A STUDY OF CATARACT SURGERY, ITS COMPLICATIONS & VISUAL OUTCOME IN DIABETICS

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Certificate

This is to certify that the dissertation entitled "**A STUDY OF CATARACT SURGERY, ITS COMPLICATIONS & VISUAL OUTCOME IN DIABETICS"** is the bonafide original work of **Dr. G.J. VASUKI** in partial fulfillment of the requirements for **M.S. Branch – III (Ophthalmology)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in September 2006.

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Declaration

I, **Dr. G.J. VASUKI,** solemnly declare that dissertation titled, "**A STUDY OF CATARACT SURGERY, ITS COMPLICATIONS & VISUAL OUTCOME IN DIABETICS"** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2003-2006 under the expert guidance and supervision of **Prof. A. PRIYA, M.S., D.O.** Head of the Department, Department of Ophthalmology.

The dissertation is submitted to The Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.S. Degree (Branch – III) in Ophthalmology.**

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ABBREVIATIONS

AC	:	Anterior Chamber
ACIOL	:	Anterior Chamber Intraocular Lens
ADVS	:	Activities of Daily Vision Scale
ARMD	:	Age Related Macular Degeneration
ATP	:	Adenosine Triphosphate
BCVA	:	Best Correct Visual Acuity
CME	:	Cystoid Macular Oedema
CMO	:	Cystoid Macular Oedema
DM	:	Diabetes Mellitus
DNA	:	Deoxyribo Nucleic Acid
ECCE	:	Extra Capsular Cataract Extraction
ECG	:	Electrocardiogram
Echo	:	Echocardiogram
GA	:	General Anaesthesia
Glut	:	Glucose Transporter
HANES	:	Health and Nutrition Examination Survey
Hb	:	Haemoglobin
HbA1C	:	Glycosylated Haemoglobin
HMP	:	Hexose Monophosphate
HT	:	Hypertension
ICCE	:	Intra Capsular Cataract Extraction
IOP	:	Intra Ocular Pressure
KD	:	Kilo Dalton
Km	:	Michaelis Constant
LA	:	Local Anaesthesia
MIP	:	Major Intrinsic Protein

MODY	:	Maturity Onset Diabetes Mellitus
NAD	:	Nicotinamide Dinucleotide
NADH	:	Reduced Nicotinamide Dinucleotide
NADP	:	Nicotinamide Dinucleotide Phosphate
NADPH	:	Reduced Nicotinamide Dinucleotide Phosphate
NPDR	:	Nonproliferative Diabetic Retinopathy
NPE	:	Non-pigmented Epithelium
OHA	:	Oral Hypoglycemic Agent
PAS	:	Peripheral Anterior Synechiae
PCIOL	:	Posterior Chamber Intraocular Lens
PCO	:	Posterior Capsular Opacification
PCR	:	Posterior Capsular Rent
PDR	:	Proliferative Diabetic Retinopathy
POAG	:	Primary Open Angle Glaucoma
PRP	:	Pan Retinal Photocoagulation
PSCC	:	Posterior Sub-Capsular Cataract
RAPD	:	Relative Afferent Pupillary Defect
RBH	:	Retrobulbar Haemorrhage
RIOP	:	Raised Intra Ocular Pressure
RNA	:	Ribonucleic Acid
SCH	:	Subconjunctival Haemorrhage
SICS	:	Small Incision Cataract Surgery
UV	:	Ultraviolet
V/A	:	Visual Acuity
VEGF	:	Vascular Endothelial Growth Factor

KEY TO MASTER CHART

1	-	Male
2	-	Female
3	-	Right Eye
4	-	Left Eye
5	-	Both Eyes
A	-	Aphakia
AC	-	Anterior Chamber Intra Ocular Lens
ACG	-	Angle closure Glaucoma
AL	-	Allergy
ARMD	-	Age Related Macular Degeneration
AS	-	Asthma
BC	-	Bread Crump Appearance
BDR	-	Background Diabetic Retinopathy
C	-	Cortical Cataract
CC	-	Calcified Capsule
D	-	Diet
E	-	Extra Capsular Cataract Extraction
EV	-	Evisceration
Ex	-	Excision
He	-	Haemorrhage
HM	-	Hypermature Cataract
HT	-	Hypertension
HY	-	Hyphaema
HYP.	-	Hypothyroidism
I	-	Insulin
IHD	-	Ischemic Heart Disease
IM	-	Immature Cataract
IOM	-	Intraoperative Miosis

IP	-	Iris Prolapse
M	-	Mature Cataract
Mod	-	Moderate
N	-	Nuclear Cataract
NPDR	-	Non-proliferative Diabetic Retinopathy
O	-	Oral Hypoglycemic agents
OBE	-	Obesity
P	-	Posterior Subcapsular Cataract
PC	-	Posterior Chamber Intra Ocular Lens
PCR	-	Posterior Capsular Rent
PI	-	Peripheral Iridectomy
PLG	-	Phacolytic Glaucoma
PMG	-	Phacomorphic Glaucoma
PO LU	-	Polychromatic Lustre
POAG	-	Primary Open Angle Glaucoma
PR	-	Pigment Release
Prgn	-	Progression
PS	-	Pseudophakia
PSC	-	Posterior subcapsular Cataract
PT	-	Pterygium
RLM	-	Retained Lens Material
RP	-	Repositioning
Rpty	-	Retinopathy
RU	-	Recurrent Uveitis
S	-	Small incision cataract surgery
SN	-	Snow Flake
T	-	Trabeculectomy
VD	-	Vitreous Disturbance
ZD	-	Zonulo Dialysis

CONTENTS

INTRODUCTION

Diabetes mellitus is a ubiquitous disease with an ancient history. It is common, being present in approximately 3% of adult population. Diabetes is an important cause of blindness in people of working age. Although we often think of retinal changes, it must not be forgotten that other parts of the visual system are affected in diabetics. Diabetes increases the possibility of cataract and is the next common cause blindness after diabetic retinopathy. Cataract is a common complication of diabetes indeed it has been estimated that upto 15% of cataract surgery is performed on diabetics.

The global prevalence of type 2 DM is expected to double in the period 2000 – 2025 and may reach a level of 300 million people. In India the urbanization factor, producing lifestyle changes that adversely affect metabolism is causing a large increase in the number of diabetic patients[14,38]. Diabetes occurs at the younger age (45-64 years) prevalence in urban Indian adults was increased from 5.2% in 1984 to 13.9% in 2000. The prevalence in Chennai was 11.6%. There are approximately 33 million adults with diabetes in India and it likely to increase to 57.2 million by the year 2025. The number of diabetics is on the rise and eventually diabetic cataract.

AIMS AND OBJECTIVES

The aim of the study is to find the incidence of cataract with regard to age, sex, the complications of the cataract surgery, (Per-operative and post-operative) and visual outcome after surgery in a diabetic population.

REVIEW OF LITERATURE

Diabetes mellitus - an overview

Diabetes Mellitus is defined as a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The American Diabetes Association Expert Panel recommends a diagnosis of diabetes mellitus when one of the three criteria is met.

Criteria for the diagnosis of Diabetes Mellitus

1) Symptoms of diabetes plus casual plasma glucose concentration ≥ 200 mg/dl.

   Casual is defined as anytime of day without regard to time since last meal. The classic symptoms of diabetes include polyuria, poly dipsia and unexplained weight loss.

2) Fasting plasma glucose ≥ 126 mg/dl. Fasting is defined as no caloric intake for at least 8 hours.

3) 2 hours plasma glucose ≥ 200 mg/dl during an oral glucose tolerance test.

DIABETES MELLITUS CLASSIFICATION

The new classification identifies four types of diabetes mellitus[36].

Type 1, Type 2, "Other specific types" and gestational diabetes.

Type 1 : (Formerly IDDM / Juvenile onset DM) is characterized by β-cell destruction due to autoimmune process leading to absolute insulin deficiency.

Type 2 : (NIDDM / Adult onset) is characterized by insulin resistance in peripheral tissues and an insulin secretary defect of the β cells.

Other Specific Types : includes persons with genetic defects of β cell function (MODY or Maturity onset Diabetes Mellitus) or with defects of insulin action. Persons with diseases of exocrine pancreas, dysfunction associated with endocrinopathies.

Gestational Diabetes Mellitus

PATHOGENIC MECHANISMS IN DIABETIC EYE DISEASE

1) Protein glycosylation
2) Aldose reductase activity
3) Glycosylated hemoglobin

↓

Relative tissue hypoxia → final common pathway

I. PROTEIN GLYCOSYLATION :

A nonenzymatic reaction of glucose with lysine residue of proteins. A high level of serum glucose is necessary to produce this type of protein glycosylation. The resulting chemical compound rearranges and is transformed into an irreversible structure called ‘Amadori’ product. Proteins and Amadori product form ‘Advanced glycosylation end products’ which causes release of biologically active molecules that cause microvascular changes similar to diabetic retinopathy.

II. ALDOSE REDUCTASE ACTIVITY

In the presence of high concentration of sugars, aldose reductase enzyme converts sugars to alcohols. Pericytes and endothelial cells of retinal vasculature have high concentration of this enzymes. Therefore an increase in blood glucose leads to an increase in alcohols in the pericytes and endothelial cells, which in turn cause death of these cells. Aldose reductase enzyme and its cellular after effects have been implicated in ocular problems associated with Diabetes mellitus.

III. GLYCOSYLATED HAEMOGLOBIN :

High blood glucose leads to increase in glycosylated haemoglobin. Release of oxygen from glycosylated Hb is slow and difficult compared to non-glycosylated Hb. This leads to a relative tissue hypoxia. Glycosylated Hb stimulates VEGF release.

Glycosylated Hb ≤ 7 prevents the development of diabetic retinopathy or slows the progression.

Anatomy and physiology of the lens

The crystalline lens is a transparent, biconvex structure whose functions are to

- Maintain its own clarity
- Refract light
- Provide accommodation

There is lack of blood supply and innervation to the lens and it depends entirely on the aqueous humor for its metabolic requirements. The lens is suspended by the zonules of zinn and lies posterior to the iris and anterior to the vitreous body. Refractive Index is 1.4 in the center and 1.36 peripherally. The lens contributes about 15-20 diopter of the refractive power of the eye.

The anterior and posterior poles of the lens are joined by an imaginary line called the axis. The greatest circumference of the lens is the equator. The crystalline lens grows throughout human life and weight about 255 mg in an adult. The lens capsule is an elastic, transparent basement membrane made of Type IV collagen. The zonular

lamella, which is the outer layer, gives attachment to the zonular fibers. The lens capsule is thickest in the anterior and posterior preequatorial zones and thinnest in the region of the central posterior pole. The zonules arising from the basal laminae of the NPE of the pars plana and pars plicata of the ciliary body support the lens. They insert on the lens capsule in the equatorial region.

Epithelium:

A single layer of epithelial cells are seen behind the anterior capsule. Epithelium is the most metabolically active structure in the lens. Its activities include synthesis of DNA, RNA, Protein, lipid and ATP generation. The epithelial cells in S-phase occur in a ring around the anterior lens, the germinative zone. These newly formed cells migrate towards the equator, where they differentiate into fibers. The epithelial cells migrate toward the low region of the lens, where the process of terminal differentiation into lens fibers takes place. While the epithelial cells elongate to form lens fibers, there is a tremendous increase in cellular proteins, loss of organelles (nuclei, mitochondria and ribosomes) and becomes dependent on glycolysis for energy.

Nucleus & cortex:

The oldest fibers are the central most, as the new fibers laid down, crowd and compact the previously formed fibers. Embryonic and

fetal nuclei persist in the center of the lens. Recently formed fibers make up the cortex. Anterior and posterior sutures are due to the interdigitations of the apical cell process and basal cell processes respectively.

Lens physiology and biophysics

Mechanisms that control the water and electrolyte balance which is most important in maintaining the transparency of the lens, forms the basic physiology. 66% Water and 33% Protein. i.e. (Cortex is more hydrated)

The lens epithelium is the metabolically most active site. The levels Na^+ and Cl^- ions are lower and K^+ ions are higher in the lens compared to the external milieu. The ionic balance is maintained by the Na^+-K^+-ATpase pump located in the cell membranes of the lens epithelium and each lens fiber.

Pump -leak theory

Active transport and membrane permeability together is often referred as the pump-leak system of the lens. According to the pump-leak theory potassium, amino acids and various other molecules are transported into the anterior lens through the epithelium. An anteroposterior ionic gradient occurs according to this theory. Potassium is more in the anterior fibers and sodium in the posterior cortex. Na^+,

K^+-ATpase activity is greatest in the epithelium. Two K^+ ions are pumped into the cell as three Na^+ ions are pumped out.

Amino acid & sugar transport:

Most of the amino acids are transported into the lens from the aqueous against a concentration gradient as part of the pump leak concept. Three different pumps one each for acidic, basic and neutral amino acids have been reported.

Glucose → Transport — Simple diffusion + facilitated diffusion

Calcium homeostasis is through a Ca^{2+}–ATpase pump which maintains a large transmembrane calcium gradient. Intracellular pH of lens = 7.

Electrophysiology:

Resting Membrane potential = –50mv at 20 yrs

= –20mv at 80 yrs

Lens transparency:

The crystalline lens is transparent because of

- Absence of chromophores in young lens that absorbs visible light.
- Highly organized regular lamellar conformation of the lens proteins, that causes minimal light scatter.
- Absence of organelles in the mature lens fibers.

- Less fibers form a hexagonal lattice and the amount of intracellular space is small.
- The difference in Refractive index between different structures of the lens is negligible.

Light transmittance:

The lens absorbs

- UV – B → 300 – 315 nm
- UV – A → 315 – 400 nm and also absorbs infrared radiation.

The transmittance of young lens begins at about 310nm and reaches 90% at 450nm, compared with older lens > 60 years, begins transmitting at 400nm but does not reach 90% total transmittance until 540nm.

Refractive indices:

- Peripheral cortex → refractive index = 1.386
- Nucleus → refractive index = 1.41

Accommodation:

The mechanism by which the eye changes focus from distant to near images, is produced by a change in lens shape resulting from action of the ciliary muscle on the zonular fibers.

BIOCHEMISTRY OF THE LENS

Lens proteins:

Proteins in lens accounts for 33% of its wet weight, which is twice that of other tissues. Intracellular proteins are of two types, water soluble and water insoluble. Water soluble are α crystallins and β crystallins

α Crystallins:

- 32% of lens protein
- Largest → 600 – 800 KD molecular weight
- Specifically involved in the transformation
- Of epithelial cells into lens fibers.

β Crystallins:

- 55% of water soluble proteins of lens
- complex group of oligomers composed of polypeptides from 23-32KD

γ Crystallins:

- Monomeric proteins with molecular mass 20 KD
- Concentrated in the nucleus
- γ Crystalline → adult lens cortex
- Taxon specific crystallins are also present.

Cytoskeletal and membrane proteins:

The water insoluble proteins form the major cytoskeletal and membrane elements. They include actin, vimentin, tubulin, filensin and phakinia. Major intrinsic protein (MIP) form 50% of the membrane protein.

Post translational modifications:

The lens proteins are the longest links in the human body. These proteins become structurally modified in a variety of ways → oxidation of sulphur and aromatic residue side chains, polypeptide, cross-links, glycosylation, racemization etc.

Glucose metabolism of the lens:

Glucose metabolism is the main source of energy.

Utilization of Glucose is by

- 80% - Glycolytic pathway
- 10% - HMP shunt
- 5% - Sorbitol Pathway
- 5% - Gluconic acid

Due to its avascularity and location in the ocular humors, the lens exists in a hypoxic environment. 70% of lens ATP is derived from anaerobic glycolysis. 3% per molecule of glucose passes into the Kreb's cycle, which generates 25% of lens ATP. Metabolism of one molecule

of glucose by anaerobic glycolysis yields only 2 molecules of ATP where as 36 molecules are produced in aerobic glycolysis. HMP shunt utilizes glucose 6 phosphate and produces NADPH and pentose. NADPH is needed to maintain lens glutathione in reduced state and in sorbitol pathway.

Protein metabolism:

Proteins are synthesized from the free amino acids that are transported from the aqueous occurs only in the epithelial cells and surface cortical fibers.

Antioxidant mechanisms:

Reactive oxygen species can easily damage lipids, proteins, carbohydrates and nucleic acids. Such radicals include super oxide anion, hydroxyl free radical, hydroperoxyl radicals, lipid peroxyl radicals etc. Protection against such damage is by antioxidants like super oxide dismutase, ascorbate, catalase or glutathione peroxidase.

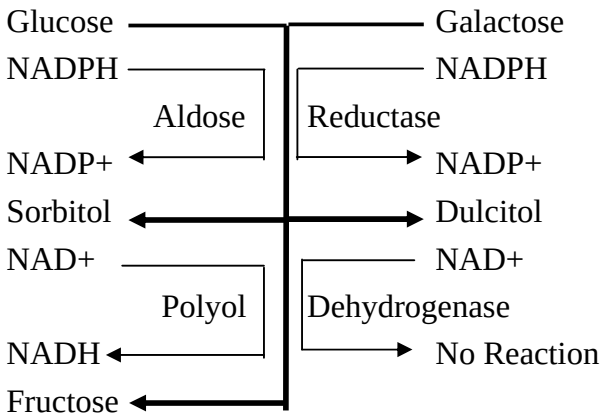
CATARACT FORMATION IN DIABETES

Pathogenesis:

(i) **Sorbitol pathway and osmotic hypothesis** :

Aldose reductase is the key enzyme in the pathway that converts sugars into the corresponding sugar alcohols. Aldose reductase has a very high Km for glucose / galactose. Under normal condition little or

no activity occurs through this pathway. In hyperglycemic state, aldose reductase competes with hexokinase for glucose.



This leads to an increase in intracellular sorbitol. It is unable to penetrate cell membranes and trapped inside the cells. Under conditions of hyperglycemia, this creates an osmotic pressure that draws water into the lens. The levels of polyol dehydrogenase are much higher in human lenses compared to aldose reductase.

Swelling of cells
↓
Damage to membranes
↓
Formation Foci of interfibrillar and intracytoplasmic light scattering
↓
Cataract Formation

Non -enzymatic glycosylation:

Non-enzymatic glycation of proteins by the Maillard reaction results in the formation of advanced glycation end products that induce

cataract formation. There is a conformational alteration progressing to thiold oxidation, aggregation, formation of disulphide and other covalent cross links and inactivation of enzymes.

Other mechanisms suggested

- carbamylation of crystallins
- Auto oxidation of sugars.

Lens changes in diabetes:

Diabetics have lenses that are larger than normal for age. The lenses of an adult diabetic are said to be in the same condition as the lenses of a non-diabetic who is 15 years older. The refractive index of the lens is altered due to increased hydration. Patients with diabetes show transient refractive changes due to changes in their blood sugars. Acute myopic shifts may indicate undiagnosed or poorly controlled diabetes. Changes in the hydration of the lens and glycogen deposits in the ciliary body decrease the ciliary body's ability to function. This leads to a decreased amplitude and presbyopia may present at an earlier age in patients with diabetes than in non-diabetics.

MORPHOLOGY OF DIABETIC CATARACT:

I. Snowflake or true diabetic cataract:

- < 30 yrs age, bilateral, uncontrolled D.M.

- widespread sub capsular changes of abrupt onset and acute progression.
- Multiple gray white sub capsular opacities that have a snowflake appearance, seen initially in the superficial anterior and posterior lens cortex.
- Total opacification can occur over a period of few weeks.

II. Polychromatic type:

- Fine needle – shaped polychromatic cortical opacities may also form.

III. Senescent cataract:

- Frequently observed in diabetes. The Health and Nutrition examination survey and the Framingham eye study found an incidence of typical senile cataracts to be increased by a factor of 3 or more in diabetic individuals compared with age matched non-diabetics. Lens changes tend to occur at a younger age than in patients without diabetes.

1. Cortical cataracts

- Bilateral but often asymmetric
- Vary in rate of progression

- The first sign is visible with slit lamp biomicroscope – vacuoles and water clefts in the anterior or posterior cortex. Cortical lamellae may be separated by fluid.
- Wedge – shaped opacities (cortical spokes / cuneiform opacities) form near the periphery of the lens, with the pointed end of the opacities oriented towards the center. It appears as white opacities when viewed with slit lamp and as dark shadows when viewed by retro illumination.
- As the lens continues to take up water it may swell and become an **Intumescent** cortical cataract.
- The entire cortex from the capsule to the nucleus becomes white and opaque, to form a **mature cataract**.
- Hypermature cataract occurs when degenerated cortical material leaks through the lens capsule, leaving the capsule wrinkled and shrunken.
- Morgagnian cataract occurs due to liquefaction of the cortex allows free movement of the nucleus within the capsular bag.

2. Posterior sub capsular cataract:

- PSCC are located in the posterior cortical layer and usually axial.
- The first indication of PSC formation is a subtle iridescent sheen in posterior cortical layers.

- Often seen in younger patients
- HPE shown posterior migration of lens epithelial cells in PSC area with aberrant enlargement. The swollen epithelial cells are called wedl or bladder cells. (Dysplastic changes in germinal epithelium)

3. Nuclear cataract:

- An excessive amount of sclerosis and yellowing is called **Nuclear Cataract** that causes a central opacity.
- Rate of progression is slow.
- Advanced cases, lens nucleus becomes opaque and brown and are called a brunescent nuclear cataract.

SYMPTOMS AND SIGNS OF DIABETIC CATARACT

1) Decreased visual acuity :

The patient often tells that he has visual impairment. Different types of cataract have different effects on visual acuity, rapid onset of decreased vision occurs in true diabetic cataract.

2) Myopic shift :

The development of nuclear sclerosis leads to an increase in the dioptric power of the lens that causes myopia. Hyperopic presbyopic

patients find their need for distances glasses diminished and this is called second sight

3) Glare :

Patients complain of increased glare sensitivity, which may vary from a decrease in contrast sensitivity in bright environment to disabling glare when looking at car headlights at night.

4) Contrast sensitivity :

It may demonstrate a significant loss of visual function not appreciated with snellen testing. However it is not a specific indicator of visual loss due to cataract.

5) Monocular Diplopia / Polyopia :

Sometimes nuclear changes are localized to the inner layers of the lens nucleus, resulting in multiple refractile areas in the center of the lens. This results in monocular diplopia, including a ghost images and occasionally a true second image.

EFFECT OF CATARACT ON VISUAL ACUITY

Type of cataract	Growth Rate	Glare	Effect on distance	Effect on near	Induced myopia
Cortical	2+	1+	1+	1+	None
Nuclear	1+	1+	2+	None	2+
Posterior sub capsular	3+	3+	1+	3+	None

INDICATIONS FOR CATARACT SURGERY

1) Patient's desire for improved visual function : It is not based on a specific level of visual acuity. Both the patient and ophthalmologist determine if reduced visual function interferes substantially with desired activities. ADVS (Activities of Daily Visional Scale) may be used.
2) Loss of stereopsis, diminished peripheral vision, disabling glare or symptomatic anisometropia are indications for surgery in monocular cataract.
3) Medical indications:
    a. Phacolytic glaucoma
    b. Phacomorphic glaucoma,
    c. Phacoantigenic uveitis
    d. Dislocation of lens into the anterior chamber.

For diagnosis or management of diabetic retinopathy or other ocular diseases when a dense cataract obscures the fundus view.

PRE OPERATIVE EVALUATION IN DIABETICS

GENERAL HEALTH

There is an association with other systemic diseases like Ischaemic heart disease, systemic hypertension, obesity and diabetes. All medical problems especially prodiabetic states, should be evaluated and treteated appropriately. Drug sensitivity and allergies should be documented. Musculo skeletal disorders that limit the ability to lie comfortably, body habitus and head tremor should be taken care.

GLYCEMIC CONTROL

The stress of surgery exacerbates the metabolic abnormalities. So meticulous attention to plasma glucose control is necessary. The goal of treatment is to prevent hypoglycemia and ketoacidosis, by keeping the plasma glucose levels between 150 – 250mg/dL and to maintain fluid and electrolyte balance. If plasma glucose exceed 300mg/dL additional regular insulin is given and rate of glucose infusion is increased if plasma glucose is less than 150mg/dL. Patients treated with diet may require small doses of regular insulin if blood sugar exceeds 300mg / dL.

Patients treated with sulphonyl ureas, the drug should be omitted on the day of surgery. Insulin may be added, with respect to the plasma glucose levels. In insulin dependent patients undergoing cataract surgery, the morning insulin dose may be delayed until the procedure is completed and the patient is fed. HbA1c gives the exact status regarding glycemic control in the recent past.

OCULAR EVALUATION :

Diabetic patients are more prone to develop infective conditions of the ocular adnexa. Blepharitis, as manifested by collarettes, marginal eye lid thickening, inspissation of meibomian gland secretions should be diagnosed and treated.

Tear film abnormalities are to be assessed, as it has been reported that there is a decreased secretion and less uniform lipid layer and reduced tear breakup time which can delay post-operative recovery. Diabetic cornea is more prone to develop keratoepitheliopathy such as superficial punctate keratitis, recurrent corneal erosion, persistent epithelial defects and damage to corneal endothelium[34]. Cataract surgery can place undue stress on the cornea and lead to keratoepitheliopathy[13]. Motility of extra ocular muscles and ocular alignment should be evaluated. Diabetic patients should be evaluated

for cranial nerve palsies as diabetes is the most common cause of an acquired 6th and 3rd nerve palsy and second most common cause after trauma in acquired fourth nerve palsy.

Evaluation of pupillary responses should be done. RAPD should be looked for as diabetic papillopathy can occur in Type 1 diabetes in the 2nd or 3rd decades of life. As a part of The Blue Mountains Eye Study, it was indicated that there was a real association between a POAG and diabetes. Ocular hypertension was more in diabetic patients. Proliferative disease may present as rubeosis iridis in the anterior segment. Diabetes is the major cause of neovascular glaucoma. Iris is examined to detect any neovascularisation, presence of synechiae, signs of uveitis and pseudo exfoliation syndrome should be noted and treated accordingly. Pre-operative goniosocpy should rule out angle abnormalities PAS and neovascularisation.

Appearance of the lens in slit lamp clarity of the media in the visual axis should be evaluated to assess the lenticular contribution to the visual deficit. The integrity of the zonules and position of the lens must be evaluated.

Patency of the nasolacrimal duct is important in avoiding complications due to infection.

Pre-operative refraction is useful in planning the IOL power necessary to obtain the postoperative refraction desired by the patients.

A-Scan biometry is used to calculate the appropriate IOL power.

Fundus examination to detect the status of the retina and staging of diabetic retinopathy is a prime importance before planning for cataract extraction. Visual acuity testing, brightness acuity, contrast sensitivity and test for macular function are done.

Fundus examination if not possible due to an opaque media, B-Scan USG of the posterior pole of the eye is useful.

TIMING OF CATARACT SURGERY IN DIABETIC RETINOPATHY [50]

Visual symptoms due to cataract	Diabetic retinopathy status	Recommendation
None	None to moderate NPDR	No surgery
None	Severe NPDR or PDR	PRP
None	Diabetic maculopathy	Macular laser
Yes	None to moderate NPDR	Cataract surgery as soon as possible
Yes	Severe NPDR or PDR with good retinal view	PRP first then cataract surgery after 4 months

Yes	Severe NPDR or PDR with poor retinal view.	Cataract surgery as soon as possible followed by perioperative indirect PRP on table in PDR and within three months in severe NPDR.
Yes	Diabetic maculopathy with good retinal view	Macular laser first then cataract surgery after 6 months
Yes	Diabetic maculopathy with poor retinal view	Cataract surgery as soon as possible and treat pre-existing diabetic maculopathy with macular laser as soon as possible (usually can be done within 4 weeks) ; macular edema that develops postoperatively can often resolve spontaneously and can be observed for 6 months before treatment.

PATIENT PREPARATION :

Mydriasis : The use of phenylephrine 10% and cyclopentolate 2% produces effective mydriasis, multiple doses administered over one hour. Pre-operative NSAIDS flurbiprofen 0.03% or suprofen 1% mitigates any intra-operative pupillary constriction. Preservative free epinephrine 1:10,000 may be injected in to the AC at the start of surgery.

High molecular weight viscoelastic substance can increase mydriasis by applying direct mechanical pressure on pupillary margin during installation. Healon 5 produces stable mydriasis[2].

PREVENTION OF INFECTION.

- Incidence of infection rate in cataract surgery is 0.072%.
- Treatment of preexisting lid and lacrimal disease
- Use of plastic adhesive drapes
- 2 drops of povidone iodine 5% in the conjunctival sac for 1-2 minutes, decreases the incidence of endophthalmitis significantly.
- Lids and lashes prepared separately with cotton tipped swabs soaked in 5-10% providon iodine.
- Care should be taken to drape the lashes out of the operative field and to minimize the time the eye is open. The pre-operative use of antibiotics is controversial and the administration varies.
- Systemic infections should be identified and treated prior to elective surgery. Oral fluroquinolones are indicated in immunocompromised patients.

ANAESTHESIA IN DIABETIC PATIENTS

J.P. Barker, P.N. Robinsmith et al[35] studied 40 elderly patients undergoing cataract surgery. 10 NIDDM received GA, 10 NIDDM

received LA. 10 non-diabetic controls received GA and the other 10 non-diabetic controls received GA.

The results showed that in both general anaesthesia groups, NIDDM and control, blood glucose and serum cortical concentration increased significantly during surgery before return to normal by 4 hours after operation. In both local anaesthesia groups blood glucose and cortisol concentration change to return during surgery. Serum insulin concentrations increased 30 minutes after surgery to coincide with the peak of the glucose increased in non-diabetics who received GA, but no insulin response was seen in the diabetic GA patients. The results showed that cataract surgery under local anaesthesia provides improved metabolic control for diabetic patient. Its use maintains glucose homeostasis, prevents the increase in cortisol and glucose which are seen under GA and obviates the need for post operative starvation.

TYPE OF CATRACT SURGERY AND INTRA OPERATIVE PRECAUTIONS IN DIABETES

The usage of heparinized infusion (concentration 10 IU /ml) in the irrigation solution, as result of anti-inflammatory and antiproliferative effects of heparin, may be beneficial in diabetic patients; however no real long term benefit was proven and most surgeons do not use it routinely. This reduction in immediate and early

postoperative inflammation could benefit diabetic patients who are at risk for greater early postoperative inflammation. Phacoemulsification is associated with better postoperative visual acuity, less postoperative inflammation, and less need for capsulotomy than extracapsular cataract surgery in patients with diabetes, as shown previous studies. Surgically induced miosis was found to have occurred more often in the diabetics and cataracts in type 2 diabetics were found to be sticky and leathery; therefore, it is advisable that phacoemulsification in this group of patients is undertaken by an experienced surgeon. The surgeon's skills affect surgery time, decrease the chance of intraoperative complications, and this lessen postsurgery inflammation, perhaps reducing the risk of retinopathy progression.

Anterior capsular phimosis (or capsular contraction syndrome) is more common in diabetic patients[37]; therefore, the capsulorhexsis size should be larger than normal but smaller than the optic size of the IOL to prevent posterior capsular thickening. Other studies have shown that combined phacoemulsification, insertion of a posterior chamber IOL, posterior capsulectomy, and pars plana vitrectomy can be used to treat patients with complications resulting from proliferative diabetic retinopathy and macular edema. The combined suegery may prevent a

second operation for postvitrectomy cataract, allowing earlier visual rehabilitation. Minimum invasion and avoiding excess contact with iris decreases the chance of post-operative inflammation and bleeding.

IOL IN DIABETICS[50]

Large diameter IOLs are still required to facilitate the visualization and pan retinal photocoagulation treatment of the peripheral retina. A 6.5-mm IOL, for example, provides a 39.7% larger optic area than a 5.5-mm IOL and could be crucial to adequate diabetic retinopathy management. Posterior capsule opacification is linked to the shape of the optic, such that a square-edged design inhibits lens epithelial cell proliferation. The hydrophobic acrylic lenses with square edges demonstrated similar low incidence of posterior capsule opacification in comparison to blunt-edged lenses. A silicone IOL causes poor visualization during vitreo-retinal procedures and should be avoided in patients at high risk of developing advanced diabetic retinopathy. Hydrophilic acrylic lenses have the lowest rate of silicone oil adherence and would be the IOL of choice in these high-risk patients. However, it should be noted that there have been some reports of progressive dystrophic calcific opacification of hydrophilic acrylic IOLs in diabetic patients and longer follow-up of these patients is indicated by

surgeons. The use of multifocal and accommodative IOLs in diabetics is controversial.

SURGICAL RISK IN DIABETICS

Surgical correction of diabetic cataract is also more problematic in these patients than their nondiabetic counterparts. Intuitively, exposing a pathologically compromised eye to surgical trauma and the resultant inflammatory mediator induction and neutrophil and macrophage recruitment is of concern. Evidence implicates the blood ocular barriers as crucial factors in the progression of diabetic retinopathy. Abnormalities in blood-retinal barrier function have been demonstrated in diabetics without demonstrable clinical retinopathy. The degree of blood-retinal barrier dysfunction increases as the level of diabetic retinopathy deteriorates as does the degree of blood-aqueous barrier dysfunction.

Numerous studies have been published that have sought to answer whether there is true progression in diabetic retinopathy, including maculopathy following uncomplicated cataract surgery. The ETDRS report #25 looked at cataract surgery in diabetes. A trend toward post surgical progression did not, however reach a pre determined level of statistical significance ($p = 0.01$). Also cataract

surgery did not appear to yield any significant long term risk of macular oedema[18]. Uncomplicated phacoemulsification cataract surgery does not cause acceleration of diabetic retinopathy postoperatively. Progression that is observed probably represents the natural history of the disease rather than being a direct effect of surgery[61]. Clinically significant macular edema present in diabetic eyes at the time of cataract surgery is unlikely to resolve spontaneously, but clinically significant macular edema arising after surgery commonly resolves, particularly if retinopathy is mild as shown by natural history[24].

Compared to non-diabetic patients, outcome after cataract surgery was reported to be worse in diabetic patients--especially in those with diabetic retinopathy.

COMPLICATIONS OF CATARACT SURGERY

The possible complications that can arise in a diabetic during cataract surgery[2] are as follows :

1) Bleeding
2) Poor dilatation
3) Delayed epithelisation
4) Iritis
5) Possible effect on diabetic retinopathy

6) Insufficient wound closure
7) Infection
8) Increased blood sugar caused by topical / general steroid

Minimum invasion is essential to avoid complications of surgery and surgery is often complicated by poor dilatation. Avoiding excess contact with iris decreases post operative inflammation and unexpected bleeding in AC.

If vitrectomy was performed, surgeon should be prepared for unstable AC pre-existing rupture of PC.

If PRP planned, large anterior capsulotomy and optic diameter should be used.

I) Inadequate Pupillary Dilatation

This is a common occurrence in cataract surgery in diabetics. This can be managed by using

1) Retraction of the pupil by self retaining, titanium Iris hooks.
2) Iris protector ring
3) Sphincterotomies.

The use of capsular dye trypanblue or ICG is recommended for improved capsular visualization in any white cataract[51].

II) Shallow anterior chamber :

Intraoperatively it may be due to

- Inadequate infusion into the AC
- Leakage through oversized wound
- External pressure on the globe
- Positive vitreous pressure
- Suprachoroidal hemorrhage or effusion
- Posterior effusion syndrome - Common during hydro dissection; fluid infused into the AC may be misdirected into the vitreous cavity, causing an increase in the vitreous volume, with subsequent forward displacement of the lens and shallowing of the AC.

Post operative **:**

- Wound leak
- Choroidal detachment
- Pupillary block
- Ciliary block
- suprachoroidal hemorrhage

III. Corneal edema

Undue surgical stress on a compromised diabetic cornea can lead to corneal oedema commonly.

1. Stroma and epithelial edema (< 1% in general) :

Stromal or epithelial edema may occur in the immediate postoperative period preexisting corneal endothelial dysfunction causes an increased incidence of edema. Mechanical trauma, inflammation, increased IOP and prolonged irrigation are factors resulting in acute endothelial decompensation which causes corneal edema. Generally resolves in 4-6 weeks. Peripheral corneal edema resolves with times as a rule. Corneal edema after three months does not clear.

2. Vitreocorneal Adherence – Persistent Corneal Edema

Vitreocorneal adherence is an early / late complication. Advanced cases may need combined vitrectomy with a penetrating keratoplasty.

3. Descemet's Membrane Detachment :

This results in stromal swelling and epithelial bullae localized to the area of detachment. This complication can result due to trauma caused by an instrument or IOC introduced through the incision or when fluid is injected between Descemet's membrane and corneal stoma.

4. Toxic solutions :

Some solutions used for either irrigation or inadvertent entry into AC can be toxic to the corneal endothelium and cause temporary or permanent corneal edema. Chlorhexidine used to clean the skin can cause irreversible corneal damage.

5. Brown Mclean syndrome :

This is a clinical condition consisting of peripheral corneal edema with a clear central cornea. More frequent after ICCE. Edema starts inferiorly and progresses circumferentially but spares the central cornea. The etiology is unknown.

IV. Hemorrhage

1. Hyphema :

Postoperative hyphema originates from the incision or the iris. If vitreous is mixed with blood, it takes a longer time to resolve. Prolonged hyphema can lead to elevated IOP and corneal blood staining.

2. Retro bulbar Hemorrhage

More common with retro bulbar anesthetic injections. Venous RB hemorrhage is self limited and spreads slowly, often requiring no treatment. Arterial RB hemorrhage occurs rapidly and is associated with taut orbital swelling, proptosis and raised IOP, reduced mobility of the globe. It is diagnosed by the rapid onset of lid and conjunctival ecchymosis and tightening of the orbit.

3. Suprachoroidal hemorrhage or effusion :

It occurs generally intra operatively. This complications can be diagnosed by a forward prolapse of posterior ocular structures including the iris and vitreous, associated with a change in the red reflex. These complications are more common in the presence of hypertension, obesity, high myopia, glaucoma, anticoagulation or chronic ocular inflammation. Suprachoroidal effusion may be a precursor of suprachoroidal hemorrhage.

4. Expulsive suprachoroidal hemorrhage :

This is rare, but a grave complication presents as sudden increase in IOP accompanied by darkening of red reflex gaping of wound, iris prolapse expulsion of the lens, vitreous and bright red blood or sudden onset of pain. As soon as it is diagnosed, wound must be closed with suture or digital pressure. Posterior sclerotomy allows escape of blood. Delayed suprachoroidal hemorrhage may occur in early post operative period.

V. Posterior Capsular Rupture :

The first signs of capsular rupture may be a sudden deepening of the AC. In a small rent, residual cortex is removed followed by PCIOL implantation without disruption of the anterior hyaloids face. If the PC

rent is large and anterior hyaloid face is broken, a vitrectomy followed by implantation of IOL is recommended.

VI. Vitreous disruption :

Vitreous can enter the AC through the pupil as a complication of cataract extraction by any technique. The presence of vitreous may be detected by touching the wound or iris with a cellulose sponge. Vitreous in the AC may lead to chronic ocular inflammation with or without CME. Pupil may be distorted exposing the edge of the IOL with resultant glare. Vitreous in the wound can lead to ‘vitreous wick syndrome’ wherein endophthalmitis sets in due to entry of bacteria through the vitreous in the wound.

VII. Elevated IOP :

It is common following cataract surgery. The possible reasons are viscoelastic substances behind the lens implant or in the posterior chamber. Pupillary block, ciliary block, hyphaema, retained lens material, endophthalmitis, release of iris pigments, steroid use, PAS, preexisting glaucoma are reasons for raised IOP.

VIII. Iridodialysis :

Tearing of iris at its insertion occurs due to manipulation of intraocular tissues. If extensive, needs permanent monofilament suturing.

IX. Cyclodialysis :

Separation of the ciliary body from its insertion at the scleral spurs due to manipulation of tissues.

X. Endophthalmitis :

- Acute → 2-5 days post operatively, runs a fulminant course. Decreasing vision and increasing pain and inflammation are characteristic.
- Chronic → weeks to months after surgery. This is characterized by chronic iritis and granulomatous reaction
- Non infectious toxic endophthalmitis is a rare complication due to introduction of toxic materials into the eye
- It occurs in 0.07 to 0.22 %

XI. Chronic uveitis :

Patients develop chronic uveitis weeks or months after surgery. The response to steroids is variable. It is has been reported in association with low grade bacterial pathogens like propionobacterium acnes and staphylococcus epidermiditis.

XII. Retained lens material :

Patients with retained lens material present with variable degrees of inflammation depending on the size of the lens fragment; type of

material and time elasped since surgery. Clinical signs are uveitis, glaucoma, corneal edema and vitreous opacities.

XIII. Cystoid macular edema :

CME is a common cause of diminison of vision after both complicated and uncomplicated cataract surgery (Irvine Gass syndrome). Increased perifoveal capillary permeability associated with intraocular vascular unexplained reduction of visual acuity 6-10 weeks after surgery. Common following ICCE & PCR. Diabetes mellitus is a risk factor for CME. Other factors are post-op-inflammation, preexisting epiretinal membrane. Malpositioned implants, vitreous adhering to the incision, iritis or IOL. The risk of CME can be reduced by pre-and post operative use of topical 5% ketorolac.

XIV. Posterior Capsular Opacification (PCO)

In 1998 a meta-analysis showed PCO rates of 11.8%after one year 20.7% after 3 years and 28.4% after 5 years. Lens epithilial cells are a continuous single cell line. Functional and pathological groups – 'A' cells and 'E' cells. 'A'cells proliferates and forms fibrous tissue by undergoing fibrous metaplasia. 'E' cells are the primary source of pearl form of PCO. Bladder / Wedl cells are swollen opacified epithelial pearls.

Factors to reduce to PCO

1. Hydro-dissection enhanced cortical cleanup.
2. In the bag fixation.
3. Small CCC with edge on IOL.
4. Biocompatable IOL to reduce cellular proliferation.
5. Maximal IOL-PC contact, angulated haptic.
6. IOL optic geometry square, truncated edge.

Wound deshicence occurs at the rate of 0.02 – 1.5%. and it is more common in diabetics.

Cheng et al[17] reviewed the results of cataract surgery in diabetic patients and the visual outcome was good in the absence of retinopathy and was not significantly different from that of non-diabetic patients. Eyes with retinopathy achieved significantly worse visual results and the prognosis was related to the severity of retinopathy. Clinical cystoid macular edema occurred significantly more frequently in eyes with retinopathy than without and there were significantly more eyes with retinopathy which became blind or partially sighted. Modern cataract surgery seems to have no influence on the progression of diabetic retinopathy. A visual improvement is achieved in the majority of

patients with NPDR, but poorer visual outcome is observed in patients developing macular edema.

Pollack A et al[52] in their study showed the course of cystoid macular edema (CMO) following extracapsular cataract extraction with posterior chamber intraocular lens implantation was prospectively studied in 44 eyes of 44 consecutive diabetic patients without preoperative CMO. In 50% of eyes CMO was observed 6 weeks after surgery and in 25% was still present at 1 year. The preoperative presence of diabetic retinopathy significantly affected the postoperative onset and persistence of CMO. They believe that cataract extraction should not be recommended for eyes with pre-existing diabetic retinopathy until the vision has deteriorated to at least 6/30-6/60.

Gupta A et al[29] in their study showed patients with diabetic mellitus have an increased risk of developing cataract. Many such patients have pre-existing diabetic retinopathy at the time of cataract surgery Although more than 90% of the patients who have no pre-existing diabetic retinopathy carry a good visual prognosis and eventually have 20/40 or better visual acuity, nearly one-third of patients with pre-existing retinopathy may show retinopathy progression. Postoperative angiographic macular edema is more common in patients

with diabetes but resolves spontaneously in patients with no or minimal diabetic retinopathy. In patients with moderate to severe NPDR or more, clinically significant macular edema tends to persist, may arise de-novo, or even worsen after cataract surgery. Patients with diabetes need a preoperative characterization of their retinopathy and a thorough discussion with the patient about the need for cataract surgery, and the risk of progression of retinopathy is mandated. Currently, early surgery is favored before the development of significant diabetic retinopathy rather than wait for the cataract to become denser. All efforts should be made to stabilize diabetic retinopathy with appropriate laser treatment before cataract surgery. All diabetic patients need close observation for at least 6 months following surgery to intervene with laser photocoagulation as and when required to prevent visual loss from diabetic maculopathy and other consequences of diabetic retinopathy.

Menchini U et al [47,48] in their study showed diabetes is a risk factor for the development of cataracts. Studies have shown an increased risk of ocular complications in diabetics after cataract surgery, but modern surgical techniques have minimized them, leading to an overall good visual outcome. Macular edema before surgery is the most common condition that limits post-operative visual recovery. Thus, pre-operative laser treatment is needed. Photocoagulation of preproliferative

or early proliferative diabetic retinopathy is also advisable, due to the increased risk of iris neovascularization or retinopathy progression after surgery.

Kutschan A et al[44] investigated whether cataract surgery in patients with diabetes mellitus influences the progression of diabetic retinopathy, diabetic macular edema and anterior segment complications. Insufficient pupil dilatation (25.2 %) was the most frequent intraoperative complication. Among early postoperative complications anterior segment inflammation was most frequent (10.1 %). During the follow-up period the diabetic retinopathy in the operated eyes showed a progression which was statistically not different from that in the non-operated eyes. A diabetic macular edema was present after an average of 19 months in 6.8 % of the operated 119 eyes and after an average of 38 months in 6.7 % of the operated 88 eyes. The main late-complication during the whole follow-up period was the development of a capsular fibrosis in 52.3 % of the operated eyes. Postoperative visual acuity at the end of the first follow-up period (average 19 months) was >/= 0.5 in 85.7 % of the operated eyes and at the end of the second follow-up period (average 38 months) in 81.8 % of the operated eyes. Thus they concluded that extracapsular cataract surgery in patients with diabetes mellitus is a procedure with good results, of high reliability and

a slightly higher rate of complications than in non-diabetic patients. Extracapsular cataract surgery does not give rise to progression of diabetic retinopathy.

Straatsman BR et al[62] in their study with prospective evaluation of extracapsular cataract extraction with posterior chamber intraocular lens (ECCE-posterior chamber IOL) in 234 eyes of 20 diabetics, with and without nonproliferative retinopathy, and 209 nondiabetics demonstrated no statistically significant difference in operative or postoperative complications. A postoperative final visual acuity of 20/40 or better was achieved in 65% of diabetic surgical eyes and in 90% of nondiabetic surgical eyes, a difference that was statistically significant (P = 0.0049). When eyes with diabetic retinopathy or other pre-existing ophthalmic disease responsible for decreased vision were excluded, postoperative visual acuity of 20/40 or better was obtained in 93% of diabetic eyes and 96% of nondiabetic eyes, a difference that was not statistically significant (P = 0.5045). Therefore, the relatively less favorable outcome of ECCE-posterior chamber IOL surgery in diabetics, with or without nonproliferative retinopathy, is due to greater frequency of retinopathy or other ophthalmic disease responsible for decreased vision

Raskauskas PA et al[53] studied diabetic patients undergoing small incision cataract surgery . One hundred fifty-four eyes of diabetic patients were evaluated preoperatively through dilated fundus examination and Snellen visual acuity. The eyes then underwent small incision cataract surgery and were followed, undergoing periodic ophthalmoscopy, Snellen visual acuity measurement and additional postoperative therapeutic and surgical intervention. Final visual acuity improved by two Snellen lines or more in 61 out of 154 (40%) eyes. Final visual acuity worsened in 38 out of 154 (25%) eyes. They concluded the results are similar to those reported for extracapsular surgery and phacoemulsification, suggesting that the prognosis is guarded for diabetics, even when undergoing small incision cataract surgery.

Tsujikawa A et al[64] retrospectively studied 255 eyes of 190 diabetic patients who underwent cataract extraction with posterior chamber intraocular lens (IOL) implantation. One hundred forty-six eyes (57.3%) achieved the best visual acuity of 20/30 or better and 221 eyes (86.7%) achieved the acuity of 20/100 or better. The results suggested that the progression of diabetic retinopathy after IOL implantation was correlated with diabetic control at the time of surgery. Additionally, patients who developed the progression of the retinopathy in

pseudophakic eyes frequently showed the progression in the fellow unoperated eyes.

Tsujikawa A et al[65] in their retrospective study of 140 eyes of 102 patients, 97 eyes (69%) achieved a best visual acuity of 20/40 or better. After a minimum 6-month postoperative period, 26 eyes (19%) had developed retinopathy: eight eyes progressed from nonproliferative to proliferative retinopathy. Glycosylated hemoglobin levels and fasting blood glucose were significantly higher at time of surgery in the eight that progressed than in those who did not (P = 0.002, P = 0.034). There were 65 unilateral IOL implantations; in 10 (15%) of these eyes, retinopathy progressed. Retinopathy also progressed in 70% of the fellow eyes of these patients. In patients whose retinopathy did not progress, 95% of the fellow eyes also showed no progression. Also, patients with progression in the pseudophakic eye frequently had progression in the fellow unoperated eye. Postoperative progression was symmetrical (P = 0.0001). Their analysis suggests that progression of diabetic retinopathy following IOL implantation can be correlated to diabetic control at the time of surgery.

Chiselita D et al[19] in their retrospective study including 100 non-diabetic patients and 50 patients with type II diabetes, with extracapsular extraction and IOL implantation in the postoperated chamber followed

for 10.3-7.3 months postoperative complications. Their results showed that postoperative visual acuity was lower in diabetic patients (0.317 +/- 0.24) versus non-diabetic patients (0.634 +/- 0.30); postoperative visual acuity was lower in diabetic patients without retinopathy (0.437 +/- 0.26) versus diabetic patients with non-proliferative retinopathy (0.348 +/- 0.26) or diabetic proliferative retinopathy (0.116 +/- 0.11); the incidence of per- and postoperative complications (early and late) is higher in diabetic patients compared with the control group. Hence cataract surgery in diabetic patients is followed by higher rate of per- and postoperative complications. The postoperative functional result is dependent, in principal, on the retinal status of the diabetic patient and, secondary, on the per- and postoperative complications.

Gabric N et al[28] recommended cataract operation (ECCE + PCIOL) to be a well tolerated surgical procedure in diabetic patients with lenticular opacity, in whom it should be even earlier performed than in nondiabetics, because cataract prevents the diagnosis or treatment of a suspect retinal disorder.

Cataract is a common complication of diabetes indeed it has been estimated that up to fifteen per cent of cataract surgery is performed on diabetics. The main indications for surgery are the same as for non-diabetic patients. In addition, surgery is indicated if the lens

opacity prevents an adequate examination of the fundus or produces excessive scatter of light during laser therapy. Standard surgical techniques are applicable e.g. Extracapsular cataract extraction or phacoemulsification. with posterior chamber intraocular flexible or non-flexible lens implantation. If phacoemulsification is performed, it is advisable to perform a large capsulorrhexis with a 7mm optic lens, thus allowing the better visualization of the fundus for PRP if required. Following surgery, the incidence of capsular opacification is greater in diabetics than in non-diabetics. Therefore, a large YAG capsulotomy to improve vision or improve visualization of the retina may become necessary. The results of surgery best in those eyes with no retinopathy and worst in those eyes with active proliferative retinopathy.

Other rare complications of cataract surgery include

- Retinal detachment
- Macular infarction
- Pupillary capture
- Epithelial down growth
- Induced astigmatism
- Capsular opacification and contractions
- Dislocation or decentering of IOL
- UGH syndrome
- Induced astigmatism
- Retinal light toxicity

MATERIALS AND METHODS

This prospective study was carried out in the Department of Ophthalmology, Stanley Medical College & Hospital, Chennai, from October 2003 to December 2005. One hundred patients having diabetes who presented to our department for cataract surgery were included in the study.

Inclusion criteria

- Patients having biochemically proven diabetes mellitus
- Complete or partial Opacification of the lens or capsule with visual acuity of less than or equal to 6/24
- Good glycemic control pre operatively by either insulin or oral hypoglycemics or diet or in combination
- Patients willing to undergo cataract surgery
- Age greater than 40 years

Exclusion criteria:

- Diabetic patients with poorly controlled glycemic status
- Partial lens opacification with V/A better than 6/24
- Patients not willing to undergo cataract operation
- Age less than 40 years

Pre-operative evaluation :

All the patients included in the study were subjected to a detailed preoperative evaluation as follows :

Detailed history regarding duration of diabetes, type, nature of treatment, associated systemic conditions, pro-diabetic states were noted. A detailed ophthalmic evaluation was done by oblique examination, slit lamp examination and the fundus examination was carried out by using direct and indirect ophthalmoscopy and slit lamp biomicroscopy using + 78 D lens.

Ocular evalulation was done with special regard to changes in the ocular structures due to diabetes. Diabetics are more prone to develop infective conditions of the lids and adnexa, tear film abnormalities, keratopathy, keratoepitheliopathy, motility disorders (cranial nerve palsy). Pupillary responses were checked as RAPD could be due to diabetic papillopathy or other causes. IOP measurement, evaluation for POAG and neovascular glaumcoma were done as there is an association between POAG and diabetes. A routine gonioscopy was done in all patients to detect any neovascularisation or other abnormalities.

The morphology of the cataract was evaluated using a slit lamp. Fundus examination was done to assess the retina and diabetic

retinopathy status if present. In a dense cataract obscuring the media, B-scan, USG was done to assess the integrity of the retina.

Preoperative refraction is useful in planning the IOL power necessary to obtain the postoperative refraction desired by the patient. A-scan biometry was used to calculate the appropriate IOL power.

Intraoperative details including the type of surgery, IOL and the type of IOL, complications during surgery were noted. Postoperative details included the immediate post operative complications, V/A at 48 hours and the late post operative complications including progression of retinopathy and the final BCVA were noted. Patients with systemic illness were treated appropriately in the respective speciality departments prior to surgery.

All patients underwent cataract operation under local anaesthesia. All patients were admitted on the day before surgery and were discharged on the second day unless there was associated post operative complication deserving inpatient management. Preoperative fasting blood glucose was checked and those with poor glycemic control were referred to the diabetologist for appropriate treatment before embarking on cataract surgery. Patients with associated other ocular co morbidities were dealt with appropriately at the same time of cataract surgery e.g for glaucoma, pterygium. All the patients who were operated during the

specified time period irrespective of the status of surgeon were included in our study. Both the conventional types of cataract surgery i.e. ECCE and SICS were included in the study since phacoemulsification was not being done in our hospital for technical and financial reasons.

Combination of topical antibiotic steroids, cycloplegics were used routinely for 6 weeks during the post operative period. Tear substitutes, prednisolone acetate were used when necessary.

All the patients were subjected to a pre-operative visual acuity testing using a Snellen`s chart. The same was repeated in 48 hrs post-operative and refraction was done in the late post operative visit in 6 weeks time. The incidence of complications each in separate time interval from intra operative, early post operative and the late post operative were noted. The final outcome of surgery and the patient satisfaction were also noted.

OBSERVATIONS AND RESULTS

In this study the following are the observations and analysis made :

Age: the incidence of cataract with regard to age in the study conduced is stratified as follows.

Table 1
Age Incidence

Age	No. of patients	Percentage
40 – 50 years	18	18
51 – 60 Years	47	47
61 – 70 years	29	29
$\geq$ 71 years	6	6

47% of the patients were between 51 – 60 years and hence they constitute the majority in the study group. 29% were between 61-70 years followed by 18% 40-50 years.

Table 2
Sex Incidence

Sex	No. of patients	Percentage
Males	33	33
Females	67	67

Among the 100 cases who underwent cataract surgery, 33% were males and 67 % were females.

Table 3
Incidence of Cataract Morphology

Morphological characteristics of each cataract was studied by detailed slit lamp examination before surgery and it is as follows.

Morphology	Percentage	Subtypes
Cortical	80%	54% Immature 23% Mature 3% Hypermature
Nuclear	16%	
Posterior subcapsular	3%	1% Polychromatic Lustre 2% Bread Crump
Anterior subcapsular	1%	1% Snowflake cataract

The majority shows cortical changes (80%). Hypermature and mature constitute 26% which is because of the low socio economic status of the study group. Nuclear sclerosis was found in 16% and posterior subcapsular cataract in 3% of which 1% was poly chromatic luster and 2% had bread crump appearance. The typical snow flake diabetic cataract was found only in 1%.

Table 4
Systemic Association – Incidence

The incidence of other systemic associations in this study group of diabetics is as follows.

Systemic Association	No of patients	Percentage
Hypertension	16	16%
Ischemic heart disease	6	6%
Asthma	3	3%
Obesity	3	3%
Hypothyroid	1	1%

Hypertension (16%) was found to be the most commonly associated systemic condition in the study group followed by 6% ischemic heart disease. Asthma and obesity had an association of 3% each.

Table 5
Pre operative Glycemic control

Mode of treatment	No. of cases	Percentage
OHA & Diet	6	6%
Insulin	11	11%
OHA & Insulin	83	83%

The majority (83%) were treated by OHA and insulin because of the poor compliance of these patients. The rest 11% were treated with insulin. OHA and diet played only in minor role in this study group.

Type of diabetes

Among the 100 diabetic patients 97% were diagnosed type 2 DM, 2% as type 1 and 1% as MODY.

Table 6
Duration of diabetes

Duration	No. of patients	Percentage
≤ 2 years	25	25%
3-5 years	45	45%
6 – 10 years	28	28%
≥10 years	2	2%

45 % of the cases were diabetic for period of 3 – 5 years. 28% were known diabetics for 6-10 years. 25% of this diabetic population were detected ≤ 2 years. Only 2 % of the patients were diabetics for 10 years or more.

Table 7
Ocular Comorbidity

Coexisting ocular comorbidities in these diabetic patients who underwent cataract extraction are as follows.

Disease	No. of patients	Percentage
POAG	2	2%
Phacomorphic Glaucoma	2	2%
Uveitis	2	2%
ARMD	2	2%
Corneal opacity	1	1%
Iris coloboma	1	1%
Phacolytic Glaucoma	1	1%
Macular Hole	1	1%
HT Retinopathy	1	1%
Ocular allergy	1	1%

Primary open angle glaucoma was found in 2% of this study group. Other coexisting eye disease are purely co incidental.

Table 8
Intraoperative complications

Complication arising due to cataract surgery intra operatively are as follows:

Complication	No. of patients	Percentage
Intra operative Miosis	12	12%
Posterior Capsule Rent	6	6%
Zonulodialysis	2	2%
Vitreous loss	8	8%
Excessive pigment release	3	3%
Bleeding from iris	1	1%
SCH during anaesthesia	1	1%
Iris Tear	2	2%
Retained lens material	1	1%
Primary PCO	1	15
Calcified anterior capsule	1	1%

Intra operative miosis was the most commonly occuring intra operative complication (12%). Posterior capsule rent occurred in 6% of patients. Vitreous loss due to PCR and ZD was 8%. Bleeding was the next common complication, SCH in 1%, iris bleeding 1%. Excessive release of iris pigments was found in 3 %.

Table 9

Post operative complications – 48 hours

Complications	No. of patients	Percentage
Corneal edema	19	19%
Post operative inflammation (Iritis)	10	10%
Retained cortical material	3	3%
Raised IOP	4	4%
Iris Prolapse	2	2%
Hyphaema	1	1%
Wound leak	1	1%
IOL Malpositon	1	1%
Vitreous in wound	1	1%
Peri orbital bruise	1	1%
Reactivation choroiditis	1	1%
Supra choroidal haemorrhage	1	1%

Corneal edema in 19% was the most commonly occurring immediate post operative complication . Post operative iritis in 10% occurred in this study group. Raised IOP was found in 4% of patients.

Table 10
Long term complication

Complication	No. of patients	Percentage
Cystoid macular edema	4	4% - Aphakia 3% - Pseudophakia 1%
Recurrent uveitis	2	2%
Prolonged RIOP	2	2%
Cyst at wound site	1	1%
PCO	2	2%
Evisceration	1	15

Macular edema occurred in 4% and this was the common long term complication in the study out of which 3% were aphakic and 1% pseudophakic. Recurrent uveitis occurred in 2%. PCO incidence could not be calculated exactly because long term follow up was not possible.

Table 11
Visual acuity – 48 hours after surgery

Visual Acuity	No. of patients	Percentage
< 6/60	16	16%
6/60 – 6/36	29	29%
6/24 – 6/18	36	36%
6/12 – 6/6	18	18%
PL/PR negative	1	1%

Table 12
Best corrected visual acuity BCVA – 6 weeks

Visual Acuity	No. of patients	Percentage
< 6/60	10	10%
6/60 – 6/36	4	4%
6/24 – 6/18	17	17%
6/12 – 6/6	68	68%
PL/PR negative	1	1%

Nearly 68% of the cases in this study achieved a V/A of 6/12 or better. The post operative V/A in the rest 32% was less because of the complications and other eye diseases.

Table 13
BCVA with age in percentage

Age	< 6/60	6/60 – 6/18	6/12 – 6/6
40 – 50 years	1	1	16
51 – 60 years	6	6	32
61 – 70 years	4	10	17
≥ 71 years	-	4	3

ECCE was performed in 36% and SICS in 64% of the patients. Bilateral simultaneous cataract surgery was done in 16% of cases within a week.

Only in 2 patients (2%) progression of diabetic retinopathy was observed and progression was responsible for diminished BCVA in 1% only.

In 94% of the cases PCIOL implantation was done. In 5% patients were left aphakic and ACIOL was implantated in 1% of the patients.

DISCUSSION

The incidence of cataract with regard to age was evaluated and this study shows that early half (47%) of the patients are between the age 51 – 60 years. This correlated with the health and nutrition examination surgery (HANES) and the Framingham study that there is an increased risk of age related cataract development in diabetic less than 65 years old. In the Wisconsin study of diabetic retinopathy 31 of the younger onset patients and 76% of older onset patients not taking insulin had visual impairment due to causes other than retinopathy cataract being the commonest cause. Females constituted the majority 67% in the study. Gender standardized prevalence of diabetes in Chennai showed that, it is higher in females[14].

Nearly 80% showed cortical changes. A cataract having a characteristic age – related clinical appearance especially in terms of cortical and subcapsular changes, is more common adult diabetes, particularly women. Nuclear changes occurred in 16%, PSCC in 3%. ASCC in 1%. Central nuclear sclerosis and posterior subcapsular cataracts occur with equal frequency in diabetic and non-diabetics patients, anterior subcapsular cataracts seem to be more frequent in diabetics[7]. Among the PSCC, one was due co-existing chronic uveitis.

The classical ‘snowflake’ diabetic cataract with an appearance of white subcapsular spots forming dense band was found only in one patient, who was a forty year old female with MODY.

Hypertension 16% was the most common systemic association in this study group followed by ischemic heart disease 6%. Asthma and obesity had an association of 3% each.

Pre operation glycemic control was achieved by either OHA and diet, insulin or OHA and insulin. The vast majority were treated by OHA and insulin (85%) because of the poor compliance of these patients as the study group belonged to a low socio economic strata. Regular insulin was used when plasma glucose was more than 300 mg/dl and good glycemic control was achieved by titrating the insulin dosage according to the plasma glucose levels. Among the hundred diabetics, 97% were diagnosed as type 2 DM and 1% as type 1 and 1% as MODY. The prevalence of type – 2 DM in Chennai was 11.6%. the duration of diabetes is a crucial factor in the pathogenesis of diabetic eye disease 45% of the case were diabetics for 3-5 years, 25% were diabetics less than equal to 2 years and 28% were diabetics for 6-10 years.

Co-existing ocular co-morbidities were as follows. 2% were diagnosed to have POAG and a combined cataract extraction with trabeculectemy was performed. Most of the literatures are equivocal

about an association between diabetes and POAG. This was investigated as a part of the Blue Mountains Eye study and the results indicated that there was a real association between the two condition. 3 cases of lens induced glaucoma, out of which two were phacomorphic glaucoma and one was a phacolytic glaucoma were operated after control of IOP. 1% had chronic anterior uveitis which was operated under good steroid cover. Another 1% had a reactivation of choroiditis after cataract surgery. The other co-existing ocular conditions like angle closure glaucoma, macular hole, ARMD, HT retinopathy, corneal opacity and ptergium were purely co-incidental.

Local anesthesia was used in all patients who underwent cataract surgery. Cataract surgery under LA provides improved metabolic control for the diabetic patient. Its used maintains glucose homeostasis – prevents the increase in cortisol and glucose which are seen under GA and obviates the need for post-operative starvation[35].

Intra operative complication were noted in all the patient and intra operature miosis 12% is the most common complication arising this study group. Kutschan A et al found insufficient pupil dilation 25.2% was the most common intra operative complication in diabetics[44]. Zaczek A. et al[68] studied the pupil size in patients with diabetes mellitus during cataract surgery and concluded that constriction during surgery is

more pronounced in diabetic eyes as compared to controls. Due to ineffective mydriasis, the entire surgical procedure was pronounced as compared to non diabetics. Out of 12 patients with intra – operative miosis, four ended with complication like posterior capsule rent in 2%, Zonulodialysis in 1% and pigment release due to excessive manipulation while placing the PCIOL.

Subconjunctival haemorrhage with chemosis occurred in the lower quadrant during peribulbar anaesthesia in one patient. Posterior capsular rent occurred in 6%, zonulo dialysis 2% and hence vitreous loss occurred in 8% patients. This is higher compared to the incidence of the complication in non-diabetics when PCR occurred on 3.1% and vitreous loss in 0.8%. Excessive pigment release occurred in 3% and is a common occurrence in diabetics due to the nature of the iris in diabetes. Lamellar changes, cytoplasmic vacuolation, lipid droplet accumulation were noted in the iris mucles. Nerve fibres frequently diminished in diabetic patients[63].

A record of post operative complications in this study showed that corneal odema (19%) was found to be the most common post operative complication occurring in the first 48hours. Diabetic cornea has reduced sensitivity and undue stress can lead to keratoepitheliopathy[34]. The reduction of expression of entactin / laminin

– 1, and 10, and of their binding $\alpha_3\beta$ integrin in diabetic retinopathy cornea may severely impair adhesive and migratory properties of corneal epithelial cells. Such alteration in the corneal cell BM adhesion may be the mechanism underlying clinically observed abnormalities in epithelial barrier function, adhesion, epithelial integrity and wound healing[13]. Raised IOP also contributes to this increase in the number of post-operative corneal edema. Bleeding in AC had occurred in 1% post operatively and 1% intra operatively. Bleeding is a common complication arising in diabetics due to the nature of the diabetic tissue. The rate of post operative inflammation was 10% in this study, when compared to non-diabetics 1.8%. Retained lens material in 3% especially the superior cortical material was due to inadequate mydriasis. One of the patient, a fifty year old female developed expulsive supra choroidal haemorrhage on the second post operative day. She was diagnosed to have phacolytic glaucoma with hypertension. Evisceration was done to remove the prolapsed uveal tissue and hence prevent sympathetic ophthalmitis.

Menchini U et al[47,48] showed an increased risk of ocular complication in diabetes after cataract surgery but modern surgical techniques have minimized them, leading to overall good visual outcome. Kutschan A et al in study said, among early post operative

complications, anterior segment inflammation was most frequent (10.1%)[44].

In this study macular edema was the common long term post operative complication in 4% as compared to other 1.4%. recurrent uveitis occurred in 2% and one among them had an ACIOL implanted. 1% developed a dense exudative membrane which caused gross vision loss. PCO occurrence is 19% in general population after 1 year. Since patients could not be followed up for a long period, the exact incidence could not be detected.

Visual acuity outcome was studied and BCVA after 6 weeks of surgery was such that 68% achieved a V/A of 6/12 or better. The remaining 32% had BCVA less than 6/12 which was due to the complications of surgery and the co-existing ocular co-morbidities.

Macular edema was responsible for V/A < 6/60 in 4%. Corneal edema with RIOP in 3%, chronic corneal edema in 3% hyphema in 1% had BCVA of 5/60 and 1% had 6/24. recurrent uveitis was also responsible for diminished vision.

Kutschan A et al studied that diabetic macular edema was present after an average of 19 months in 6.8%. Cheng et al[17] found clinical cystoid macular oedema occured significantly more frequently in eyes with retinopathy than without. Pollack A et al[52] in the study showed that

in 50% of eyes CMO was observed 6 weeks after surgery and in 25% was still present at 1 year. The pre operative presence of diabetic retinipathy significantly affected the post operative onset and persistence of CMO. Gupta et al [29] studied that post operative angiographic macular edema is more common in patients with diabetes but resolved spontaneously in patients with no or minimal diabetic retinopathy.

ECCE was performed 36% and SICS 64%. Bilateral simultaneous cataract surgery was done in 16 cases within a gap of one week. Gabric N et al [28] recommended cataract operation (ECCE + PCIOL) to be a well tolerated surgical procedure in diabetic patients with lenticular opacity, in whom it should be even earlier performed than in nondiabetics, as it prevents the diagnosis or treatment of a suspect retinal disorder.

Raskauskas PA et al[53] studied diabetic patients undergoing SICS and concluded that the results are similar to those reported for extra capsular surgery and phacoemulsification suggested that prognosis is guarded for diabetics even when undergoing SICS.

Single piece biconvex mod.C step vault PCIOL with 6.50mm optic of PMMA material with UV absorbing optic was used in this study.

Tsujikawa A et al[64] suggested that the 57.3% achieved the BCVA of 20/30 or better and 86.7% achieved the acuity of 20/100 or better. Straatsman BR et al[62] demonstrated of final visual acuity of 20/40 or better was achieved in 65% of diabetic surgical eyes and in 90% non diabetic surgical eyes, a difference that was statistically significant (P=0.0049).

Retinopathy progression on occurred only in 2%. Gupta et al[29] suggested that patients with diabetes need pre operated characterization of the retinopathy and discussion with the patients about the need of cataract surgery and risk of progression of retinopathy is mandated. Early surgery is favoured before the development of significant diabetic retinopathy.

Stratsman BR et al[62] said when eyes with diabetic retinopathy or other pre-existing ophthalmic disease responsible for decreased vision were excluded, post operative visual acuity of 20/40 or better was obtained in 93% of diabetic eyes and 96% of non-diabetic eyes.

SUMMARY AND CONCLUSION

Hundred diabetic patients who underwent cataract surgery were evaluated. Diabetes leads to an increase in age related cataract development and more so in females. The typical snowflake diabetic cataract has become a rare entity and the cortical variety is the commonest. Hypertension and IHD were found to be more associated with diabetes.

A thorough ocular evaluation before cataract surgery is mandatory. Pre operative preparation especially mydriasis is a crucial factor in determining the outcome of surgery. LA with peribulbar / retro bulbar is the anaesthesia of choice in diabetic patients for cataract surgery.

In the study, intra operative miosis 12% was most common intra operative complication followed by posterior capsular rent 6% and vitreous loss in 8%. This is more compared to non diabetics. Bleeding in one of the complications occurring more in diabetics. Post operatively corneal edema (19%) was more common than others. Post operative iritis occurred in 10%.

Macular edema was a most common long term complication(4%) and recurrent uveitis in (2%) retinopathy progression was found only 2% of the patients. BCVA of 6/12 or better was achieved in 68% of the

cases. 17% had BCVA of 6/24 – 6/18. 15% had BCVA < 6/36. BCVA of < 6/12 was due to the complications of surgery due to diabetes and other co-existing ocular co morbidity.

Best result of cataract surgery in diabetics was achieved by adapting the following measures.

Pre operative

- Decision regarding the timing the cataract surgery in patients with diabetic retinopathy.
- Meticulous patient work up regarding other systemic complications and glycemic control.
- A detailed ocular evaluation with special regard to lid and adnexal infections, tear film abnormalities, keratoepitheliopathy, endothelial integrity, motility disorders, optic nerve assessment, retinopathy status and other ocular diseases. Routine gonioscopy and evaluation of the morphology of the lens was done.
- Care was taken regarding adequate pre operative mydriasis and maintain with NSAIDS .

Intro operative

- Local anaesthesia is the anaesthesia of choice in diabetics.
- Patients were draped with lashes out of the operative field.

- Intra cameral preservative free 1:10000 epinephrine at the start of surgery was used. High molecular weight visco elastic substance (Healon) was used only in a very few patients due to financial constraints. Heparanised infusion concentration 10 IU/ml was used in the irrigation solution and was found to be beneficial.
- SICS was associated with better post operative visual acuity and less post-operative inflammation. Minimal invasion and surgical skill is an essential. A large anterior capsulotomy with 6.5mm IOL was used. Silicon IOL should be avoided in patients with advanced diabetic retinopathy.
- Post operative topical antibiotic steroid was used for a period of 6 weeks and long term follow up was done regarding retinopathy status and complications of surgery

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PROFORMA

Name : Age/sex: IP No :

Occupation : OP No :

Address :

DIAGNOSIS : DOA :

DOS :

DOD :

HISTORY :

Duration of D.M.

Type

Mode of treatment

Associated systemic illness :HT/IHD/Hypercholestrlemia

Asthma,Steroid intake,prior surgery,

Other history

GENERAL EXAMINATION & INVESTIGATIONS:

CVS : FBS :

RS : PPBS :

CNS : ECG :

PR : BP : Glycosalated Hb :

OCULAR EXAMINATION : Oblique and Slit lamp

OD OS

- Face
- Lids/Brow
- Conjunctiva
- Cornea
- AC
- Pupil
- Iris
- Lens
- V/A
- RR
- IOT
- Duct
- Fundus

Pre-op control of Blood sugar:

1. OHA&Diet
2. Insulin
3. OHA&Insulin

PCIOL: ACIOL:

Surgery: ECCE/SICS/otherprocedures

PER-OP COMPLICATIONS

- Anesthetic
- AC Haeomorrhage
- AC Collapse
- IRIS-tear/dialysis/prolapse
- Choroidal Haemorrhage
- PC rupture
- Vitreous Disturbance
- Descemet`s stripping
- Retained Cortex
- Loss of nuclear fragments into vitreous
- Abnormality in wound closure

POST-OP COMPLICATONS

Corneal odema
Raised IOP
Periocularbruising/ odema
External eye infection
Wound leak
Vitreous in the wound
Hyphaema
Uveitis
Iris abnormality
Retained cortex
Pupil block
IOL dislocation
Endophthalmitis

LONG TERM COMPLICATIONS:

- CMO
- Uveitis-Refractory
- Retinopathy Status

POST-OP DIABETES MANAGEMENT:

- Fundus-
- Retinoscopy-
- Best corrected V/A-

Note/Other details-

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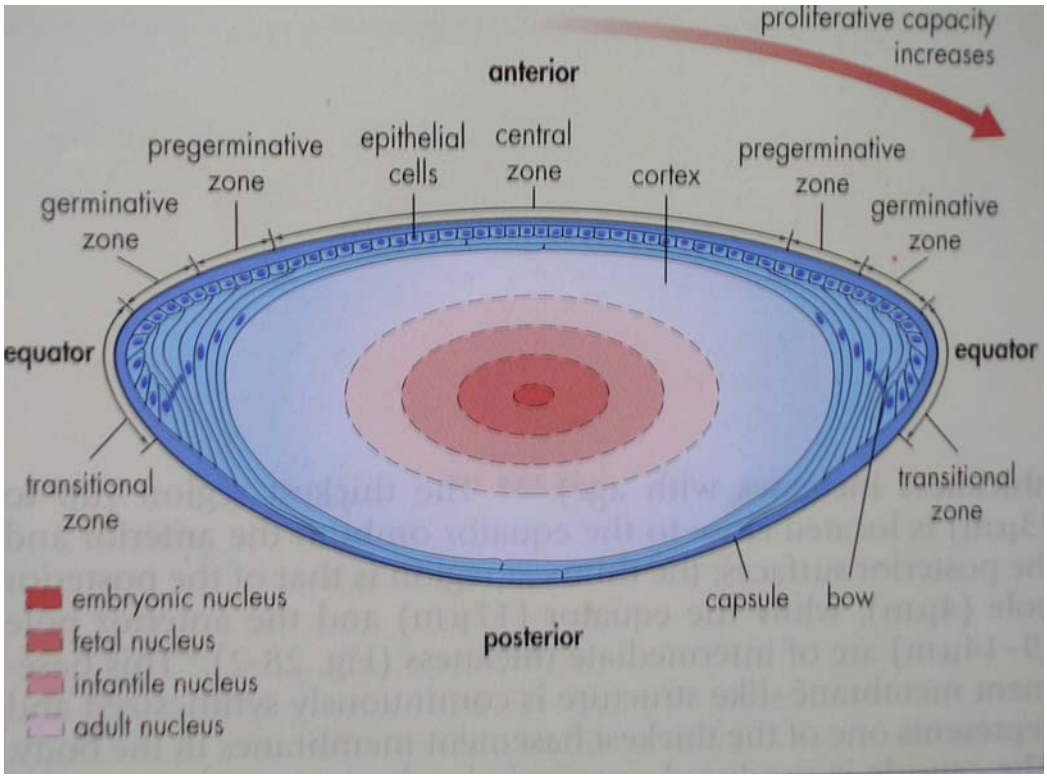
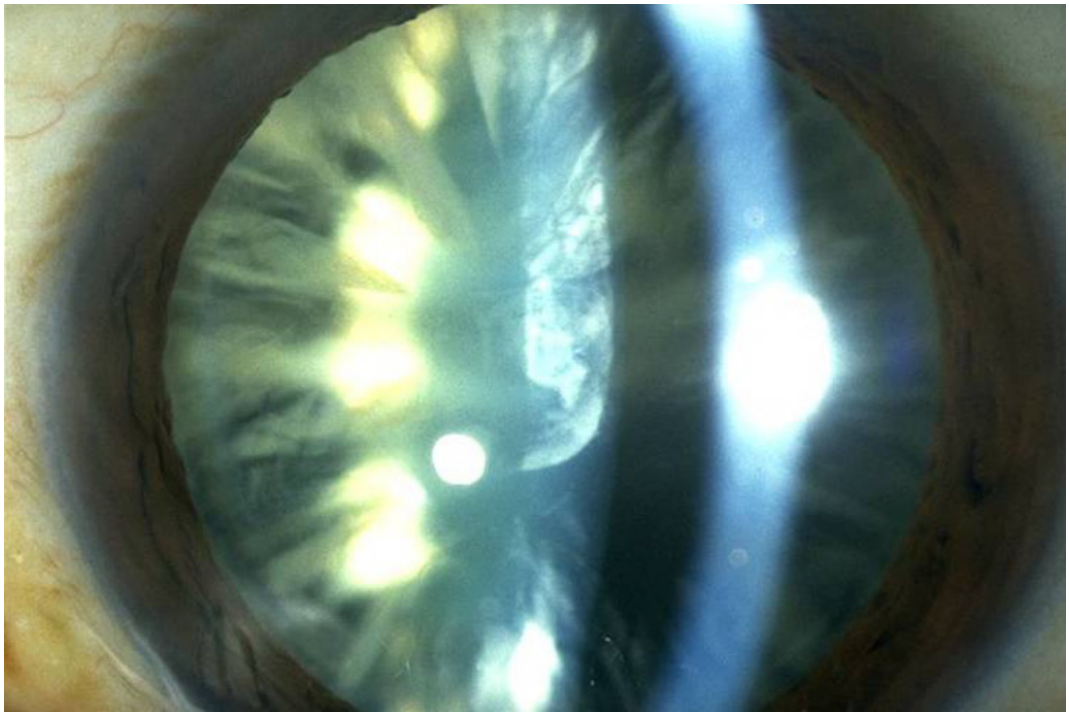
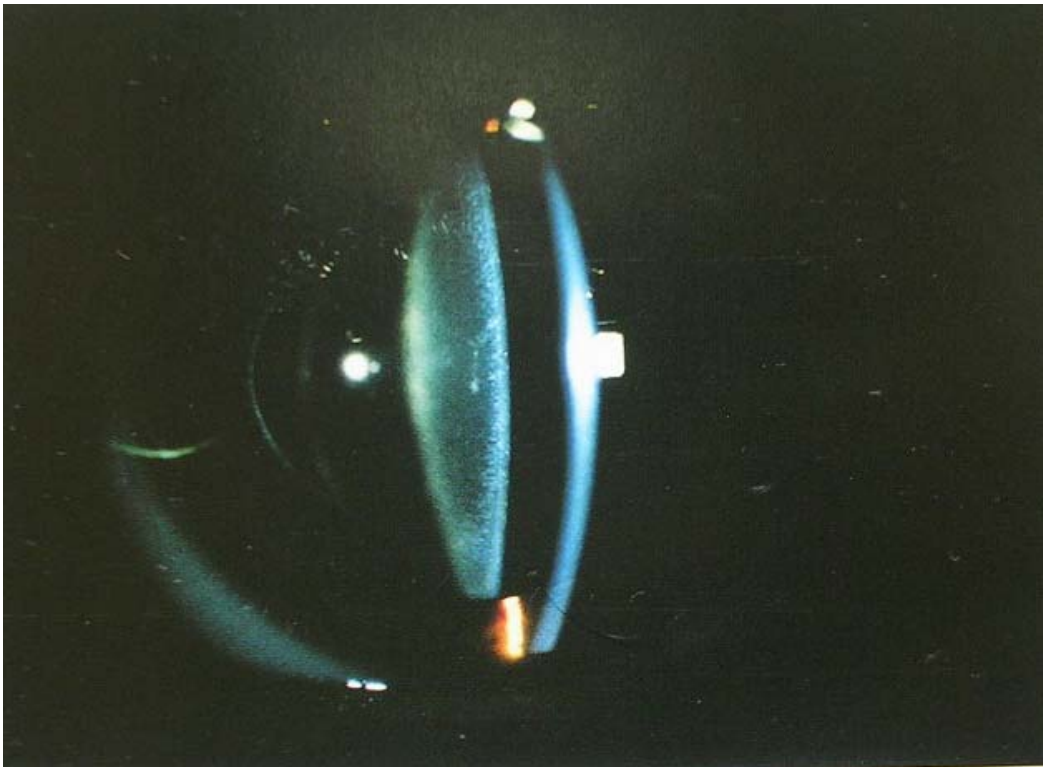


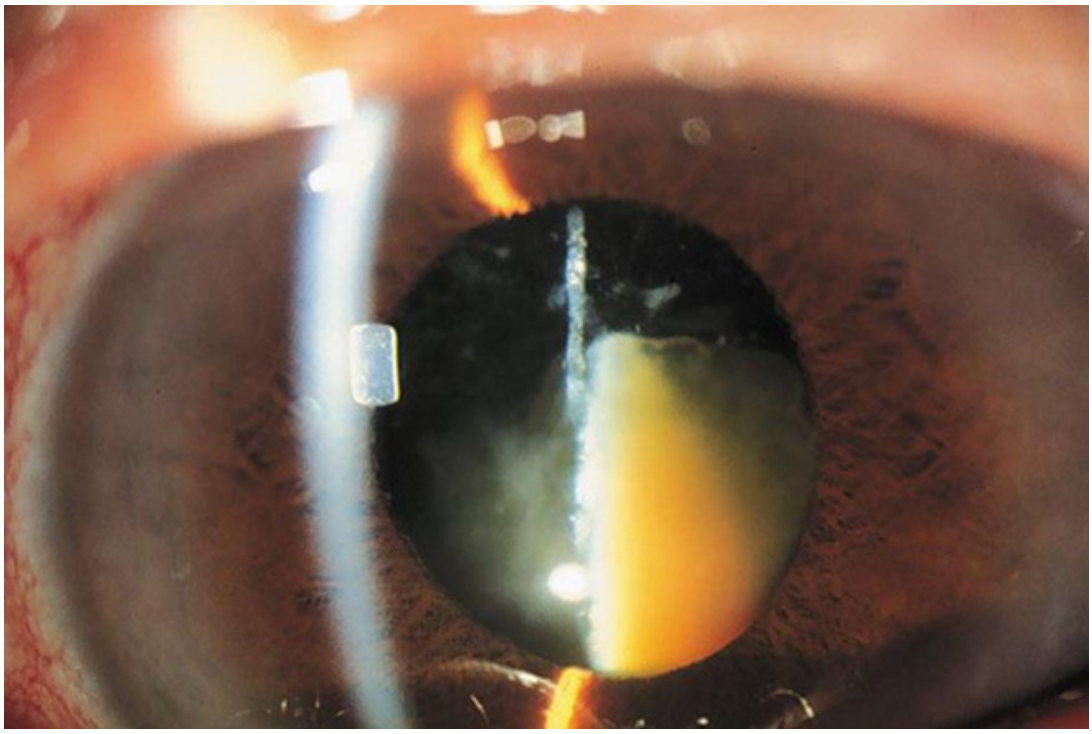
WEDGE SHAPED OPACITIES AND VACUOLES IN CORTICAL CATARACT



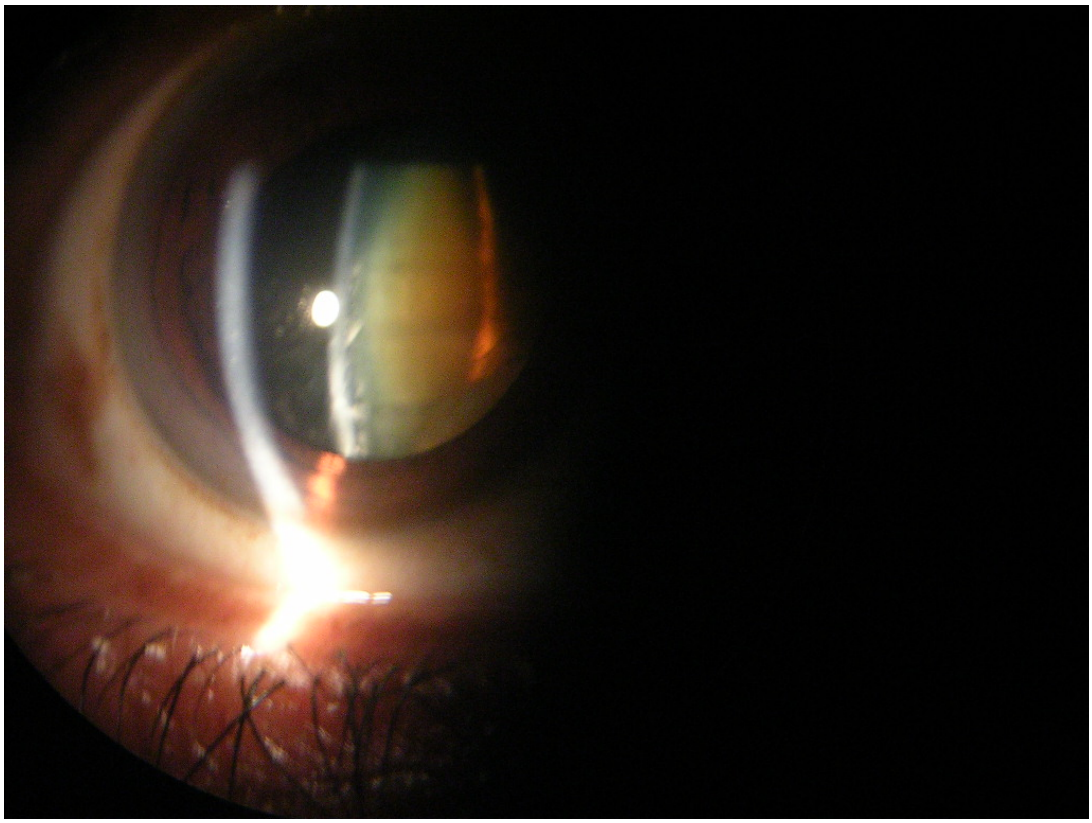
SNOW FLAKE CATARACT



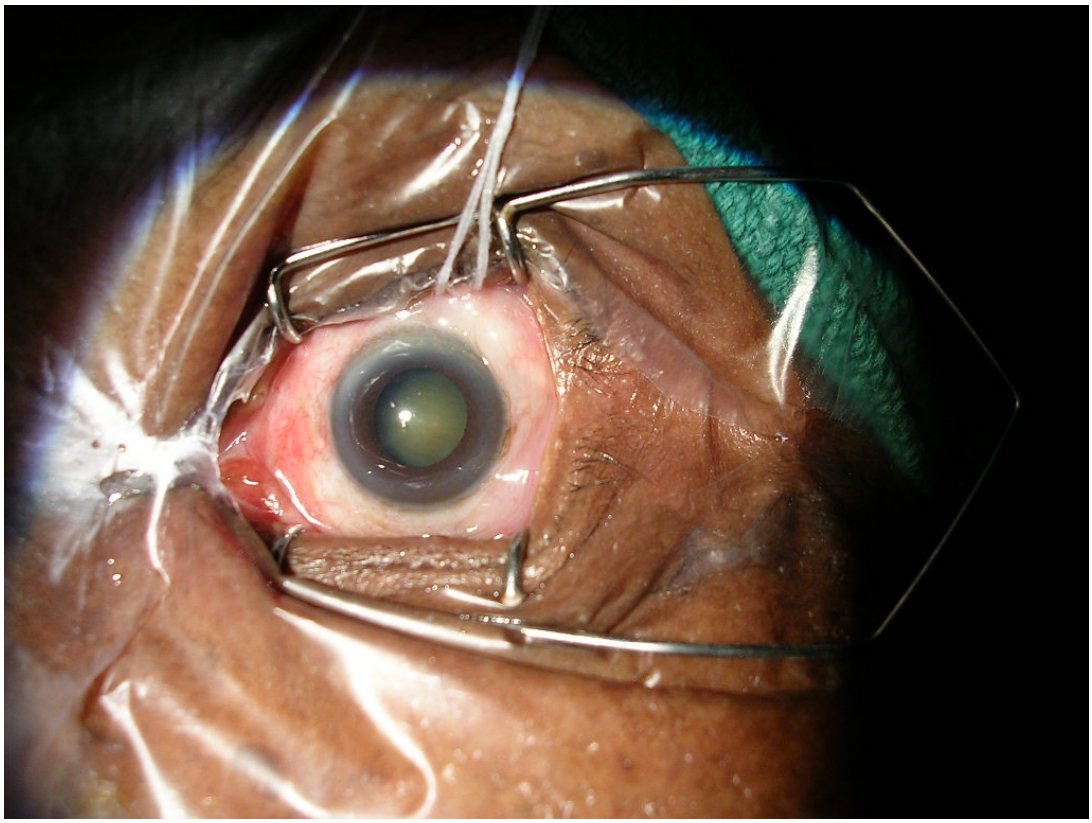
MORGAGNIAN CATARACT



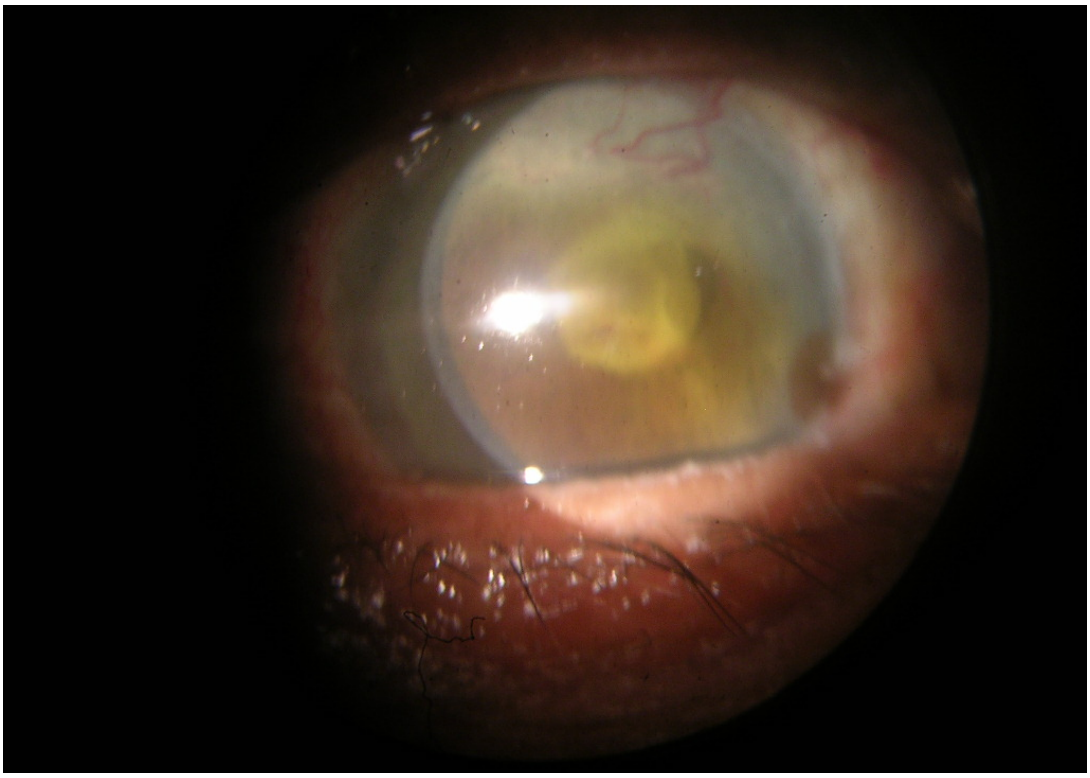
ANTERIOR AND POSTERIOR SUBCAPSULAR OPACIFICATION



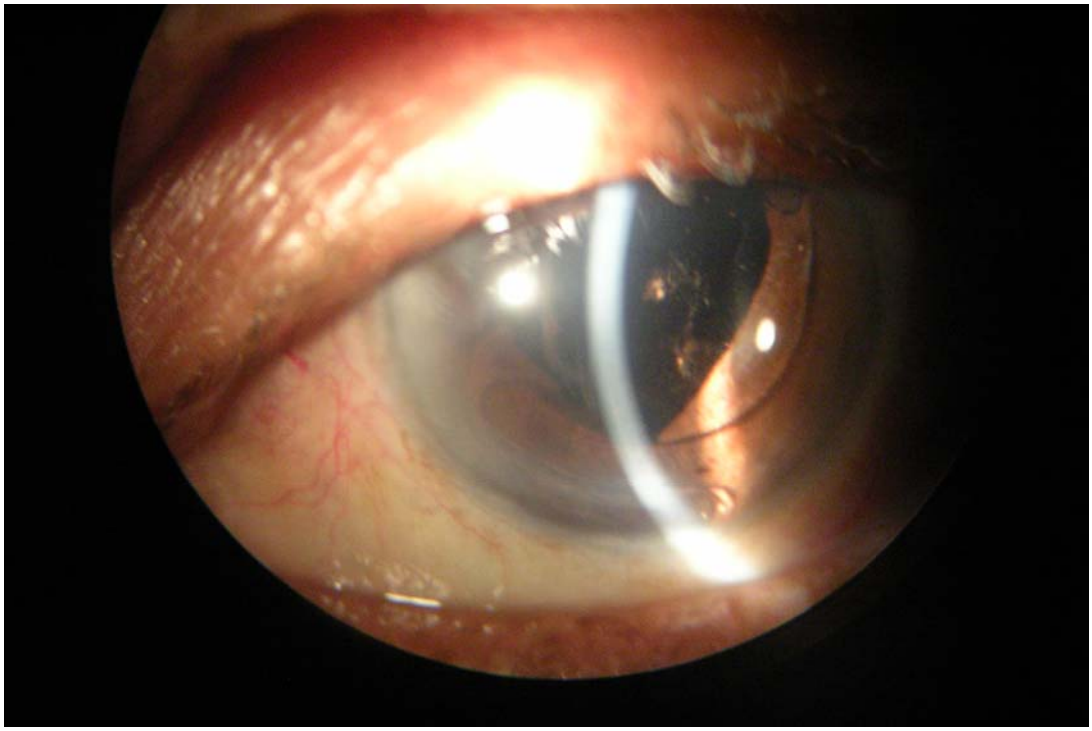
PREOPERATIVE PREPARATION



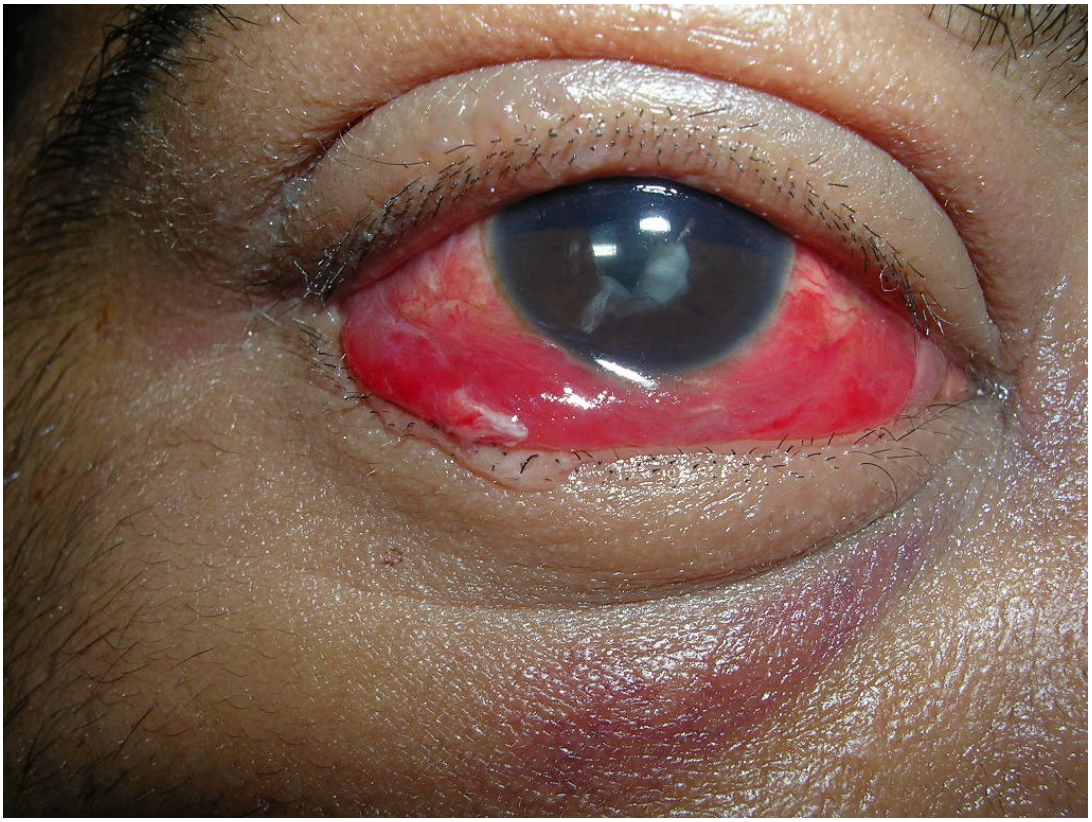
EXUDATIVE MEMBRANE IN A CASE OF RECURRENT UVEITIS



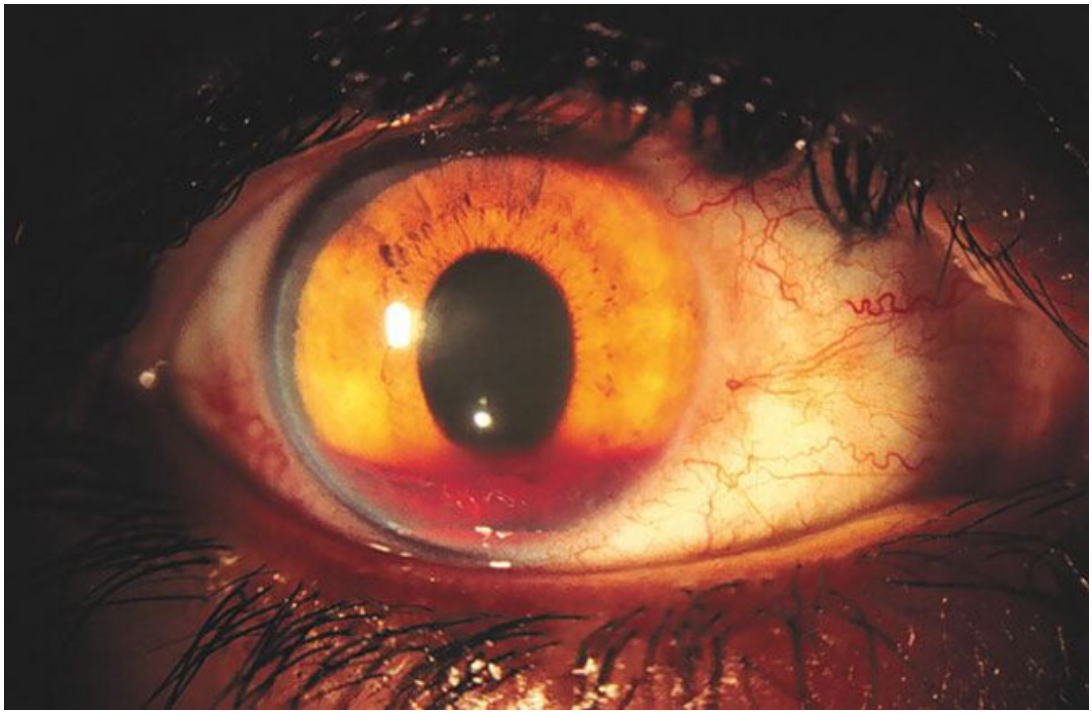
RECURRENT UVEITIS WITH ACIOL IMPLANTATION



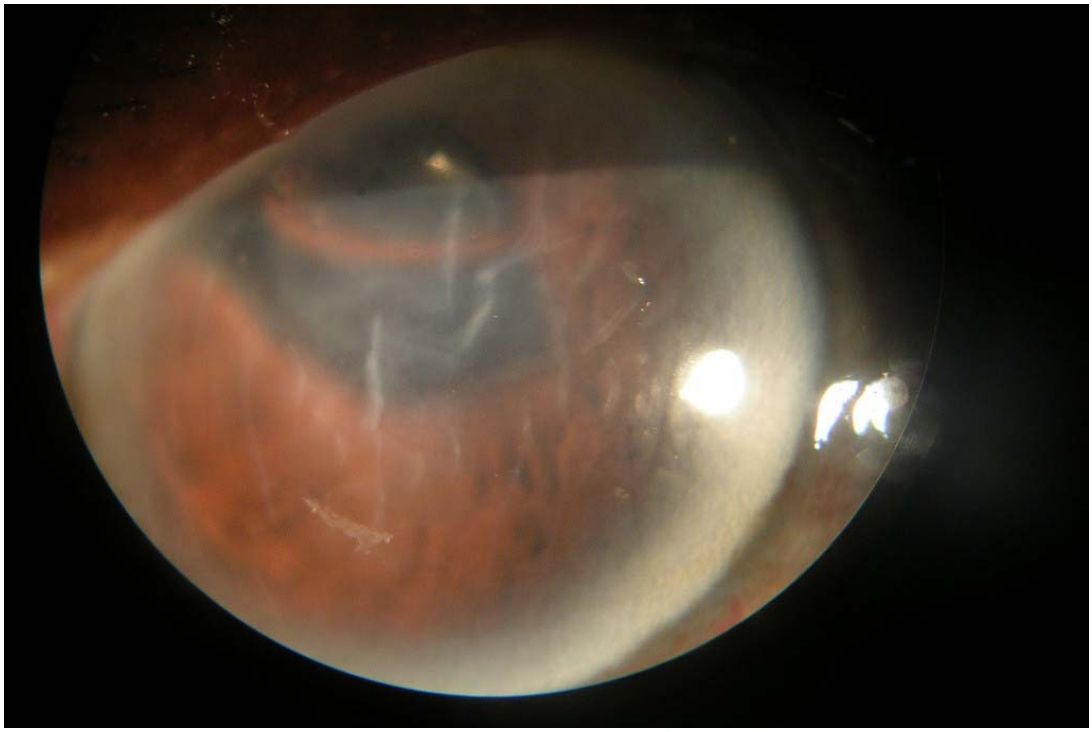
SCH AND CHEMOSIS THAT OCCURED DURING ANAESTHESIA



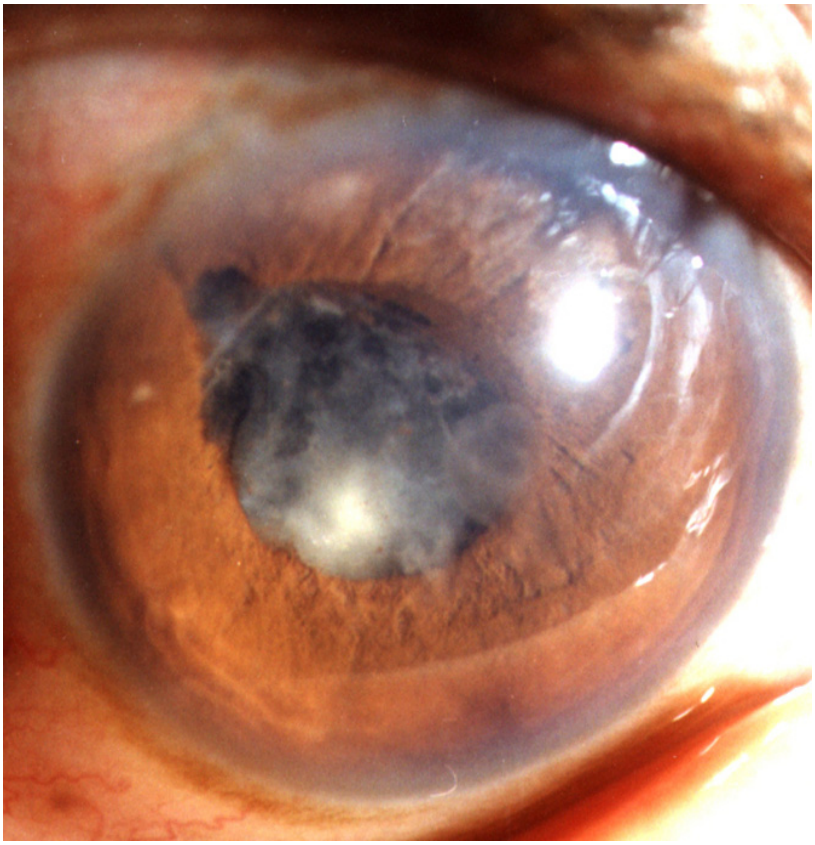
POSTOPERATIVE HYPHAEMA



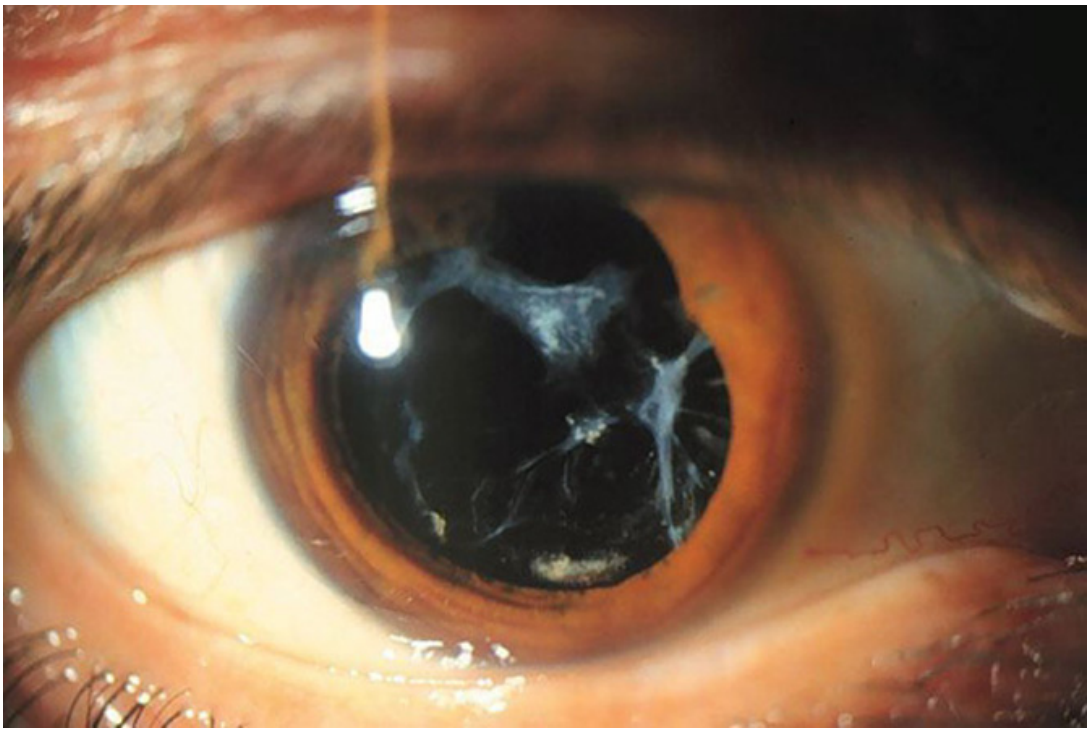
CORNEAL EDEMA WITH DESCEMETS FOLDS



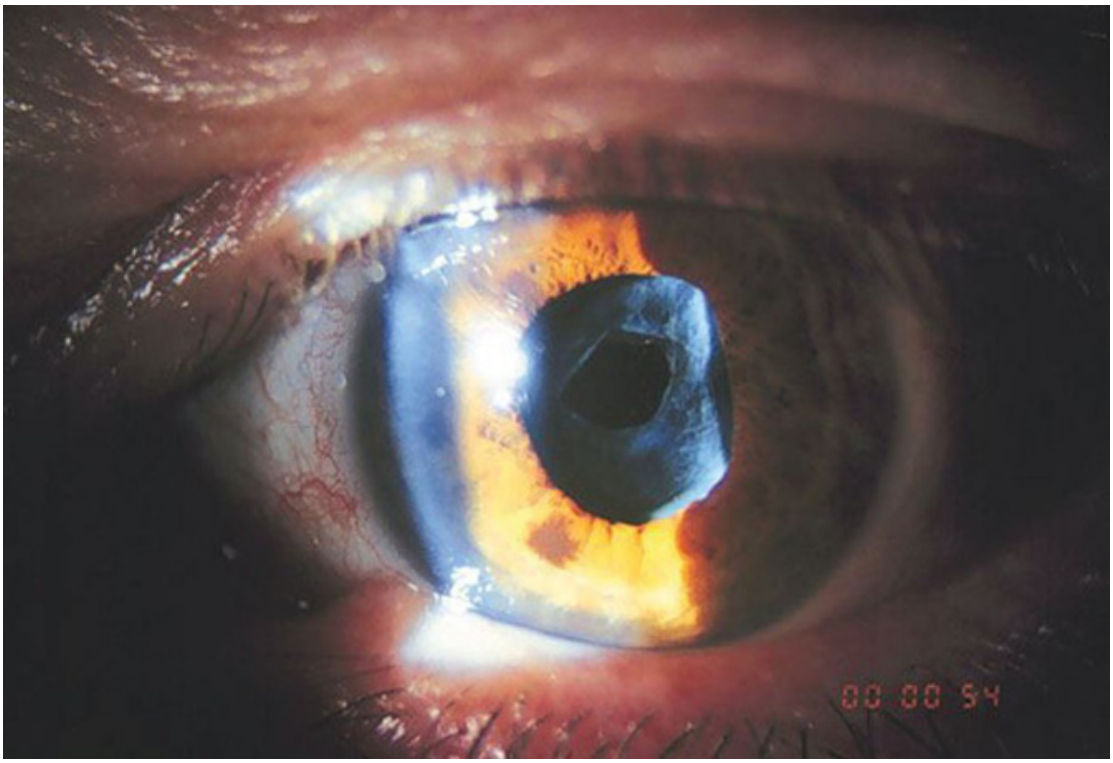
PUPILLARY BLOCK WITH CORNEAL EDEMA



RETAINED CORTICAL MATERIAL



YAG CAPSULOTOMY DONE IN A PATIENT WITH PCO



40-50 years	51-60 years	61-70 years	≥ 71 years
18%	47%	29%	6%

Male	Female
33%	67%

Immature C	Mature Cor	Hypermatu	Nuclear	Polychroma	Bread Crur	Snowflake
54%	23%	3%	16%	1%	2%	1%

Intraopeati	PCR	Zonulodialy	Vitreous Lo	Pigment re	Iris Bleed	SCH	Iris Tear
12%	6%	2%	8%	3%	1%	1%	2%

Corneal Ed	Iritis	RLM	RIOP	Iris Prolaps	Hyphaema	Wound lea	IOL Malpos
19%	10%	3%	4%	2%	1%	1%	1%

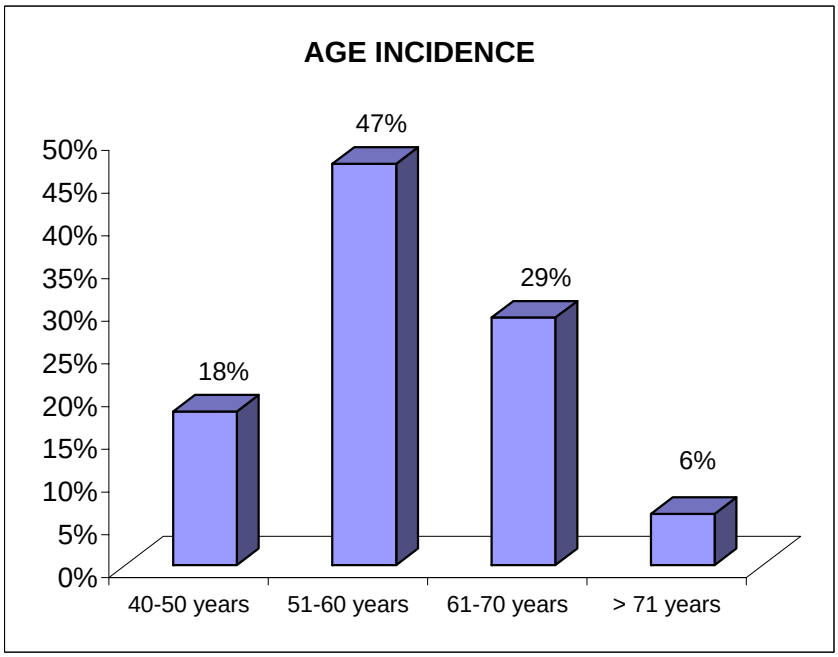
CMO	Recurrent I	Prolonged	Cyst	PCO	Evisceration
4%	2%	2%	1%	2%	1%

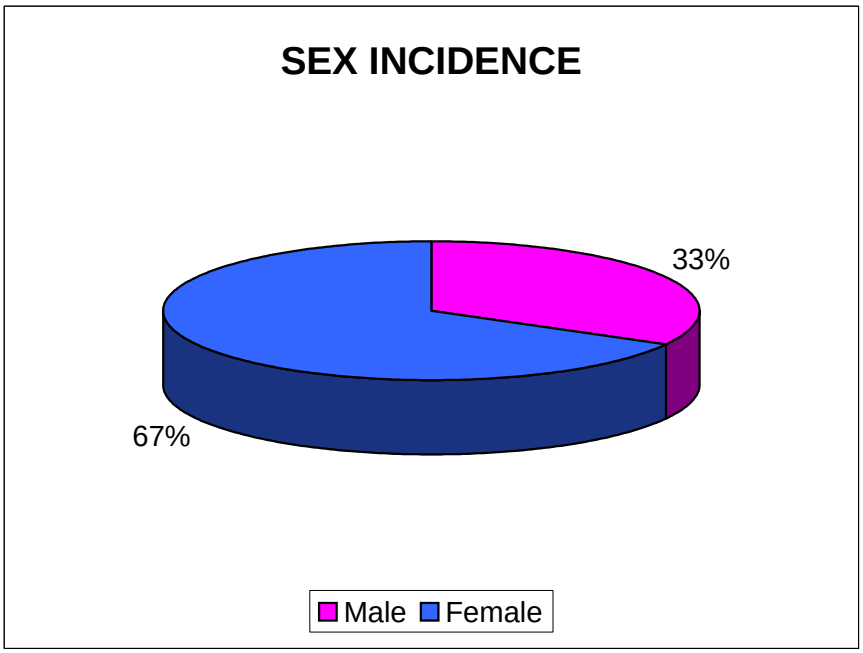
<6/60	6/60 - 6/36	6/24 - 6/18	6/12 - 6/6	No PL / PR
10%	4%	17%	68%	1%

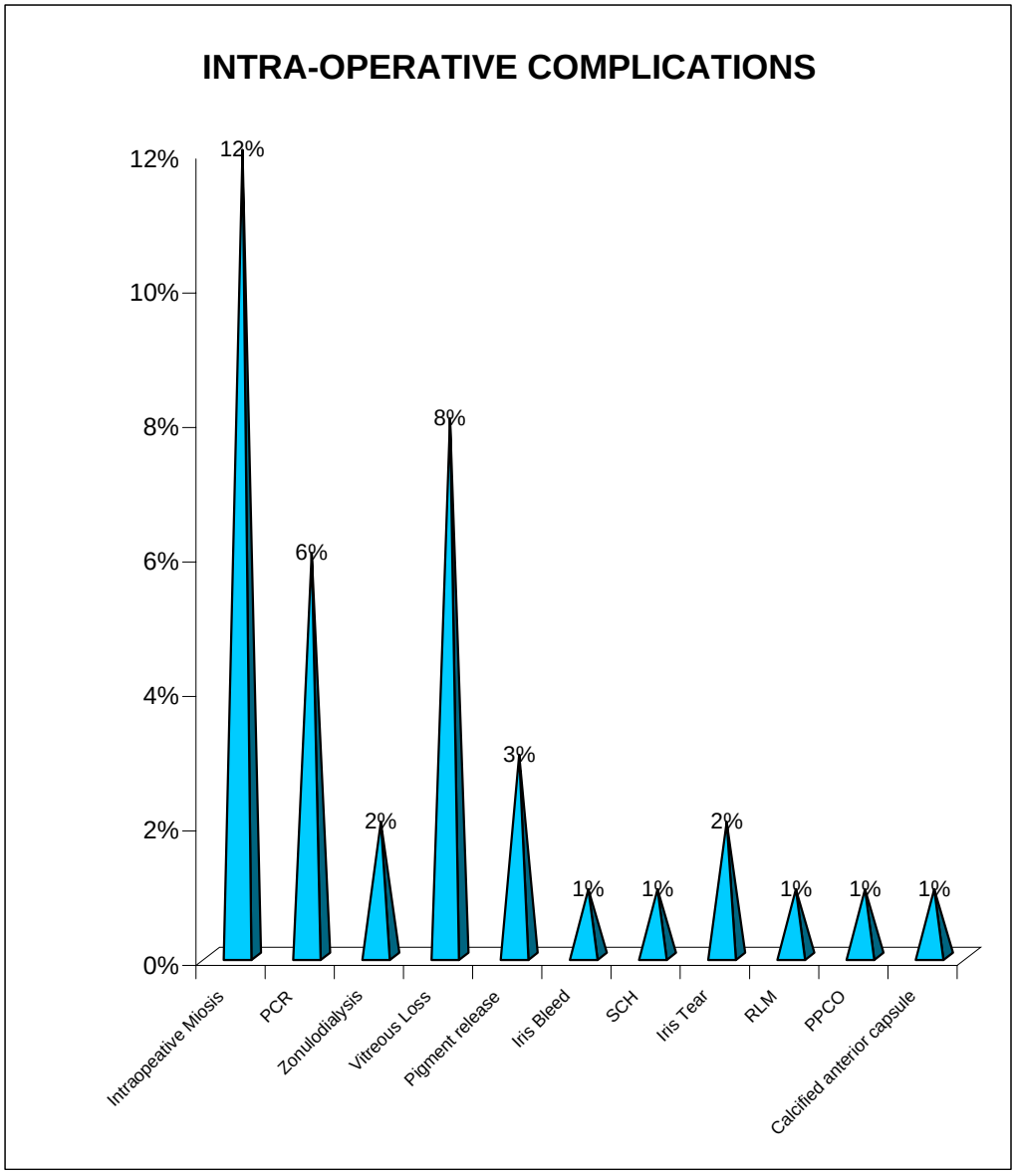
	<6/60	6/60 - 6/18	6/12 - 6/6
40-50 years	1%	1%	16%
51-60 years	4%	6%	32%
61-70 years	4%	11%	17%
≥ 71 years	0%	4%	3%

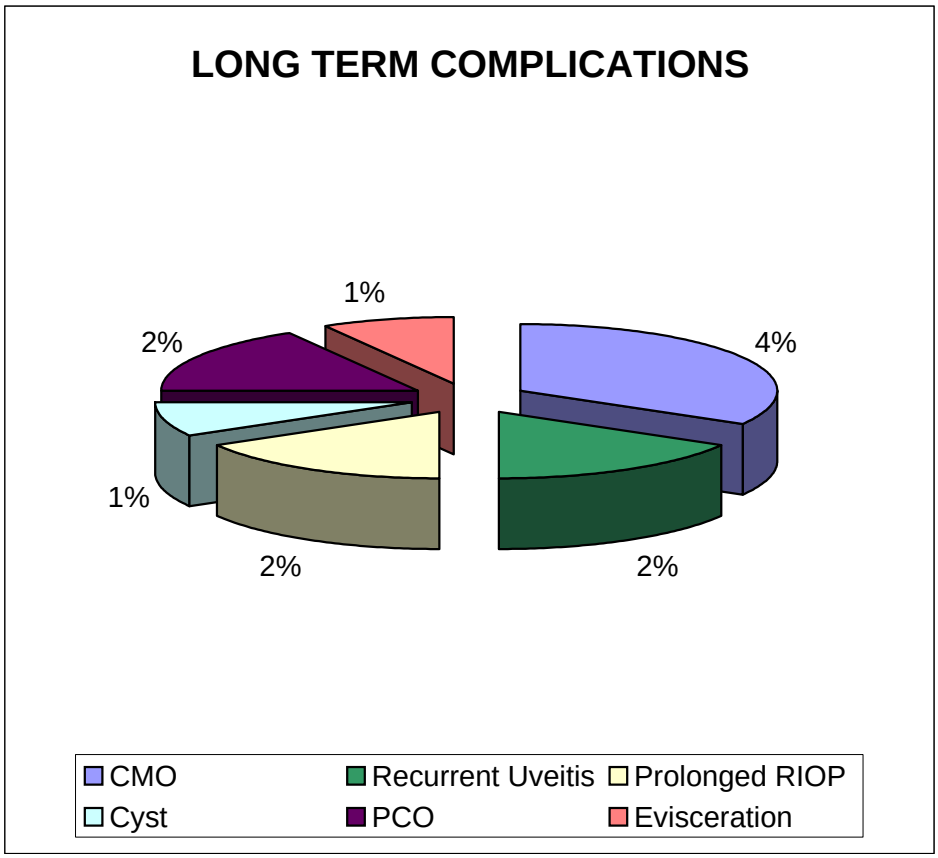
RLM	PPCO	Calcified anterior capsule
1%	1%	1%

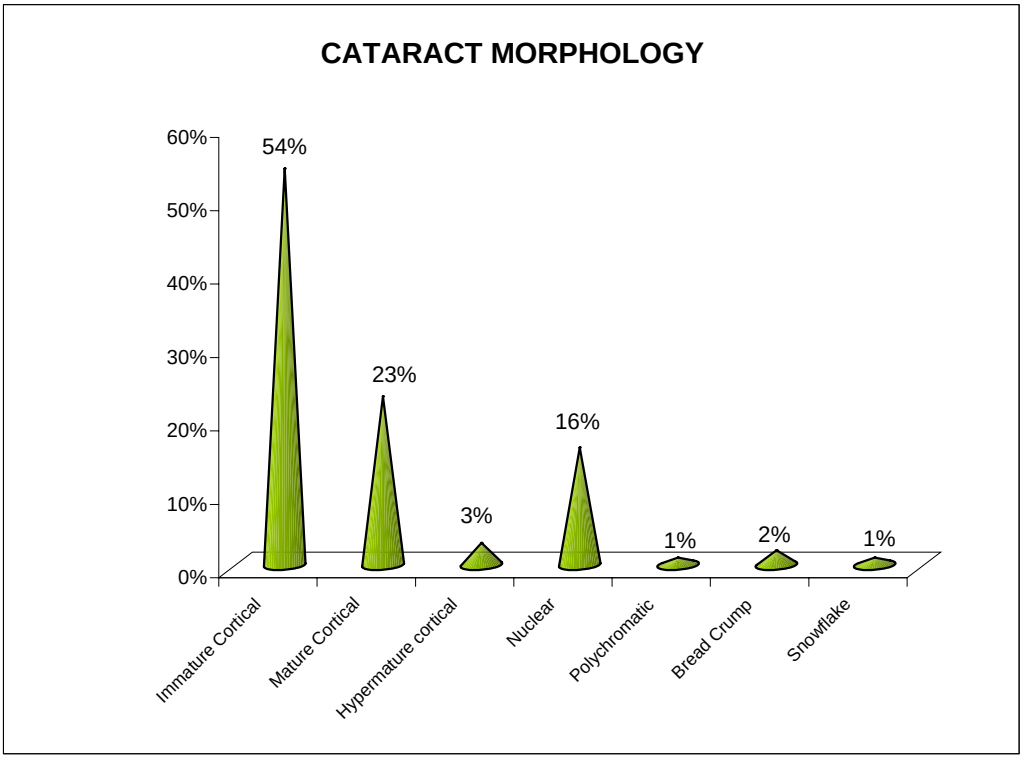
Vitreous in	Periorbital	Reactivatio	Suprachoroidal haemorrhage
1%	1%	1%	1%

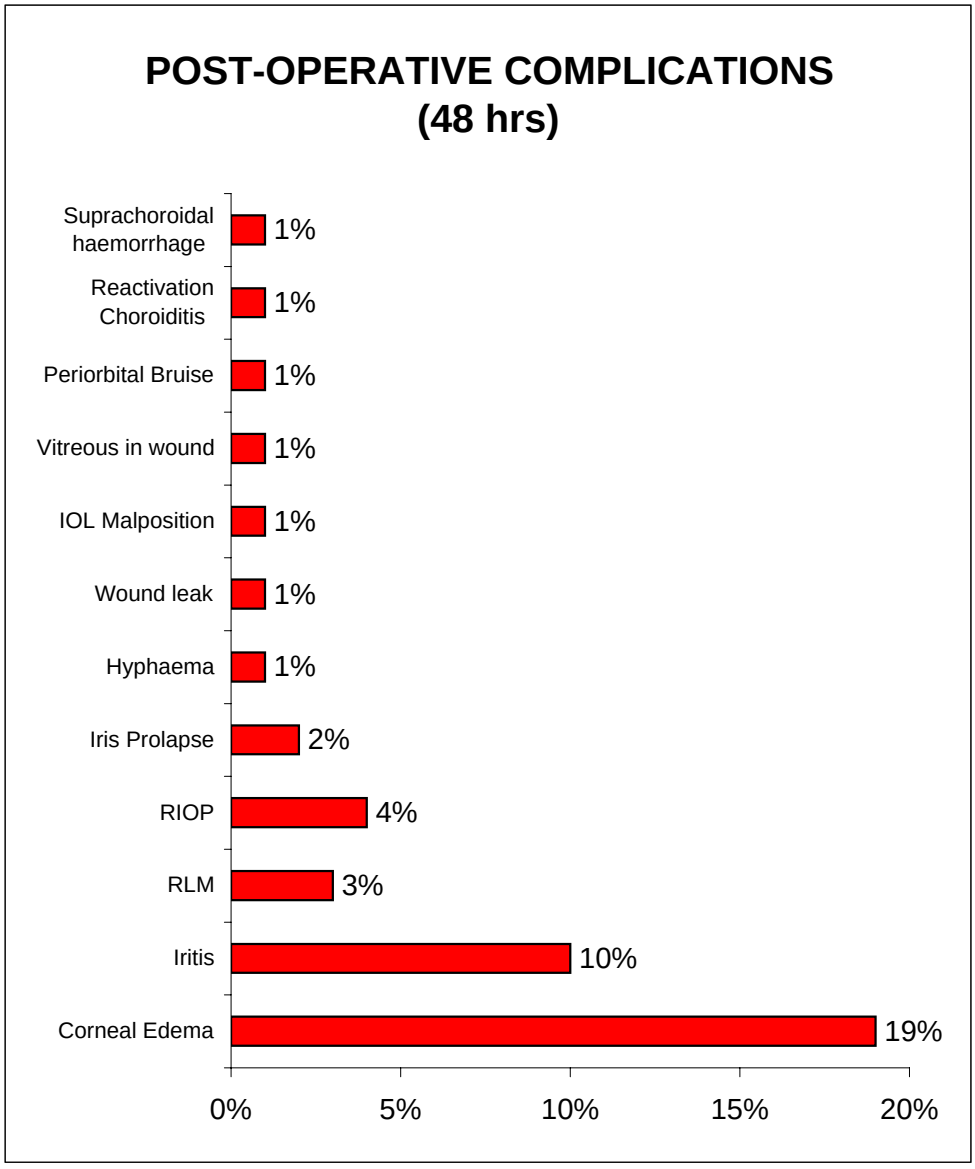

AGE INCIDENCE
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
18%
47%
29%
6%
40-50 years
51-60 years
61-70 years
> 71 years

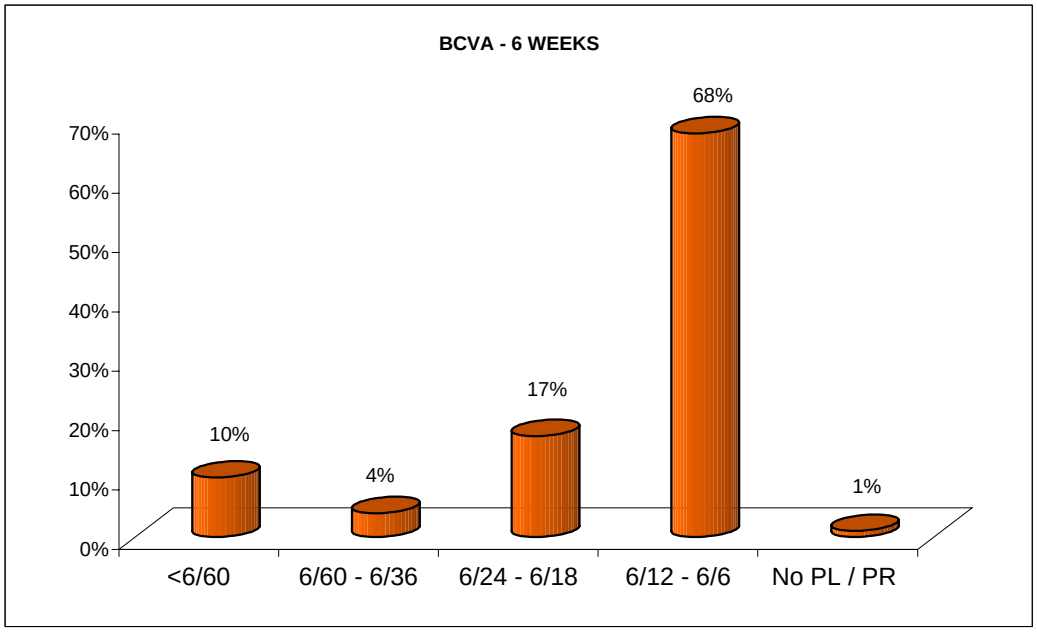

SEX INCIDENCE
33%
67%
Male
Female

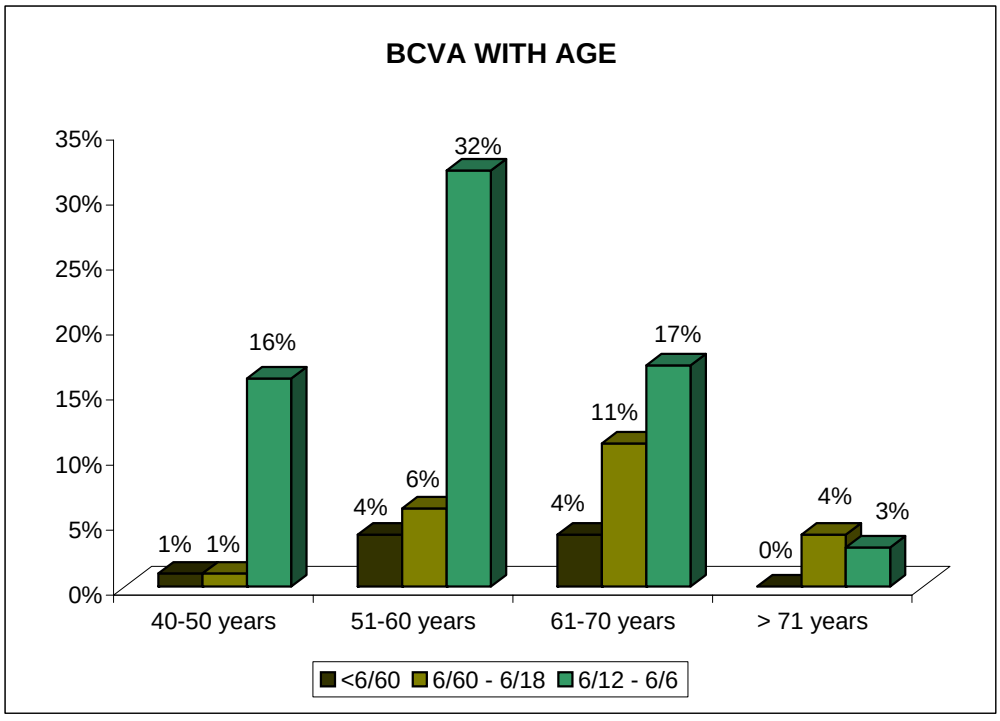

INTRA-OPERATIVE COMPLICATIONS
12%
10%
8%
6%
4%
2%
0%
12%
6%
2%
8%
3%
1%
1%
2%
1%
1%
1%
Intraopeative Miosis
PCR
Zonulodialysis
Vitreous Loss
Pigment release
Iris Bleed
SCH
Iris Tear
RLM
PPCO
Calcified anterior capsule


LONG TERM COMPLICATIONS
1%
2%
4%
1%
2%
2%
CMO
Recurrent Uveitis
Prolonged RIOP
Cyst
PCO
Evisceration


CATARACT MORPHOLOGY
60%
50%
40%
30%
20%
10%
0%
54%
23%
3%
16%
1%
2%
1%
Immature Cortical
Mature Cortical
Hypermature cortical
Nuclear
Polychromatic
Bread Crump
Snowflake


POST-OPERATIVE COMPLICATIONS
(48 hrs)
Suprachoroidal haemorrhage
1%
Reactivation Choroiditis
1%
Periorbital Bruise
1%
Vitreous in wound
1%
IOL Malposition
1%
Wound leak
1%
Hyphaema
1%
Iris Prolapse
2%
RIOP
4%
RLM
3%
Iritis
10%
Corneal Edema
19%
0%
5%
10%
15%
20%


BCVA - 6 WEEKS
70%
60%
50%
40%
30%
20%
10%
0%
10%
4%
17%
68%
1%
<6/60
6/60 - 6/36
6/24 - 6/18
6/12 - 6/6
No PL / PR


BCVA WITH AGE
35%
30%
25%
20%
15%
10%
5%
0%
1%
1%
16%
4%
6%
32%
4%
11%
17%
0%
4%
3%
40-50 years
51-60 years
61-70 years
> 71 years
<6/60
6/60 - 6/18
6/12 - 6/6

S NO.	NAME	AGE	SEX	DOS	IP NO	MORPHOLOGY	Diagnosis	SURGERY	IOL	SYSTEMIC ASSOCIATIONS	PRE OP GL Y CONTRL	DURATION OF DM	TYPE OF DM	OCULAR CO MORBITIDY	PER OP COMPL	POST OP COM	LONG TERM COMP	48 HRS V-A	BCVA	BSCS	RETINOPATHY STATUS
1	CHINNAPONNU	70	2	6/12/2005	40225	N	5-M	4-E	A	HT	O / D	6	2	NIL	PCR / VD /	CO / RIOP	CMO	2./60	3./60	NIL	NIL
2	KESAVAN	69	1	15/10/5	41729	C	5-I	4-S	PC	NIL	O / I	4	2	NIL	NIL	NIL	NIL	6./18	6/9P	NIL	NIL
3	SEENIAMMAL	60	2	18/9/5	41833	C	5-I	3-S	PC	NIL	O/I	4	2	NIL	NIL	NIL	NIL	6./12	6./6	NIL	NIL
4	MAHESWARI	50	2	18/9/5	41870	C	3-PS 4-IM	4-S	PC	AS	I	3	2	AL	NIL	NIL	NIL	6./9	6./6	NIL	NIL
5	RAGHUPATHY	75	1	20/9/5	42586	C	3-PS 4-M	4-S	PC	HT	O / I	8	2	NIL	NIL	NIL	NIL	6./24	6/9P	NIL	BDR & HT GR-1 / SAME
6	SULEKA BEE	53	2	25/9/5	04421	P-POLU	5 - PSC	3-S	PC	HT / IHD / HYP/OBE	O / I	8	2	NIL	PR	NIL	NIL	6./24	6./9	YES	NIL
7	VALLIAMMAL	60	2	2/10/2005	04428	C	3-IM 4-PS	3-S	PC	NIL	O /I	2	2	NIL	IOM	IRITIS	NIL	6./18	6/6P	NIL	NIL
8	KASTHURI	57	2	17/9/4	45228	C	4-IM 3-PS	4-S	PC	HT	O /I	7	2	NIL	NIL	NIL	NIL	6./12	6./9	NIL	BDR SAME
9	BEGUM BEE	50	2	3/10/2005	45247	C	3-PS 4-IM	4-S	PC	NIL	I	3	2	NIL	NIL	NIL	NIL	6./9	6./6	NIL	NIL
10	KASTHURI	52	2	6/10/2005	46410	C	5-IM	3-S	PC	OBESITY	O/I	3	2	NIL	NIL	RLM	NIL	6./60	6/9P	NIL	NIL
11	KALIAMMAL	75	2	3/10/2005	46407	N	5-N	3-E / PI	A	HT	I	2	2	NIL	NIL	CO / RLM	NIL	10D - 3/60	6./36	NIL	NIL
12	VEDHA MANICKAM	66	1	15/10/5	46417	C	5-IM	3-S	PC	NIL	O/I	8	2	NIL	NIL	HY	NIL	HM	5./60	NIL	NIL
13	KANNIAPPAN	61	1	12/10/2005	46412	C	3-A 4-PMG	4-S	A	NIL	O/I	6	2	PM.GL	CC/PCR/VD/ SY	IRITIS	CMO	10D-6/36	4./60	NIL	NIL
14	INDRANI	40	2	25/10/5	47403	SF	5 IM 3 PTERYGIUM	3-S PT EX	PC	NIL	I	3	MODY	PTERYGIUM	NIL	NIL	NIL	6./24	6/6P	NIL	NIL
15	RAJESHWARI	58	2	27/9/05	44083	C	3-PLG/ 4-PS	3-S/EV	PC	HT/	I	8	2	HT/PLG	IOM	SUPRA CHOROIDAL HE	EVISCERATION	NIL	NIL	NIL	NIL
16	ROSE	55	1	13/6/5	22847	C	5-M	4-S	PC	NIL	I	5	2	CFHOROIDITIS	NIL	CO/UVEITIS	UVEITIS	CFCF	HM	NIL	NIL

S NO.	NAME	AGE	SEX	DOS	IP NO	MORPHOLOGY	Diagnosis	SURGERY	IOL	SYSTEMIC ASSOCIATIONS	PRE OP GLY CONTRL	DURATION OF DM	TYPE OF DM	OCULAR CO MORBITIDY	PER OP COMPL	POST OP COM	LONG TERM COMP	48 HRS V-A	BCVA	BSCS	RETINOPATHY STATUS
17	KRISHNAN	60	1	13/6/05	25400	C	5-IM	5-S	PC	HT	O/I	4	2	NIL	IP/IRIS TEAR/ IOM	IP	NIL	6./36	6./9	YES	NIL
18	RAJAMANI	60	2	21/5/5	48421	C	5-IM	3-S	PC	AS	O/I	1	2	NIL	NIL	NIL	NIL	6./9	6./6	NIL	NIL
19	LAKSHMIAMMAL	65	2	15/6/5	48413	C	4-M 3-A	4-S	A	NIL	O/I	7	2	NIL	NIL	CO	NIL	2./60 WITH 10D	6./18	NIL	NIL
20	RUKHMANI	50	2	7/12/2005	53361	N	4-N 3 A	4-S	PC	NIL	I	1	2	NIL	PRIMARY PCO	NIL	NIL	6./18	6./12	YES	NIL
21	MUTHU	65	1	5/12/2005	47443	C	5-POAG/IM	5-T/E	PC	NIL	O/I	7	2	POAG	NIL	NIL	NIL	6./24	6./18	YES	NIL
22	LAKSHMI	50	2	7/12/2005	53391	C	3-IM 4-M	4-S	PC	NIL	O/D	3	2	NIL	NIL	NIL	NIL	6./36	6./12	NIL	NIL
23	ABOORVAM	60	2	15/6/5	52906	C	5-IM	4-S	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./36	6./12	YES	MOD NPDR SAME
24	MADHEENA BEGUM	50	2	5/12/2005	53856	C	5IM	3-S	PC	IHD	I	4	2	NIL	PCR / VD/IOM	NIL	NIL	6./60	6./9P	NIL	NIL
25	KANIAMMAL	65	2	15/6/5	54585	C	3-M 4-IM	3-S	PC	NIL	O/I	9	2	NIL	RLM/IOM	CO	NIL	HM	FC	NIL	NIL
26	SUBRAMANI	76	1	21/9/5	39971	C	3-IM 4-PS	3-S	PC	NIL	I	4	2	NIL	Nil	IRITIS	NIL	6./24	6./12	NIL	NIL
27	SULOCHANA	64	2	21/9/5	19361	N	5 N	4-S/3-S	PC	IHD	O/I	18	2	NIL	NIL	NIL	NIL	6./36	6./24	YES	MILDprgnMOD NPDR
28	ADHILAKSHMI	50	2	23/3/5	11819	C	3-M,4-IM	5 S	PC	NIL	O/I	1	2	NIL	NIL	NIL	NIL	6./12	6./6	YES	NIL
29	REGINAMARY	60	2	21/9/5	20253	C	3-PS 4-IM	4-S	PC	NIL	O/I	1	2	NIL	NIL	NIL	NIL	6./18	6./9	NIL	NIL
30	SUNDARI	65	2	4/7/2004	20798	C	3-PS 4-IM	4-S	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./18	6./9	NIL	NIL
31	THAMYLNAYAGI	70	2	24/6/4	17472	C	3-PS 4-IM	4-S	PC	HT	O/I	8	2	HT-RPTY/ARMD	NIL	NIL	NIL	6./36	6./18	NIL	MILDNPDR SAME
32	PAPATHY	68	2	13/6/4	14181	P-BC	5-IM 4-PSCC	4-S	PC	NIL	O/I	6	2	OLD UVEITIS	IRIS TEAR	IRITIS	NIL	6./24	6./18	NIL	NIL

S NO.	NAME	AGE	SEX	DOS	IP NO	MORPHOLOGY	Diagnosis	SURGERY	IOL	SYSTEMIC ASSOCIATIONS	PRE OP GLY CONTRL	DURATION OF DM	TYPE OF DM	OCULAR CO MORBITIDY	PER OP COMPL	POST OP COM	LONG TERM COMP	48 HRS V-A	BCVA	BSCS	RETINOPATHY STATUS
33	INDRANI	50	2	8/6/2004	18095	c	5-IM	3-S	PC	NIL	O/I	8	2	NIL	ZD/VD/IOM	VITREOUS IN WOUND	CMO	5./60	3./60	NIL	BDR SAME
34	KHADER	65	1	2/6/2004	17664	C	5-IM	3-S	PC	NIL	O/I	8	2	NIL	Nil /IP	NIL	NIL	6./36	6./18P	NIL	BDR SAME
35	LAKSHMI	60	2	26//5/4	18655	C	5-IM	3-S	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./24	6./9	NIL	NIL
36	DILLIBABU	69	1	13/5/4	15047	C	5-IM	4-S	PC	IHD	O/I	3	2	NIL	NIL	NIL	NIL	6./36	6./12	NIL	NIL
37	MANNAN	65	1	5/5/2004	13726	C	3-N/4-M	3-S	PC	NIL	O-D	5	2	NIL	NIL	NIL	NIL	6./36	6./12	NIL	NIL
38	RAJAMMAL	55	2	3/5/2004	16013	C	3-PS/4-M	4-S	PC	HT	O/D	7	2	NIL	NIL	CO	NIL	6./24	6./9	NIL	MILDprgnMOD NPDR
39	RANI	60	2	7/12/2005	54588	C	3-M 4-PS	3-S	PC	HT	O/D	5	2	NIL	NIL	CO	NIL	6./60	6./18	NIL	NIL
40	BAREETH	70	1	5/1/2005	3972	C	5-M	4-S	PC	HT	O/I	10	2	NIL	NIL	NIL	NIL	6./24	6./9	YES	NIL
41	VELAYUTHAM	55	1	5/1/2005	45350	C	3-M 4-IM	3- S	PC	NIL	O/I	2	2	NIL	PR	NIL	NIL	6./18	6./9	NIL	NIL
42	NAGAMMAL	60	2	5/1/2005	1103	C	5-IM	4-S	PC	OBE	O/I	6	2	NIL	NIL	NIL	NIL	6./18	6./12	YES	NIL
43	JAGEERABEE	60	2	5/1/2005	4115	N	5-N	3 S	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./24	6./6	NIL	NIL
44	VENKATAMMA	65	2	11/2/2005	5120	C	3-PS 4-M	4-E	PC	NIL	O/I	5	2	NIL	ZD/VD	CO	NIL	6./24	6./9	NIL	NIL
45	JEYA	69	2	4/2/2005	2280	C	5-IM	3-S	PC	NIL	O/I	5	2	NIL	NIL	IOL RP DONE	NIL	6./60	6./12	YES	NIL
46	VASUGI	58	2	4/2/2005	4731	N	3-N4-PS	3-E	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./36	6./6	NIL	NIL
47	MANICKAM	79	1	7/1/2005	730	N	5-N	4-E	AC	HT/AS	O/D	8	2	NIL	PCR/VD/IOM	CO	RU / PCO	6./60	1/60	YES	NIL
48	CHINNAPONNU	40	2	20/12/4	41218	C	5-IM	4-S	PC	NIL	I	10	1	NIL	NIL	NIL	NIL	6./24	6./6	YES	NIL
49	MADHAVAN	61	1	16/12/4	43082	C	5-IM	3-S	PC	HT	O/I	4	2	NIL	IOM	NIL	NIL	6./9	6./6	NIL	NIL
50	KUPPUSAMY	60	1	14/12/5	43109	C	5-IM	4-S	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./12	6./6	NIL	NIL
51	KATHIRVEL	55	1	11/12/2004	41342	C	3-HM 4-IM	3-E	PC	NIL	O/I	4	2	IRIS COLOBOMA	NIL	NIL	NIL	2./60	4./60	NIL	NIL

S NO.	NAME	AGE	SEX	DOS	IP NO	MORPHOLOGY	Diagnosis	SURGERY	IOL	SYSTEMIC ASSOCIATIONS	PRE OP GLY CONTRL	DURATION OF DM	TYPE OF DM	OCULAR CO MORBITIDY	PER OP COMPL	POST OP COM	LONG TERM COMP	48 HRS V-A	BCVA	BSCS	RETINOPATHY STATUS
52	SABHAPATHY	70	1	16/4/4	11394	N	5-N	4-E	PC	HT/IHD	O/I	8	2	NIL	NIL	CO	R IOP	5./60	6./36	NIL	NIL
53	RAMAIAH	65	1	17/9/4	30851	C	3-M 4-PS	3-S	PC	NIL	O/I	4	2	MACULAR HOLE	NIL	NIL	NIL	2./60	4./60	NIL	NIL
54	MAZUK BEGUM	65	2	22/9/4	30934	C	3-M 4-IM	3-T/E	PC	NIL	O/I	7	2	POAG	NIL	NIL	NIL	6./36	6./18	NIL	NIL
55	MUTHULAKSHMI	69	2	15/10/4	34824	N	5-N	3-S	PC	IHD	O/I	6	2	NIL	NIL	NIL	NIL	6./24	6./9	YES	NIL
56	NAGAMMAL	60	2	15/10/4	34805	C	5-IM	3-E	PC	NIL	O/I	4	2	NIL	PCR/ VD	CO	RU	6./36	6./24	NIL	NIL
57	CHANDRAMMA	75	2	27/11/4	38457	C	3-M 4-PMG	4-E	PC	NIL	O/I	7	2	PMG	NIL	NIL	NIL	6./36	6./12P	NIL	NIL
58	KASTHURI	55	2	25/11/4	38867	C	4-IM 3-AC G	3-E/T	PC	NIL	O/I	3	2	ACG	NIL	NIL	NIL	3./60	6./60	NIL	NIL
59	RADHABAI	52	2	6/12/2004	41725	C	3-IM 4 PS	3-E	PC	NIL	O/I	1	2	NIL	NIL	NIL	NIL	6./36	6./12	NIL	NIL
60	CECILY	62	2	7/12/2004	42026	C	3-M 4-IM	3-E	PC	NIL	O/I	3	2	NIL	IOM	IRITIS	NIL	6./36	6./12	NIL	NIL
61	FATHIMA BEE	54	2	3/12/2004	41469	C	5-IM	3-E	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./24	6./6	NIL	NIL
62	RAJESHWARI	60	2	3/12/2004	40785	C	3-IM 4 M	4-E	PC	NIL	O/I	5	2	NIL	NIL	NIL	NIL	6./36	6./9	NIL	NIL
63	CHINNAPPAN	75	1	25/11/4	38939	N	5-N	4-E	PC	NIL	O/I	4	2	NIL	NIL	CO	CO	5./60	6./24	NIL	NIL
64	VEERAMUTHU	42	1	24/11/4	38995	C	4-M 3IM	3-S	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./24	6./9	NIL	NIL
65	VALLIAMMAL	60	2	10/11/2004	34850	C	3-M 4 PS	3-E	PC	NIL	O/I	6	2	NIL	NIL	NIL	NIL	6./18	6./9	NIL	NIL
66	PUSPHA	59	2	9/11/2004	38130	C	5-IM	3-E	PC	NIL	O/I	4	2	NIL	NIL	CO/IRITIS	RIOP	6./36	6./24	NIL	NIL
67	MANGALASUNDARI	63	2	10/11/2004	37640	C	5-IM	3-S	PC	NIL	O/I	5	2	NIL	NIL	NIL	NIL	6./12	6./6	YES	NIL
68	LAKSHMI	55	2	3/11/2004	37445	N	3-N 4-HM	4-E	PC	NIL	O/I	8	2	NIL	NIL	NIL	NIL	6./36	6./24	YES	NIL
69	RAMALINGAM	60	1	2/11/2004	37145	C	5-IM	4-E	A	NIL	O/I	6	2	NIL	PCR/ VD	CO	CMO	6./60	5./60	NIL	NIL
70	EILAMANTHAI	70	1	29/10/4	36389	C	5-IM	3-S	PC	NIL	O/I	4	2	NIL	NIL	NIL	NIL	6./18	6./9	YES	NIL

S NO.	NAME	AGE	SEX	DOS	IP NO	MORPHOLOGY	Diagnosis	SURGERY	IOL	SYSTEMIC ASSOCIATIONS	PRE OP GLY CONTRL	DURATION OF DM	TYPE OF DM	OCULAR CO MORBITIDY	PER OP COMPL	POST OP COM	LONG TERM COMP	48 HRS V-A	BCVA	BSCS	RETINOPATHY STATUS
71	MUNYAMMAL	59	2	4/11/2004	327218	C	5-IM	3-S	PC	NIL	O/I	3	2	NIL	IOM	NIL	NIL	6./36	6./12	NIL	NIL
72	MOHANA	55	2	29/10/4	36380	C	5-IM	3-S	PC	NIL	O/I	1	2	NIL	NIL	NIL	NIL	6./12	6./6	NIL	NIL
73	ZULAKA BEE	40	2	14/10/4	34678	C	5-IM	3-E	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./24	6./9	NIL	NIL
74	MANONMANI	60	2	12/10/2004	34373	C	5-IM	3-E	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./24	6./9	NIL	NIL
75	VALLI	55	2	8/10/2004	33867	C	5-HM	4=S	PC	NIL	O/I	5	2	NIL	NIL	CO/RIOP	NIL	6./36	6./12	NIL	NIL
76	SUNAITA BEE	65	2	15/10/4	33845	C	5-IM	3S	PC	NIL	O/I	3	2	NIL	IOM, PR	NIL	NIL	6./24	6./9	NIL	NIL
77	HANIFA	60	1	7/10/2004	33697	N	3-PS 4 N	4-E	PC	NIL	O/I	4	2	NIL	NIL	NIL	NIL	6./12	6./6	NIL	NIL
78	NAGARAJ	46	1	8/10/2004	32127	C	3-IM 4-PS	3-E	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./18	6./9	NIL	NIL
79	IMMANUEL	68	1	30/9/4	32831	C	5-IM	4-E	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./12	6./6	NIL	NIL
80	KANNIGA	50	2	30/9/4	32589	C	3-M 4 PS	3-E	PC	NIL	O/I	1	2	NIL	NIL	RLM / IRITIS	NIL	6./18	6./12	NIL	NIL
81	KUPPAMMAL	65	2	22/9/4	3122	C	3-IM 4-M	4-S	PC	NIL	O/I	2	2	ARMD	NIL	NIL	NIL	6./60	6./36	NIL	NIL
82	PREMILA	45	2	22/9/4	31661	C	3-IM 4-PS	3-S	PC	NIL	I	20	1	NIL	NIL	NIL	NIL	6./24	6./9	NIL	MOD NPDR SAME
83	AMARALLI	52	1	22/9/4	31739	C	5-IM	3-S	PC	NIL	O/I	4	2	NIL	NIL	IRITIS	NIL	6./18	6./12	NIL	NIL
84	RANGANATHAN	65	1	17/9/4	30931	C	3-IM 4-PS	3-S	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./18	6./9	NIL	NIL
85	SAMPATH	61	1	16/9/4	31015	N	5-N	3-E	PC	HT	O/I	3	2	COR-NEAL OPACITY	NIL	CO	NIL	6./36	6./18	NIL	NIL
86	NEELA	48	2	15/9/4	30852	C	5-IM	3-E	PC	NIL	O/I	2	2	NIL	NIL	NIL	CYST	6./12	6./9	NIL	NIL
87	KUPPAMMAL	70	2	2/9/2004	28487	C	5-IM	3-E	PC	NIL	O/I	7	2	NIL	IRIS BLEED	CO/RIOP	NIL	6./36	6./24	NIL	NIL
88	JABURALA KHAN	64	1	30/8/4	28286	C	5-I	4-S	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./9	6./6	NIL	NIL
89	JAGADHEESAN	55	1	30/8/5	28325	C	3-PS 4-IM	4-S	PC	NIL	O/I	2	2	NIL	NIL	WOUND LEAK	NIL	5./60	6./24	NIL	NIL

S NO.	NAME	AGE	SEX	DOS	IP NO	MORPHOLOGY	Diagnosis	SURGERY	IOL	SYSTEMIC ASSOCIATIONS	PRE OP GLY CONTRL	DURATION OF DM	TYPE OF DM	OCULAR CO MORBITIDY	PER OP COMPL	POST OP COM	LONG TERM COMP	48 HRS V-A	BCVA	BSCS	RETINOPATHY STATUS
90	SAROJA	60	2	30/8/4	27763	C	5-IM	4-S	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./12	6./9	NIL	NIL
91	BEENA	50	2	29/4/4	13614	N	3-IM 4-N	4-E	PC	NIL	O/I	2	2	NIL	IOM	CO	PCO	6./60	6./12	NIL	NIL
92	SHANTHA	65	2	28/4//4	12836	N	3-N 4-PS	3-E	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./18	6./9	NIL	NIL
93	THANGAM	55	2	19/4/4	12213	C	3-PS 4-IM	4-S	PC	NIL	O/I	2	2	NIL	NIL	NIL	NIL	6./12	6./9	NIL	NIL
94	PONNUSAMY	50	1	6/4/2004	10556	C	3-IM 4-M	4-E	PC	NIL	O/I	2	2	NIL	NIL	IP/IRITIS	NIL	6./60	6./24	NIL	NIL
95	SULEKA BEE	70	2	7/7/2004	11094	N	5-N	3-E	PC	NIL	O/I	4	2	NIL	NIL	NIL	NIL	6./24	6./12	NIL	NIL
96	PONNI	55	2	6/4/2004	31024	C	5-IM	3-S	PC	NIL	O/I	4	2	NIL	NIL	PERIORBIT AL BRUISE	NIL	6./18	6./12	NIL	BDR SAME
97	FATIMA	60	2	16/9/04	32784	C	5-IM	4-E	PC	NIL	O/I	3	2	NIL	Nil	CO	NIL	5./60	6./24	NIL	NIL
98	PENCILLAMMA	52	2	4/4/2004	31274	C	5-IM	3-S	PC	HT	O/I	2	2	NIL	NIL	NIL	NIL	6./12	6./6	NIL	NIL
99	RATINAMMA	57	2	28/4/4	12876	C	3-M 4IM	3-E	PC	NIL	O/I	5	2	NIL	SCH-ANES	NIL	NIL	6./18	6./9	NIL	NIL
100	RASU	49	1	17/9/4	31298	C	5IM	4-S	PC	NIL	O/I	3	2	NIL	NIL	NIL	NIL	6./12	6./6	NIL	NIL