananjappa	59	M	58342	RE	28/9/2004	-	-	-	-	-
16	Chikkamma	65	F	57543	RE	28/9/2004	-	-	+	-	-
17	Channamma	52	F	56890	RE	29/9/2004	-	-	-	-	-
18	Huchamma	60	F	51380	RE	29/9/2004	-	-	-	-	-
19	Gundaiah	56	M	57987	RE	29/9/2004	+	-	-	-	-
20	Gangappa	59	M	53478	LE	29/9/2004	-	-	-	-	+
21	Boregowda	65	M	52178	LE	10/09/2004	-	-	-	-	-
22	Ramegowda	58	M	52367	LE	10/09/2004	-	-	-	-	-
23	Hanumaiah	65	M	52543	RE	10/09/2004	-	-	-	-	-
24	Thimmegowda	53	M	52763	RE	26/10/2004	-	-	-	-	-
25	Manjaiah	75	M	59653	RE	26/10/2004	-	-	-	-	-

SL. No.	NAME	AGE	SEX	I.P.No.	EYE OPERATED	DATE OF OPERATION	STRIPPING OF D M	SIGNIFICANT A C BLEEDING	IRIS PROLAPSE	IRIDODIALYSIS	P C TEAR WITH V L
26	Anjanappa	73	M	53789	RE	26/10/2004	-	-	-	-	-
27	Mayappa	68	M	56789	RE	28/10/2004	-	-	+	-	-
28	Shankare Gowda	58	M	53553	RE	28/10/2004	-	-	-	-	-
29	Mayamma	51	F	56745	LE	28/10/2004	-	-	-	-	-
30	Muddaiah	53	M	58723	RE	11/06/2004	-	-	-	-	-
31	Thimappa	68	M	54134	RE	6/11/2004	-	-	-	-	-
32	Shankaraiah	72	M	52869	RE	16/11/2004	-	-	+	-	-
33	Nanjamma	75	F	55499	LE	16/11/2004	-	-	-	-	-
34	Siddagangamma	55	F	55495	LE	23/11/2004	-	-	-	-	-
35	Mallavva	84	F	55446	LE	23/11/2004	-	-	-	-	-
36	Erre gowda	53	M	56775	RE	23/11/2004	-	-	-	-	-
37	Lakshmamma	60	F	55553	RE	16/12/2004	-	-	-	-	-
38	Kengamma	53	F	55489	RE	16/12/2004	-	-	-	-	-
39	Puttamma	50	F	55629	RE	20/12/2004	-	-	-	-	-
40	Gowramma	51	F	55501	LE	20/12/2004	-	-	-	+	-
41	Rathnamma	66	F	55520	LE	20/12/2004	-	-	-	-	-
42	Shive Gowda	70	M	55533	RE	20/12/2004	-	-	-	-	-
43	Javare Gowda	65	M	55513	RE	20/12/2004	-	-	-	-	-
44	Kamalamma	51	F	56731	RE	26/12/2004	-	-	+	-	-
45	Yellamma	81	F	61348	LE	26/12/2004	-	-	-	-	-
46	Sakamma	70	F	56983	RE	26/12/2004	-	-	-	-	-
47	Devegowda	70	M	61001	LE	26/12/2004	-	-	-	-	-
48	Bettegowda	52	M	60039	LE	30/12/2004	-	-	-	-	-
49	Manjaiah	67	M	61003	LE	30/12/2004	-	-	-	-	-
50	Lakshmamma	80	F	62009	LE	30/12/2004	-	-	-	-	-

LATE POST-OPERATIVE COMPLICATIONS IN CASES WITH MANUAL SICS + PC IOL I

SL. No.	NAME	AGE	SEX	I.P.No.	EYE OPERATED	DATE OF OPERATION	RECURRENT IRIDO CYCLITIS	PERSISTANT CORNEAL OEDEMA & B K	P C O	C M E	LATE ENDOPHTHALMITIS	B C V A AFTER 6 MONTHS OF SURGERY
1	Kalamma	52	F	64890	RE	01/02/2005	-	-		-	-	6/6
2	Ramegowda	59	M	64578	RE	01/02/2005	-	-		-	-	6/6
3	Rangappa	74	M	65432	RE	01/12/2005	-	-		-	-	6/6
4	Puttananjappa	55	M	63490	RE	01/12/2005	-	-		-	-	6/6
5	Gundaiah	65	M	64358	RE	20/1/2005	-	-		-	-	6/6
6	Gangappa	57	M	64598	RE	20/1/2005	-	-		-	-	6/6
7	Boregowda	64	M	65540	RE	20/1/2005	-	-		-	-	6/6
8	Ramegowda	58	M	65590	LE	25/1/2005	-	-		-	-	6/9
9	Hanumaiah	68	M	66123	LE	25/1/2005	-	-		-	-	6/6
10	Lakshamma	62	F	66345	LE	25/1/2005	-	-		-	-	6/6
11	Thimmegowda	68	M	66342	LE	25/1/2005	-	-		-	-	6/6
12	K.N.Krishnegowda	55	M	66785	LE	28/1/2005	-	-		-	-	6/6
13	Ningamma	64	F	66981	LE	28/1/2005	-	-		-	-	6/12
14	Manjaiah	68	M	66342	LE	02/02/2005	-	-		-	-	6/6
15	Javaraiah	66	M	66324	LE	02/02/2005	-	-		-	-	6/9
16	Sannappa	56	M	66343	LE	02/02/2005	-	-		-	-	6/9
17	Yellamma	53	F	66789	LE	15/2/2005	-	-		-	-	6/12
18	Sakamma	49	F	66987	LE	15/2/2005	-	-		-	-	6/6
19	Subbamma	68	F	66990	LE	21/2/2005	-	-		-	-	6/6
20	Kempamma	69	F	66843	LE	21/2/2005	-	-		-	-	6/6
21	Nagamma	75	F	67543	RE	21/2/2005	-	-		-	-	6/9
22	Chikkamma	59	F	67458	RE	21/2/2005	-	-		-	-	6/6
23	Bommamma	78	F	67832	RE	26/2/2005	-	-		-	-	6/6
24	Javaramma	81	F	67990	RE	26/2/2005	-	-		-	-	6/18
25	Channamma	56	F	67998	RE	26/2/2005	-	-		-	-	6/6

SL. No.	NAME	AGE	SEX	I.P.No.	EYE OPERATED	DATE OF OPERATION	RECURRENT IRIDO-CYCLITIS	PERSISTANT CORNEAL OEDEMA & B K	P C O	C M E	LATE ENDOPHTHALMITIS	B C V A AFTER 6 MONTHS OF SURGERY
26	Huchamma	69	F	68001	RE	28/2/2005	-	-		-	-	6/6
27	Kenchaiah	90	M	68004	RE	28/2/2005	-	-		-	-	6/6
28	Javaregowda	85	M	68006	RE	28/2/2005	-	-		-	-	6/6
29	Shivalingegowda	78	M	68354	RE	03/05/2005	-	-		-	-	6/9
30	Kempanna	72	M	68377	RE	03/05/2005	-	-		-	-	6/6
31	Gous Khan	71	M	68543	RE	03/05/2005	-	-		-	-	6/9
32	Thimmappa	63	M	69003	LE	03/05/2005	-	-		-	-	6/6
33	Manjamma	69	F	69339	LE	03/12/2005	-	-		-	-	6/9
34	Shivalingamma	73	F	69099	LE	03/12/2005	-	-		-	-	6/6
35	Javaramma	79	F	69567	LE	23/3/2005	-	-		-	-	6/12
36	Sannamma	74	F	69668	LE	23/3/2005	-	-		-	-	6/6
37	Amir Banu	56	F	69779	LE	29/3/2005	-	-		-	-	6/6
38	Bettamma	68	F	69881	LE	29/3/2005	-	-		-	-	6/6
39	Ningamma	82	F	69008	LE	04/03/2005	-	-		-	-	6/6
40	Boramma	59	F	69989	LE	3/4/2005	-	-		-	-	6/6
41	Rangamma	67	F	70021	LE	04/03/2005	-	-		-	-	6/6
42	Nagamma	82	F	70036	LE	13/4/2005	-	-		-	-	6/12
43	Javaraiah	53	M	70067	RE	13/4/2005	-	-		-	-	6/6
44	Karim Khan	56	M	70123	RE	23/4/2005	-	-		-	-	6/6
45	Gangamma	52	F	70267	RE	23/4/2005	-	-		-	-	6/9
46	Thimmamma	58	F	71221	RE	05/03/2005	-	-		-	-	6/6
47	Doddalingamma	75	F	71324	RE	15/5/2005	-	-		-	-	6/6
48	Puttananjamma	65	F	72201	LE	23/5/2005	-	-		-	-	6/9
49	Hanummamma	85	F	72339	LE	30/5/2005	-	-		-	-	6/6
50	Chikkamma	65	F	73233	RE	30/5/2005	-	-		-	-	6/6

KEY TO MASTER CHART

SL.No.	=	Serial Number
IP.No.	=	Inpatient Number
D.M.	=	Descemet's Membrane
A.C.	=	Anterior Chamber
P.C.	=	Posterior Capsule
V.L.	=	Vitreous Loss
S.K.	=	Striate Keratopathy
B.K.	=	Bullous Keratopathy
P.C.O.	=	Posterior Capsular Opacity
C.M.E.	=	Cystoid Macular Edema
B.C.V.A.	=	Best Corrected Visual Acuity