Comparative Study Of Outcome Of Cataract Surgery In Diabetic And Non Diabetic Patients

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LIST OF ABBREVIATIONS USED

BCVA	Best Corrected Visual Acuity
CCC	Continuous Curvilinear Capsulorrhexis
CME	Cystoid Macular Edema
DME	Diabetic Macular edema
DR	Diabetic Retinopathy
ECCE	Extra Capsular Cataract Extraction
ETDRS	Early Treatment of Diabetic Retinopathy Study
FBS	Fasting Blood Sugar
ICCE	Intra Capsular Cataract Extraction
IL	Interleukins
IOL	Intraocular lens
IOP	Intraocular pressure
LEC	Lens Epithelial Cell
logMAR	Logarithm of Minimum Angle of Resolution
NPDR	Non Proliferative Diabetic Retinopathy
NVE	Neovascularization Elsewhere
NVI	Neovascularization of Iris
NVG	Neovascular Glaucoma
PCO	Posterior Capsular Opacification

PCIOL	Posterior chamber intraocular lens
PDR	Proliferative Diabetic Retinopathy
POST OP	Post-operative
PRE OP	Pre-operative
SICS	Small incision cataract surgery
SRK	Sanders, Retzlaff, Kraff
VEGF	Vascular Endothelial Growth factor

ABSTRACT

Aims & Objectives:

To compare the visual outcome following cataract surgery in diabetics and non-diabetics.

Method:

A comparative study of 58 eyes in the diabetic and 58 eyes in the non-diabetic group that underwent small incision cataract surgery with posterior chamber intraocular lens implantation. Age, sex, surgical technique, follow up, pre- and postoperative best corrected visual acuity (BCVA) and post-op complications were evaluated

Results:

Out of 116 patients, 58 were diabetic and 58 were non diabetics. In this study, in diabetic group 26 (44.8%) were males and 32 (55.2%) were females. Among non-diabetic 19 (32.8%) were males and 39 (67.2%) were females. The mean age group of patients in diabetic group was 56.5±7.4 and 59.6±5.2 in non-diabetic group. In this study all diabetics were on treatment and 58.6% patients had good glycemic control prior to surgery. All patients underwent small incision cataract surgery. Follow up duration was 4 weeks. The mean preoperative best corrected visual acuity in the diabetic group was 1.60±0.81 and in non-diabetic group was 1.62±0.87. The mean post-operative best corrected visual acuity in log MAR units in the diabetic group was 0.39±0.32 and in the non-diabetic group was 0.32±0.27. The difference in pre and post op visual outcome was statistically significant (p=0.001). The difference in pre and post op visual outcome was statistically significant (p=0.001). Post-operative visual acuity of 6/12 or better was achieved in 62.1% eyes in diabetics and 69% among non-diabetics. Post-operative complications included: posterior capsular

opacification, corneal edema, anterior chamber reaction striate keratopathy, pigment dispersion, cystoid macular edema and vitreous. The incidence was higher in the diabetic group.

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INTRODUCTION

Diabetes mellitus is one of the common systemic problems affecting a variety of people worldwide. Various risk factors for development of diabetes include population growth, aging, sedentary lifestyles, urbanization and an increased obesity prevalence. Epidemiological data suggests that there is an increasing incidence of diabetes mellitus in developing countries. By 2030, it is estimated that global prevalence of diabetes would reach approximately 4.4%.

Cataract is one of the leading cause of blindness globally, nearly 18 million People are affected. Approximately two third of the diabetic population shows evidence of cataract. Diabetes mellitus influences the function and morphology of the eye lens. Diabetes mellitus is a risk factor for development of cataract. Cataract is the second most common ocular complication of diabetes mellitus after diabetic retinopathy. Cataracts occur at an early age in diabetics compared to non- diabetics and 2-5 times more common in diabetic patients. So cataract surgery in diabetics is often done earlier. Apart from visual improvement, diabetic patients need cataract surgery for the assessment and treatment of posterior segment pathology. In India approximately 20% of all cataract surgery is done in diabetics[1]

Poor visual outcome after cataract surgery in diabetics associated with the severity of pre-existing retinopathy and diabetic maculopathy prior to the surgery. Peroperatively in diabetics it is recognized that there is higher incidence of pigment dispersion and fibrinous reaction in the anterior chamber, together with the development of posterior synechiae, as well as increased risk of capsule rupture and vitreous loss. Diabetic patients are more prone to postoperative complications such as Rubeosis, neovascular glaucoma, macular edema (Diabetic and cystoid), severe inflammation, (Iritis, uveitis, endophthalmitis) vitreous hemorrhage, synechiae to

IOL, retinal detachment and corneal decompensation. Diabetic are more prone to develop posterior capsule opacification postoperatively.

The increasing incidence of diabetes in developing countries such as India necessitates an assessment of the surgical outcome of diabetic cataract among the study population.

AIM AND OBJECTIVE OF THE STUDY

TO COMPARE THE VISUAL OUTCOME FOLLOWING CATARACT SURGERY IN DIABETICS AND NON DIABETICS

REVIEW OF LITERATURE

Study done by Raj Kumar Gupta in 2017 on "Evaluation of Outcome of Cataract surgery in Diabetic and Non-Diabetic Patients: A Comparative Study" at a Tertiary Care Teaching Hospital in Uttar Pradesh assessed prognosis of cataract surgery in 50 diabetic patient and 50 non-diabetic patient. At one week's time, the occurrence of post surgical acuity in the diabetic and non diabetic group was found to be 0.15 and 0.21 respectively. At one month's time, the occurrence of post surgical acuity in the diabetic and non diabetic group was found to be 0.30 and 0.37 respectively.

Pigment dispersion is the most commonly encountered post-surgical complication in both the groups[2].

Study done by Zhu et al in 2017 on "Visual-related quality of life and visual outcomes from cataract surgery in patients with vision-threatening diabetic retinopathy: a prospective observational study", included total of 126 (153 eyes) who were diagnosed with cataract and stabilized vision threatening diabetic retinopathy. Postoperative BCVA improvement was seen in 92.86% of the patients. The variation of BCVA after surgery was statistically significant after.

The Log MAR BCVA improved from to 0.58 +/- 0.30 after 3 months from preoperative vision of 0.82 +/- 0.34 and the total score of CLVQOVL improved to 95.35+/- 20.65 after 3 months from pre-operative vision of 76.02+/- 24.82[3].

Study done by Afzal Qadir in 2013 "To Determine the frequency of common complications following cataract surgery in diabetic patients". Out of 129 patients, Uveitis (most common complication) was seen in 20 (15.5%) eyes, while progression of diabetic retinopathy (least common) seen in only 10 eyes (7.75%). Worse visual acuity was seen in 14(10.85%) and there was improvement in visual acuity in 89.15%.

Striate keratopathy and PCO were found in 16(12.40%) and 15(11.62%) eyes respectively. Complications were seen more frequently in the age group 51-60 years as compared to the other age group[4].

Study done by Alex A Ilechie in 2012 on "Evaluation of Post-Operative Visual Outcomes of Cataract Surgery in Ghana": concluded that Over 41.2% of post-operative eyes in this study had very good visual outcome. Nevertheless, greater attention to post-operative care and uncorrected refractive error is needed[5].

Study done by Oluwatoyin H et al in 2009 on "Cataract surgical outcomes in diabetic patients: case control study" concluded that visual improvement was observed following cataract surgery of advanced cataract in diabetic patients in this study [6].

Lavanya R et al. in their study in 2009 showed 5% of the sample of the Malay population aged 40-80years in Singapore had cataract surgery. One in ten had post operative best corrected visual acuity of 20/60 or worse, largely related to in Diabetic Patients. The first group consisted of 50 patients with concomitant retinal diseases[7].

Study done by Ivancic D in 2005 on "Cataract Surgery And Postoperative Complications In Diabetic Patients". It consisted of 50 non diabetic patients with cataract who had not suffered from systemic or local diseases and 50 patients with cataract and diabetes mellitus that had lasted for atleast for 5 years. In both groups patients underwent identical cataract exctracapsular extraction with intraocular lens implantation. Postoperative visual acuity was worse in the patients with diabetes on 1 week and 6 months. It was diabetic retinopathy and its progression that caused deterioration of visual acuity.

Study done by Squirrell D in 2002 on a "prospective case control study of the natural history of diabetic retinopathy and maculopathy after uncomplicated

phacoemulsification cataract surgery in patients with type 2 diabetes" concluded that uncomplicated surgery does not cause acceleration of diabetic retinopathy postoperatively and any progression that is observed probably represents the natural history of the disease. Although macular oedema is common after cataract surgery it may follow a benign course and in many patients the development of clinically significant macular oedema postoperatively probably represents natural disease progression rather than being a direct effect of surgery.

Study done by Zaczek A in1999 on "Cataract surgery and IOL implantation in diabetic patients"

concluded that the final visual outcome was improved in the majority of diabetic eyes. Eyes with clinically significant macular oedema at the time of surgery had the worst prognosis regarding postoperative VA[8].

Study done by Gabric N on "Timing of cataract surgery in diabetics" in 1996. In this retrospective study, they studied the outcome of extracapsular cataract extraction (ECCE) followed by posterior chamber intraocular lens implantation (PCIOL), in 2864 operated eyes, out of which 546 (19%) were in diabetics. The preoperative retinal status was recorded in all patients. In 6 (1%) patients, laser photocoagulation was performed preoperatively. The post-op visual acuity was 0.4 or better in 90.0% of diabetics and in 91.8% of non-diabetics, showing no significant difference in the postoperative complications between diabetics and non- diabetics. The results indicated the cataract operation (ECCE + PCIOL) to be a well tolerated surgical procedure in diabetic patients with cataract, in whom it should be even earlier performed than in non-diabetics, because cataract prevents the diagnosis or treatment of a suspect retinal disorder.

Study done by Dowler JG et al in 1995 on "Visual acuity in diabetics following extracapsular cataract extraction: meta analysis" concluded that severity of maculopathy and retinopathy prior to cataract surgery in diabetics are the major determinants of visual acuity in diabetics[9].

Study done by Pasquier N et al in 1991 on "Cataract surgery and implants in diabetics" concluded that visual acuity improved by 2 lines or more in 65% cases[10].

Study done by I A Cunliffe in 1987 on "Extracapsular cataract surgery with lens implantation in diabetics with and without proliferative retinopathy". Retrospective study of all diabetics and non-diabetic matched controls who underwent extracapsular cataract extraction with intraocular lens implantation over a two-year period . Of the diabetic patients 76% eyes improved by at least two lines of Snellen acuity postoperatively. Of these patients 68% eyes and of the control eyes 83% achieved an acuity of 6/12 or better. In the diabetics the visual outcome depended on the state of the retinopathy and in particular the maculopathy[11].

LENS ANATOMY AND EMBRYOLOGY

LENS EMBRYOLOGY

Learning of lens embryology helps us to know better the anatomy of lens and cataracts.

Lens cells (surface ectodermal origin) formed during embryogenesis, overlying the optic vesicle (neuro-ectodermal origin) which thickens to form lens placode. In the meantime, the optic vesicle starts invaginating and lens pit formed in lens placode. Lens pit invaginates in to the optic cup as cells multiply and eventually pinches off as the inverted lens vesicle. During this time lens vesicle consists a cuboidal cells layer within the outer basement membrane. The lens capsule is formed by the outer basement membrane

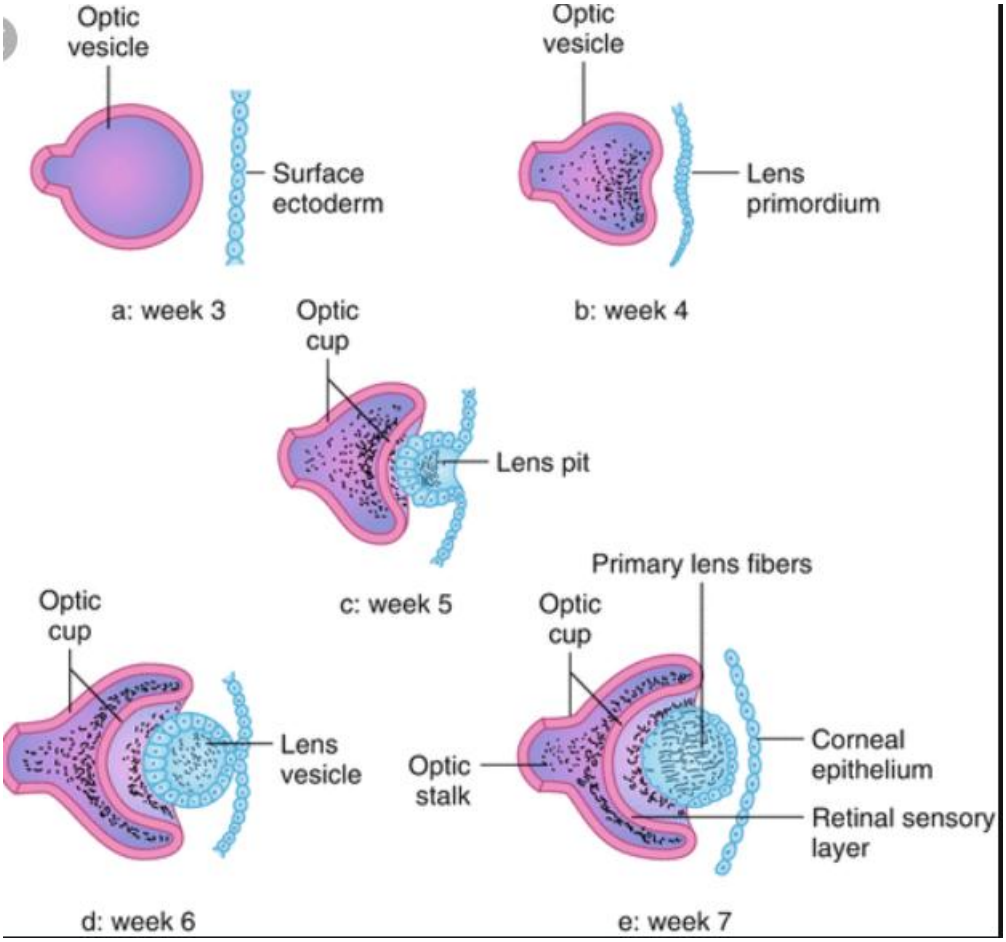
The retinal half of the lens vesicle differentiate and anteriorly, cuboidal cells begin to elongate to become primary lens fibres. Primary lens fibres meet the cuboidal cells of anterior lens, lumen of the vesicle is obliterated. Primary lens fibres forms the embryonic nucleus. Anterior cuboidal cells, commonly known as the lens epithelium. Lens epithelial layer present anterior and posterior to the equator. Posterior part of lens is devoid of epithelial cells.

Lens epithelial cells near the equator undergo mitotic division and forms secondary fibres. These secondary fibres elongate under lens epithelium anteriorly and beneath lens capsule posteriorly.

Fetal nucleus formed by secondary lens fibres during gestation and continue to develop adding fresh layers. As lens fibres grow, they stretch out from the equator and meets anteriorly and posteriorly during fetal growth and forms Y-shaped sutures during fetal growth. Lens fibres surround the fetal nucleus during childhood and early adolescence to form the juvenile or infantile nucleus. These lens fibres grows further

to form the adult nucleus. Lens cortex formed by subsequent growth of lens fibres around the entire nucleus.

During fetal development the lens nucleus becomes enveloped within the tunica vasculosa lentis, a nutritive support structure supplied by the hyaloid structure atrophies and usually disappears by birth[12].



SURGICAL ANATOMY OF LENS

Lens grows throughout life. After lens vesicle filled with lens fibre cells and starts formation of cortical fibres, the lens contains a capsule, an anterior and equatorial epithelial layer, a peripheral cortex and an inner nuclear core. In Children and young adults with visually significant cataracts will have strong capsule, firm vitreous, and soft nucleus. Older adults with age-related cataracts will have fragile capsule, a syneretic vitreous body, and a hard nucleus.

Understanding of the surgical anatomy and age-realted changes in the lens helps surgeon to plan and perform a successful procedure irrespective of patient's age.

Equatorial diameter of adult lens is 9-10 mm. By direct measurement its axial sagittal width is about 3.5-4.0 mm at birth, about 4mm at 40years and increases slowly to 4.75- 5.0 mm in extreme old age. The equatorial diameter is 6.5 mm at birth, 9-10mm in second decade and changes little there after[13].

CAPSULE

The capsule formed by the epithelial cells basement membrane of the embryonic lens vesicle. As the vesicle filled with elongated posterior vesical cells anteriorly, the capsule assumes an anterior and posterior aspect. Anterior capsule is thicker than the posterior capsule. Posterior capsule is a thin membrane that is merely adherent to the fiber cells growing along its inner surface. A two- fold increase in anterior capsule thickness occurs with age, there is two-fold increase in the thickness of anterior capsule[14]. Capsule is the thickest basement membrane in the body.

EPITHELIUM

The epithelial cells height decreases with the age and the width increases with the age. The epithelium is more sensitive to trauma. The lens epithelium consists of cuboidal cells spread over the front of the lens deep to the capsule and extending outwards to the equator. Central zone represents stable population of cells whose numbers like those of corneal endothelium slowly reduce with age. They are polygonal in flat section. Hemidesmosomes connects the basal aspects of the cells to the lens capsule. The central cells usually do not mitose but they can do so in response to damage including wide variety of injuries. Peripheral to the central zone is the intermediate zone and it consists cells are smaller. Mitoses are occasionally seen. Germinative zone is the most peripheral and is located just pre-equatorially.[14]

THE LENS FIBRES

The transition of epithelial cells in the germinative zone to elongated nucleated lens fibre cell is accompanied by decreasing of lateral interdigitations and the initiation of a pronounced elongation of the basal and apical portions of the cell, which extend backwards beneath the capsule, and forwards beneath the epithelium, respectively. Deposition of successive generations of lens fibers is associated with the formation of the nuclear bow, in which the now- flattened nuclei of lens fibers form an arch forwards when traced into the deeper portions of the lens.

The lens fibers spindle-shaped cells which arch over the lens in concentric layers from front to back. They are hexagonal in equatorial cross-section and forms radial rows in the cortex, which lose their regularity with increasing depth within the lens. Their average width is 10-12μ m and average thickness at equator is 1.5-2.

CORTEX

Different regions of adult lens cortex:

- Peripheral cortex - just underneath the anterior epithelium or the posterior capsule.
- Supra-nuclear cortex - adjacent to adult nucleus.
- Epi-nucleus - the supra-nuclear region.
- Sutures are the lines formed by adjacent ends of lens fibres[12].

NUCLEUS

Different concentric layers of the nucleus:

- Embryonic nucleus – primary lens fiber cells, formed in the lens vesicle forms the embryonic nucleus
- Fetal nucleus – the fibers laid down around the embryonic nucleus before birth forms the fetal nucleus
- Infantile nucleus – after birth, new fibers formed before puberty forms infantile nucleus
- Adult nucleus – formed after puberty

THE CILIARY ZONULE

Ciliary zonules consists of a series of fibers passing from the ciliary body to the lens. It keeps the lens in position and during accommodation it allows ciliary muscle to act on it. The lens and zonules form a diaphragm which divides the eye into a smaller anterior portion and a larger posterior portion. The zonule forms a ring which is roughly triangular in meridional section. The base of the triangle is concave and faces the equatorial edge of the lens. The apex is elongated and curved, and follows the valleys of ciliary processes to be prolonged over pars plana ciliaris to the

ora serrata. Each zonular fibre is composed of fibrils averaging 10 nm in cross-section (8 to 12 nm), and tubular in profile. They show a microperiodicity of 12 -14 nm or occasionally 40- 55µm when the fibrils arc densely aggregated.

There are four main topographic zones: the pars orbicularis (lying on the point of angulation of the zonule at the mid zone of the ciliary valleys), the zonular plexus (between ciliary processes), the zonular fork (point of angulation of zonule at the mid zone of the ciliary valleys), and the anterior, equatorial and posterior limbs of the zonule. Structurally the anterior zonule runs mainly from the pars plana to the equatorial lens, but is supplemented from the pars plicata, while the posterior zonule runs chiefly from the pars plicata to the post-equatorial lens and is supplemented from the pars plana. The equatorial zonule passes from pars plicata to lens equator.

The hyaloid zonule is a flimsier structure and plastered as a thin layer deep to the main suspensory apparatus and run a course from pars plana to the lens at the edge of the patellar fossa and attaches to the hyalocapsular zonule (corresponding to the annular fibres of Wieger's ligament).The vast majority of the zonules arise from the posterior end of the pars plana to 1.5 mm from the ora serrata, where they either run into its inner limiting membrane without entering the cytoplasm of the underlying cells become continuous with fibres of the anterior vitreous. The fibres pass forward over the pars plana as a meshwork until they reach the posterior margin of the pars plicata. They segment into the zonular plexuses and pass through the valleys between the ciliary processes and closely attached to lateral wall of ciliar process. Each zonular plexus consists of broad flattened fibre strands which cross and join each other in a regular pattern. The zonular plexuses are firmly attached to the bases of the ciliary valleys by fine and coarse fibrils which leave the main strands and run anteriorly at an acute angle. These tension fibres find anchorage to the basement membrane within the

depths of the valleys. Towards the anterior margin of the pars plicata each plexus divides into zonular fork consisting of three fibre groups running to the anterior, equatorial and posterior lens capsule, respectively. The pre-equatorial, equatorial and post equatorial insertions of the zonules into the lens capsule are macroscopically different.

At the point of insertion, the anterior zonule sends fibres and smaller fibrils (0.07 to 0.5 µ.m) to a depth of 0.6 to 1.6µm into the capsular surface. The zonular lamell thickens from 1.0 to 1.7µm, lateral to the anterior zonular insertions. The insertion of the meridional zonular fibres, 0.5 to 1.0µ.m wide, creates an antero-posterior ribbing. The equatorial fibres are sparse and poorly developed but again fan out in a brush-like manner to insert into the capsule almost perpendicularly to the surface. The posterior fibres insert in two or three layers, over a zone of 0.4 to 0.5 mm wide. They fan out more and show less inter connections than the anterior zonule. The space between the anterior and posterior zonules is known as the canal of Hannover.[14]

BIOCHEMISTRY

Based on molecular weight the structural proteins of lens are divided into alpha, beta, and gamma crystallins. Most of the enzymes are the size of beta-crystallins. They constitute the aerobic metabolism pathways in the epithelium and most superficial cortical fibre cells and anaerobic metabolism pathways in the cell cytoplasm. Main metabolic substrate of lens is glucose and it is obtained from the aqueous humor, and the energy produced by glucose metabolism is used in synthesis of protein and lipids, active transport of ions and amino acids, and normal lens hydration maintenance

SENILE CATARACT: GENERAL FEATURES

Three major types of senile cataracts are cortical, nuclear, and posterior sub-capsular—which vary in both the initial opacity location and underlying pathology. All three types of aging- related cataracts have many common risk factors and even though cataracts often start as a single type, as they mature they usually become mixed cataracts[15].

Cortical cataracts seen in the outside edge (about 25%) of the lens and are characterized by water clefts, vacuoles and wedge shaped opacities. Cortical cataracts are associated with local disruption of the structure of lens fiber cells. This damage compromises the membrane integrity and causes ionic imbalances, which leads to accumulation of water in the lens cells. In cortical cataracts, there is decrease in potassium level and increase in sodium, chloride and calcium and this imbalance leads to water influx[16].The vacuoles or "lakes" filled with water have a low refractive index compared to the protein-rich cytoplasm in the fibers, and these discontinuities causes scattering of light and cataract.

Nuclear cataracts seen in the central portion of the lens and involve an acceleration of processes that occur during aging even in the normal lens. Post-synthetic modifications, especially resulting from oxidation, leading to formation of protein aggregates that scatter light[17]. With increasing age nuclear proteins become more pigmented; in some nuclear cataracts the colour can become dark brown (brunescent nuclear cataract) or even black. Nuclear cataracts tend to become harder and less hydrated than normal, age matched lens nuclei.

Posterior sub-capsular cataracts seen in the most posterior cortical layer, just underneath the lens capsule. It may result from improper posterior suture formation or from abnormal differentiation of lens fibers[18]. Posterior sub-capsular cataracts causes

are radiation exposure and long-term steroid therapy and secondary to retinal degeneration diseases; they also occur idiopathically.

MECHANISMS OF CATARACT FORMATION

Osmotic stress

Osmotic stress leads to cataract in diabetic and galactosemic patients. In both the galactosemic and diabetic cataract, galactose and gluscose both metabolized to galactitol and sorbitol respectively by the enzyme aldose reductase through the sorbitol pathway[19,20]. The polyol pathway consists of the aldose reductase and polyol dehydrogenase (iditol dehydrogenase) enzymes. Both sorbitol and galactitol cannot cross the plasma membranes and so accumulates in the cytoplasm creating osmotic stress. This causes water influx into the cell which leads to swelling of the epithelial and fiber cells. Fluid accumulates between fiber cells in diabetic cataract. Intracellular and intercellular changes lead to formation of light scattering foci and lens opacification[21].

The role of the polyol pathway in diabetic and senile cataract is uncertain as there is not much aldose reductase activity in the lens epithelium and even less in the fiber cell cytoplasm of the cortex and nucleus[22].

It is important to maintain the proper tonicity (ion concentration) and osmolarity of fluids infused into the eye while performing intraocular surgery. Osmotically induced secondary cataracts were a frequent postoperative complication in vitrectomy surgery before the introduction of balanced salt solution. The prophylactic removal of clear lens was often done during vitrectomy to avoid formation postoperative osmotic secondary cataract as it was seen very often[23].

Protein aggregation

None of the individual crystalline proteins are large enough in the clear lens to scatter light. In Nuclear cataract and in the aging lens, different crystallins combine to form large aggregates that scatter light. Cataract is formed by the aggregation of numerous light-scattering foci in the lens. In nuclear cataract these light scattering foci aggregates may found freely in the cytoplasm and bound to cell membranes in cortical and sub-capsular cataracts[24,25].

Oxidative stress

Oxidative stress refers to oxygen and its multiple redox forms adverse effects on the lens constituents[26]. Oxygen can exist as hydrogen peroxide, singlet oxygen, hydroxyl radical, and superoxide. These redox species are produced and destroyed by enzyme processes in the lens.

The relative balance between the systems producing these oxidants and the systems destroying them determines whether or not the lens suffers oxidative damage. The defect in defense mechanism can lead to accumulation of hydrogen peroxide and (1) sulfhydryl-dependent enzyme systems are deactivated, (2) causes aggregation of proteins by forming protein–protein disulfide bridges, (3) formation of chromophores changes the lens colour or (4) membrane structure is disrupted[27,28].

Glutathione (GSH), an important antioxidant in the lens[29,30,31]. In the lens GSH takes part in a redox cycle and detoxifies hydrogen peroxide, hydroxyl radical, and dehydroascorbic acid. GSH loss is associated with damage to the membrane and aggregation of protein – factors causing early opacification[32].

Post-translational protein changes

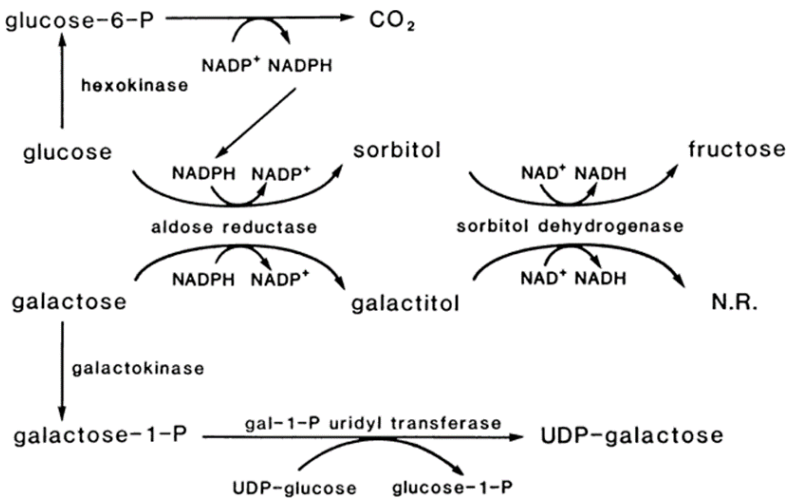
Besides oxidative damage, other lens proteins modification happen after the protein is formed and these constitute post-translational changes and comprises non-enzymatic glycosylation, racemization and aggregation[33].

Phase separation

Phase separation is the reversible mechanism of aggregation[34,35]. Certain protein molecules aggregate to form colossal groups as the temperature; although the single protein molecules are not covalently bound together, the group size is big enough to scatter light[36]. Its importance in human cataract is still unknown. Whether phase separated proteins are more likely to form covalently bound aggregates or not remains to be determined[37].

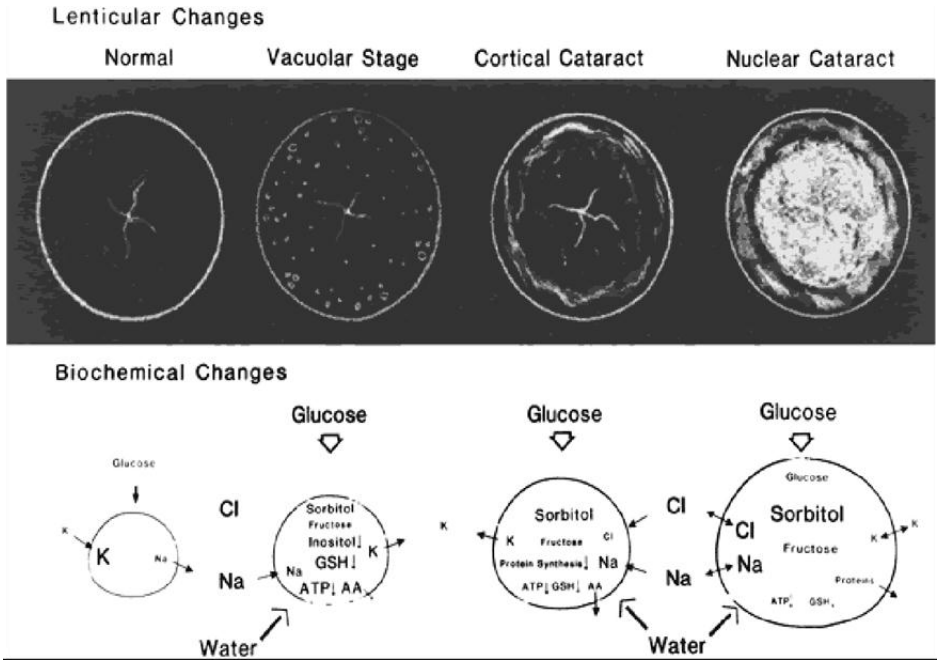
DIABETIC CATARACT: PATHOGENESIS

Diabetic cataracts have been studied most thoroughly. In 1959, observation of van Heyningen[38,39] of sorbitol accumulation in the lenses of rats with diabetes stimulated the interest in the diabetic cataract. Sorbitol is formed by the glucose metabolism by aldose reductase (the first enzyme of the polyol pathway).



Polyol pathway

Since van Heyningen's findings, the polyol pathway has been found to function not only in the lens but also in other tissues, including the cornea, iris, retina, nerve, and kidney.



Changes leading to diabetic cataract formation.

The appearance of vacuoles at the lens periphery is the first change seen in theses diabetic rats.

When cataract progresses, vacuoles extend to the anterior cortex. A dense nuclear opacity develops eventually. Histologic appearance of hydropic lens fibers is an evidence to the aldose reductase role in diabetic cataract formation. Swollen lens fibers are seen in the outer regions of the lens and the deeper layers consists of normal size lens fibers. Osmotic swelling of the affected lens fibers are swollen due to osmotic stress caused by accumulation of sorbitol[40].

Sorbitol normally do not penetrate biologic membranes well, so once its formed it accumulate in high levels in the cell if they are not metabolized rapidly. This is what occurs in the lens fiber cells in diabetes because although activity of

aldose reductase activity is high, sorbitol dehydrogenase activity is very low. Hypertonicity created due to retention of sorbitol is corrected by water influx, which leads to the swelling of the lens fibres. This osmotic swelling has harmful effects on the lens. The permeability of the membrane is adversely affected; therefore, substances such as amino acids, potassium ions and myo-inositol, which are normally kept at higher concentrations in the lens compared to the intraocular fluids around, begin to leak out[41].

As potassium level in the lens decreases, sodium and chloride level eventually increases and this electrolyte imbalance lead to the nuclear opacity. The initial action of aldose reductase thus triggers a number of events leading to opacification of the lens. Due to the extensive swelling of cortical lens fibers, the role of osmotic stress is especially important for rapid formation of cataract in young patients with type 1 diabetes mellitus. A study conducted by Oishi et al.explored whether aldose reductase is associated with the formation of cataracts in adult with diabetes. Aldose reductase levels in red blood cells of patients below 60 years of age with a short duration of diabetes were strongly associated with the incidence of sub-capsular cataracts. In diabetic patients, a negative correlation was observed between the level of aldose reductase in erythrocytes and the density of lens epithelial cells (known to decrease in diabetics compared to non-diabetics), indicating a potential role of aldose reductase in this patho-mechanism.

Many latest publications describe lens fibers damage caused by oxidative stress due to free radicals in diabetic patients. There is no evidence that free radicals start the cataract formation process. Free radicals accelerate and aggravate cataract development and there is no evidence that it begins the process of cataract formation.

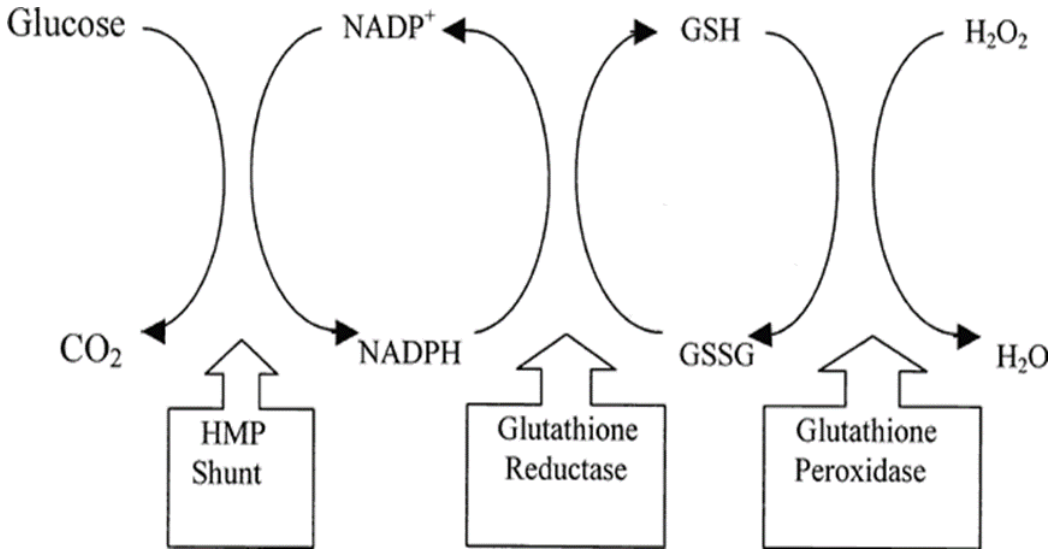
In diabetic patients aqueous humor hydrogen peroxide level will be higher and induces the generation of hydroxyl radicals (OH^-) through

Fenton reactions after entering the lens. The free radical nitric oxide (NO) also elevated in the diabetic lens and aqueous humor, which lead to an increased production of peroxynitrite, which in turn causes cell damage due to its oxidizing properties.

Further increase of glucose levels in the aqueous humor may cause glycation of lens proteins, which resulting in the superoxide radicals (O2-) generation and formation of advanced glycation end products (AGE). In the epithelium of the lens, AGE interacts with receptors for advanced glycation end products and H_2O_2 is generated.

In addition to high levels of free radicals, diabetic lenses also show an impaired antioxidant capacity, which increases its susceptibility to oxidative stress. Antioxidants loss is aggravated by glycation and lens antioxidant enzymes like superoxide dismutase inactivation. The most dominant superoxide dismutase isoenzyme in the lens is copper-zinc superoxide dismutase 1 (SOD1), which helps in the degradation of superoxide radicals (O_2^-) into hydrogen peroxide (H_2O_2) and oxygen.

Several in-vitro and in-vivo animal studies has shown the importance of SOD1 in the protection against cataract in diabetic patients



Glutathione redox cycle and its relation to the hexose monophosphate (HMP) shunt and the detoxification of H_2O_2 by glutathione peroxidase

RISK FACTORS FOR OCULAR COMPLICATIONS IN DIABETIC PATIENTS

Diabetes mellitus is a metabolic disease that affects multi organs. Concurrent systemic disorders, on the other hand, can have a significant impact on ocular complications development and progression in diabetic patients. Intensive controlling of blood sugar level and hypertension lowers the risk of fresh diabetic retinopathy and slowdown the progression of current diabetic retinopathy[42,43].

The progression of diabetic retinopathy affected by several renal diseases. Serum lipids elevation related with macular exudates and moderate visual loss. Strenuous work or exercise in patients with advanced retinopathy may cause vitreous hemorrhage. During pregnancy diabetic retinopathy progression may occur. Anemia can lead to progression of diabetic retinopathy and smoking should be avoided[43]. Several studies regarding diabetic cataracts related to development have shown that

high blood sugar level is associated with loss of lens transparency in a cumulative manner[44]. Sudden decrease in the blood sugar level in patients with marked hyperglycemia may cause transient lens opacification and swelling as well as hyperopia. It also been suggested that sudden glycemic control can increase the opacification of lens irreversibly[45].

RISK FACTORS FOR CATARACT FORMATION IN DIABETES

Cataract is one of the earliest complications of diabetes mellitus. Klein et al[46] reported that risk of developing cataract is 2-5 times more in diabetics than non-diabetics. In diabetics less than 40yrs of age, the risk may reach 15-25 times[47]. Even impaired fasting glucose (IFG), a pre-diabetic condition is considered as a risk factor for cortical cataracts development[48]. "In a study from Iran, Janghorbani and Amini evaluated 3,888 type 2 diabetic patients who were free of cataracts at initial visit and reported a rate of cataract formation of 33.1 per 1000 person-years of observation after a mean follow-up of 3.6 years"[49].

PREVENTION OF CATARACTS

Non enzymatic glycation of lens proteins, oxidative stress and activated polyol pathway are the three molecular mechanism involved in the development of cataract. Even though Inhibitors of glycation (Aspirin, Ibuprofen, Aminoguanidine and Pyruvate), antioxidants (Vitamin C, Vitamin E, Carotenoids, Trolox and Hydroxytoluene) and aldose reductase inhibitors (Zenarestat, Eplarestat, Imirestat, Ponalrestat, Zopolrestat, M-79175 and BALAR18) have showed potential for prevention of cataracts in animal models[50].

ANTERIOR SEGMENT CHANGES IN DIABETES

Diabetes affects the morphological, metabolic and physiological properties of the cornea. The diabetic keratopathy seen in more than 70% of diabetic patients[51] and it includes increased epithelial fragility and recurrent erosions[52], reduced corneal sensitivity,[53,54,55,56] increased auto-fluorescence [57], impaired wound healing[58], altered epithelial and endothelial barrier functions [59], and predisposition to cornealedema[60] and infectious ulcers.

In diabetic patients, confocal microscopy has shown lower basal cell density. It may be due to alterations in the basement membrane, increased turnover rate in the basal epithelial cells and decreased innervation at the level of sub-basal nerve plexus. Confocal microscopy reveals abnormal stromal and sub-basal nerve plexuses in the cornea of diabetics. These alterations are seen more in diabetic with proliferative retinopathy than diabetics without retinopathy. Sub-basal nerve plexus appears more thick and tortuous[61]. Diabetics shows reduced cell density at the level of mid corneal stroma[62]. Study conducted by Inoue et al showed decreased corneal endothelial cell density and increased coefficient of variation in cell area in diabetics than non-diabetics[63].

Not much difference seen in the central corneal thickness. Formation of cataract is most important change in lens. Thicker lens capsule is seen in diabetics which is more friable and may rupture during intra-capsular lens extraction in diabetics[64]. This capsule changes may affect capsulorrhexis.

Study conducted by Schafer et al in diabetics found higher incidence of cortical cataract as documented by Schiemflug photography and densitometric analysis[65]. Study conducted by Saxena et al shows two times higher incidence of cortical cataract in diabetics of duration more than 5 years. They also found increased

incidence of posterior capsule cataracts in recently diagnosed diabetic patients. They reported no significant association between nuclear cataract and diabetes or IFG. Snow-flake cataract (subcapsular opacities) with abruspt onset and acute progression typically seen in young patients with uncontrolled diabetes. Its rare and initial presentation of diabetic cataract[66].

Iris changes like leathery consistency and a miotic pupil were seen more common in diabetics than non-diabetics. Glycogen accumulation leads to vacuolization of iris pigment epithelium. Pigment dispersion seen due to iris trauma or surgery[65]. In patients with severe retinopathy abnormal iris trans-illumination seen. Changes in iris pigment epithelium is due to hypoxia[68].

Various studies found high prevalence of POAG (primary open angle glaucoma) and elevated mean IOP in diabetic patients. On other hand, high prevalence of impaired glucose metabolism is seen in glaucoma patients[69]. Due to its effect on small vessels, diabetes found to increases the susceptibility of optic nerve fibers to glaucoma damage Studies have reported a higher prevalence of both elevated mean IOP. Whether diabetes is an independent risk factor POAG remains controversial. Ocular hypertension study (OHTS) found that no association between diabetes and conversion of ocular hypertension to glaucoma. Neovascularization of iris (NVI) is one of the most dreaded anterior segment complication in diabetics. NVI is due to hypoxia and ocular ischemia ischemia and its characterized fine branching vessels on stroma of iris and trabecular meshwork. It also accompanied by fibrous membrane. Contraction of these fibrovascular membrane lead to formation of peripheral synechiae which causes secondary angle closure glaucoma and neovascular glaucoma (due to disorders which causes retinal or ocular ischemia, most commonly diabetes)

TIMING OF CATARACT SURGERY

Now a days approach towards diabetic cataract and its management has changed. Study conducted by Pollack et al[70] showed visual acuity better than 20/40 in 31% of patients and found the main cause for poor visual prognosis was macular edema. They also suggested that cataract surgery should not be done in diabetics with retinopathy until the vision is less than 20/100- 20/200. Later study done by Schatz et al[71] reported better pot-operative vision of 20/40 only in 9% of patients. Change in the attitude regarding management of cataract surgery has contributed to an improved visual outcome. Cataract surgery also helps in the evaluation and timely treatment of diabetic retinopathy and macular edema[72].

In various studies where late cataract extraction was done shows worst visual outcomes due to late identification and treatment of CSME because of lens opacity. In diabetics where cataract extraction is done before lens opacity precludes evaluation of posterior segment. This early intervention shows decrease risk of CSME and improved visual outcomes[73].

PREOPERATIVE CONSIDERATIONS

Prior to surgery, detailed ophthalmic examination such as visual acuity assessment, best corrected visual acuity (BCVA), detailed slit lamp examination and grading of cataract, measurement of intraocular pressure, sac syringing, gonioscopy (for neovascularization) and dilated fundoscopy is done. patients should have controlled blood sugar and no evidence of infection. Other diagnostic procedures like OCT and B-scan done when its necessary. In complicated cases, vitreoretinal surgeon opinion is advised by few authors[74]. As there is chance of progression of proliferative diabetic retinopathy after cataract surgery, it is recommended to do PRP

preoperatively unless lens opacity precludes PRP[75]. In cases where lens opacity precludes PRP its done after surgery. As pre-existing macular edema aggravate posteriorly and associated with poor visual prognosis it should be treated adequately prior to surgery[76]. Patients with neovascularization of iris (NVI) also need PRP. Risk of postoperative complications are high with active NVI. Anti-VEGF agents such as bevacizumab shows promising role in treatment of neovascular glaucoma[77,78]. In diabetics, one of the major cause of transient refractive changes is hyperglycemia. It is due to morphologic and functional changes seen in the crystalline lens[79]. Glycemic changes cause corneal topographic parameters changes and cause error in kerato-refractive and cataract surgery[79,80].

GRADING OF CATARACT ACCORDING TO LOCS III

The LOCS III system evaluates four features which include

- Nuclear Opalescence(NO)
- Nuclear colour(NC)
- Cortical Cataract(C)
- Posterior Subcapsular Cataract(PSC)

Nuclear opalescence and colour are graded on a decimal scale, based on a set of six standardized photographs (N01 TO NO6 AND NC1 TO NC6).

Cortical and posterior sub-capsular cataracts are graded based on five standardized photographs. (C1 TO C5 AND P1 TO P5)

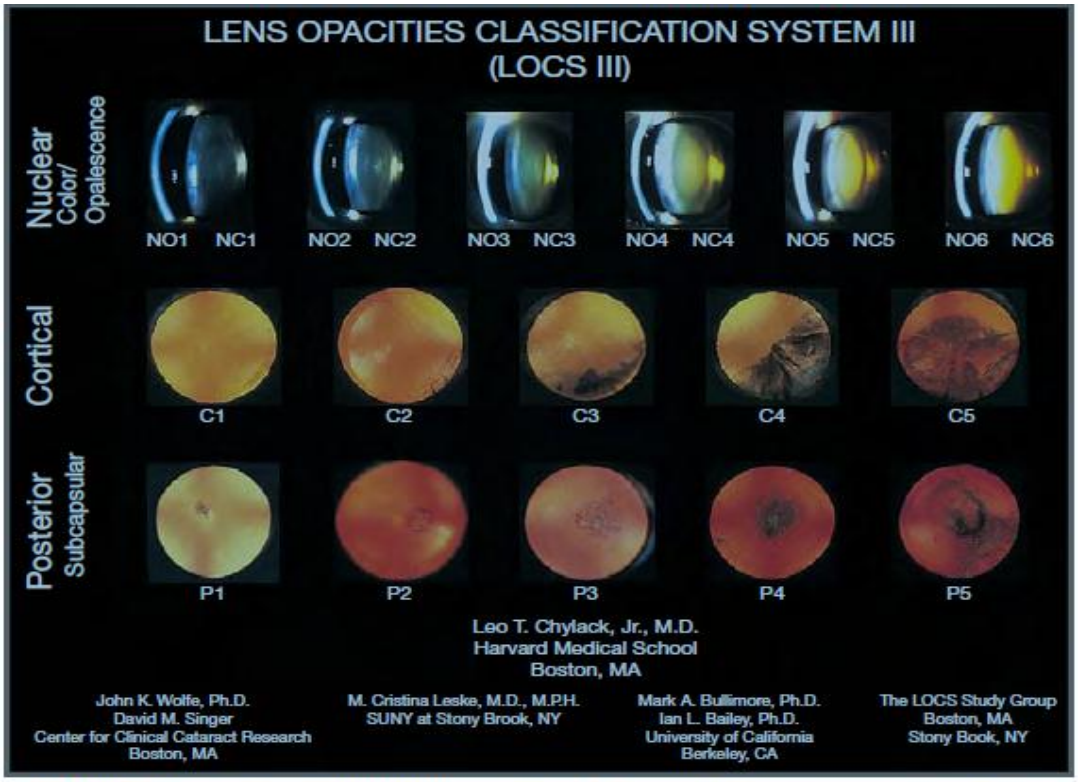


FIGURE 108.4. LOCS III. This set of standards is prepared as a set of slides for grading standardized photographic images of opacity. The five or six individual standard slides for the cataract type of NC being graded are projected at the same size as the slides of unknown opacity. NO1 to NO6 and NC1 to NC6 are the standards for NO and nuclear color, respectively. C1 to C5 are the standards for cortical cataract, and P1 to P5 are the standards for posterior subcapsular cataract.

CATARACT SURGERY

TECHNIQUE:

In diabetic patients post-operative complications after cataract surgery are more compared to non-diabetic patient. The most done cataract surgeries are small incision cataract surgery and phacoemulsification. The most preferred cataract surgery technique now a dyas is phacoemulsification Phacoemulsification. Kelman developed phacoemulsification technique in 1967 and it became popular after 1996. It advantages over SICS are rapid visual rehabilitation, less post-operative astigmatism and inflammation and less incidence of pot-op PCO due to modern foldable lenses. Now a days cataract surgery done earlier in diabetics as it require clear media for evaluation and treatment of posterior segment pathology

CORNEAL COMPLICATIONS:

In diabetic patients corneal sensation will be often reduced due to involvement of peripheral nerves and limbal vasculopathy. Diabetic patients exhibit corneal epithelium abnormalities, which leads to corneal erosion, persistent epithelial defect, or corneal ulcers. Recurrent corneal erosion recurrence is due to trauma and mild epithelial breakdown following cataract surgery.

The adhesion of corneal epithelium to the underlying stroma in diabetics is weakened due to hemidesmosomes reduction. High level of erythrocyte aldose reductase is seen in type 2 diabetes and it leads to increase sorbitol accumulation and damage the corneal epithelium. Few studies reported corneal abnormalities like punctate keratopathy, recurrent corneal erosion, limbal vascularization and endothelial dystrophy in diabetic patients up-to 73.6%. Extra care is needed during cataract surgery in diabetics to protect the corneal epithelium.

PUPIL RELATED COMPLICATIONS:

Diabetic eyes often have poor pupillary dilation, particularly when active rubeos is iridis or even regressed neovascularization[78] is present. Pupil stretching should be avoided because these vessels can rupture and cause intraocular bleeding. In patients with small pupil during cataract surgery, pupil is enlarged using intracameral adrenaline, by performing multiple sphincterotomies and using pupil-stretching techniques or iris retractors. Capsulorrhexis size should be larger than normal to prevent anterior capsular phimosis and it should be smaller than the diameter of IOL optic to reduce incidence of PCO.[79,80,81].

RETINAL PHOTIC INJURY:

Altug et al[83] found that increase prevalence of photic retinopathy in diabetic patients compared to non-diabetic during cataract surgery. Its necessary to precautions to prevent photic injury in diabetics as they are more vulnerable to it

INTRAOCULAR LENS CHOICE:

In diabetics use of large diameter IOLs helps in the visualization and treatment of the peripheral retina in the management of diabetic retinopathy[84]. Incidence and severity of PCO is more in the diabetics than non-diabetics following cataract surgery. A square edge optic design inhibits proliferation of lens epithelial cells proliferation and thus prevent formation of PCO. Use of hydrophobic acrylic shows less incidence of PCO and thus may be the IOL of choice in diabetic patients. It also shows low propensity to silicone oil adhesion and hence the IOL of choice in diabetics anticipating vitreo-retinal surgery.

High level of phosphorus in serum aqueous humor in diabetics, especially with proliferative diabetic retinopathy may cause hydrophilic acrylic IOLs opacification[85].

"Rodriguez-Galietero et al evaluated changes in contrast sensitivity and colour discrimination in diabetic patients with implantation of a blue-light filtering IOL compared with an ultraviolet- only filtering IOL. They found that blue-light filtering IOLs did not cause chromatic discrimination defects[86,87]."

INTRAOCULAR LENS IMPLANTATION SITE:

In diabetics as there is high risk of iris neovascularization, anterior chamber IOLs and iris claw lenses should be avoided. Various studies reported that the safest procedure for diabetics is extracapsular surgery with in the bag implantation of a posterior chamber IOL.

VISUAL PROGNOSIS FOLLOWING CATARACT SURGERY

Recent studies on diabetic cataracts shows less incidence of complications and better visual outcomes than previous studies. These changes may be due to improved preoperative management of diabetic retinopathy, advanced surgical techniques and extensive glycemic and hypertensive control. Diabetic patients generally show favorable visual outcomes following cataract surgery. Diabetic patients without retinopathy shows visual prognosis as same as non- diabetic patients. Presence of significant retinopathy, CSME and poor preoperative visual acuity (due to diabetic maculopathy, ischemia and traction) are risk factors for poor visual prognosis following cataract extraction.

A prospective study conducted by Mozaffarieh et al found that patients with more severe diabetic Retinopathy may show no functional improvement despite

apparent improvement in visual acuity[88]. This shows the significance of educating patients regarding visual prognosis prior to surgery.

INDICATORS OF POOR VISUAL OUTCOMES FOLLOWING CATARACT SURGERY

Several factors are associated with poor visual prognosis in diabetics after cataract surgery. According to the Early Treatment of Diabetic Retinopathy Study (ETDRS)[89], pre-existing CSME is associated with poor postoperative visual prognosis and final visual acuity worse than 6/60.

The severity of diabetic retinopathy at the time of cataract surgery is also associated with poor post-operative visual acuity. Poor preoperative visual acuity (due to macular edema, ischemia and traction) also a risk factor for poor postoperative visual prognosis.

COMBINED CATARACT SURGERY AND VITRECTOMY

In diabetic patients who undergoes vitrectomy often have co-existing cataracts and cataract also one of the post-operative complications of vitrectomy[90]. Advancement in technology of vitreoretinal and cataract surgeries allow us to perform combined cataract surgery and vitrectomy. Combined surgery is safe, effective and visual outcome is comparable with sequential surgery. Combined surgery also provides rapid visual rehabilitation and avoids multiple surgical intervention. Various studies reported that laser photocoagulation and vitreoretinal interface plays a role in development of persistent CSME and combined surgery in these patients show significant visual improvement[93]. Selection of patient is very important for good

outcome following combined surgery. Combined surgery is recommended in the patients above 60years of age as they have high chances of developing cataract following vitrectomy and it also recommended in patients where cataract precludes membrane peeling or with pre-existing cataract[94]. Combined surgery is not recommended in patients with severe traction and ischemia and with active neovascularization of iris and in younger patient with little lens opacity[95].

CATARACT SURGERY AND INTRAVITREAL INJECTIONS

Use of intravitreal steroids has shown its efficacy in reducing macular edema. In patients with CSME and no epi-retinal membrane or traction intravitreal steroids may be given during cataract surgery[96,97]. Intravitreal bevacizumab (Avastin) is used in the treatment of neo-vascular and exudative ocular diseases since 2005[98]. Various studies have shown its effect on neovascular complications seen in diabetes but its use during cataract surgery has been not evaluated[99].

POSTOPERATIVE CONSIDERATIONS

Patients whose retina was not visualized prior to cataract surgery and patients with pre-existing proliferative diabetic retinopathy should evaluate and monitor retinal status closely after surgery. As there is a chance of progression of retinopathy after cataract surgery, patients diagnosed with non-proliferative diabetic retinopathy prior to surgery should undergo a detailed fundus examination within 3 months of surgery. The most dreaded anterior segment complication following cataract surgery in diabetic patients is neovascularization of iris(NVI).

The incidence of NVI reduced due to advancement in technique of cataract surgery. PRP and intravitreal bevacizumab used to control NVI but its effect is short

lived. Common anterior segment complications seen in diabetics are posterior synechiae, pupillary block, pigments on the IOL and iritis. High incidence of fibrin reaction is seen in diabetics. Several studies have suggested that diabetes have an increase tendency to develop endophthalmitis after cataract surgery and its associated with poor visual prognosis[100].

Corneal complications in diabetes are mostly due to excessive surgical manipulation and it may also occur spontaneously[101]. Due to impaired corneal sensation they are more prone to develop corneal epithelial defects and persistent erosions, it occurs more frequently with increasing age and duration of diabetes. Wavelike epitheliopathy after phacoemulsification has also been reported[102]. Patients are treated with lubricating drops and ointments, eye patching or temporary tarsorrhaphy. Discontinuation of topical medications may help in the healing of corneal defect as many preparations contain preservatives which are toxic[103,04]. For persistent epithelial defects therapeutic soft contact lens is used but the drawback is the risk of corneal ulceration. Newer options are fibronectin, growth factors (epidermal and insulin like growth factor and substance P)[105], plasmin/plasminogen activator[106,107] and amniotic membrane transplantation. The randomized study conducted in rats with diabetes showed topical application of insulin (QID for one week) normalized the delayed corneal wound healing[108]. Severe corneal endothelial cell damage and delay in the recovery of corneal edema was seen in diabetic after cataract surgery.

"Study conducted by Lee et al compared corneal endothelial cell damage following phacoemulsification and intraocular lens implantation in diabetic patients categorized by the severity of diabetic retinopathy and normal patients, and they found greater reduction of corneal endothelial cell density and increased coefficient of

variation in cell size in patients with high risk proliferative diabetic retinopathy(PDR)[109"].

DIABETIC RETINOPATHY:

Many studies are done regarding role of cataract surgery in the acceleration of progression of diabetic retinopathy. The rate of progression diabetic retinopathy found to be 21%-32% after 1 year of cataract surgery. Study conducted by Borrillo et al showed progression rate of 25% after 6 months of follow-up period. Retrospective study of 150 eyes of 119 diabetics undergoing phacoemulsification showed same progression of retinopathy in 25% of pateints within 6-10 months follow-up period. A study conducted by Krepler et al of 42 patients undergoing cataract extraction found diabetic retinopathy progression of 12% in operated eyes and 10.8% in non- operated eyes in 12 months follow-up period. Study conducted by Squirrell et al found out of 50 patients, progression of retinopathy in 20% of operated and 16% of non-operated eyes during 12 months follow-up period.

"A retrospective study conducted by Liao and KU reported out of 19 eyes with preoperative mild to moderate non proliferative diabetic retinopathy, 11eyes (57.9%) showed progression of diabetic retinopathy 1 year after surgery, while 12 eyes (63.2%) had progressed 3 years postoperatively[112]". The progression rates were statistically significant when compared to eyes without preoperative retinopathy. A recent prospective study evaluating 50 diabetics with and without retinopathy by OCT found 22% incidence of macular edema but macular edema not seen in pateints without retinopathy[113]. Incidence of postoperative macula edema and cystoid abnormalities increased to 42% (11 of 26 eyes) when patients with diabetic

retinopathy were evaluated exclusively. In eyes with no retinopathy, minimal changes in center point thickness baseline values was seen. In moderate non proliferative diabetic retinopathy or proliferative diabetic retinopathy an increase from baseline of 145 microns and 131 microns at 1 month and 3 months, respectively was found. Statistically significant difference was seen in retinal thickening between the 2 groups at 1 and 3 months.

The progression of diabetic retinopathy after intracapsular (ICCE) and extracapsular (ECCE) cataract extraction is well documented. Few studies show progression in diabetic retinopathy after phacoemulsification surgery and few studies found no significant change. Many believe cataract surgery accelerates progression of diabetic retinopathy while others found natural course of the condition per se is more important. "In a retrospective study, Hauser et al evaluated the occurrence and progression of diabetic retinopathy; their data suggested that diabetic retinopathy was associated with male sex, disease duration and poor glycemic control. Progression of pre- existing diabetic retinopathy was associated with poor blood sugar control. This study is limited by its retrospective nature, the relatively small number of cases and not being able to differentiate the natural course of the disease from the effect of surgery".

A prospective study conducted by Dowler et al in which surgery was performed in one eye and the fellow eyes served as controls, they found uneventful phacoemulsification surgery does not accelerate progression of diabetic retinopathy[114]. Another prospective study conducted by Squirrell et al also found uneventful phacoemulsification does not accelerate progression of diabetic retinopathy and any observed retinopathy progression is due to natural course of disease.

ETDRS report number 25 shows trend towards acceleration of progression of retinopathy in operated eyes compared to non-operated fellow eyes, but this trend was not statistically significant. The progression of diabetic retinopathy is due to impaired retinal blood barrier and postoperative inflammation after cataract surgery.

Uneventful phacoemulsification and IOL implantation, on post-op day 1 shows increase in the concentration of vascular endothelial growth factor (VEGF), hepatocyte growth factor, interleukin-1 and pigment epithelium derived factor but it takes one month to decline to preoperative levels. These results confirm altered concentrations of angiogenic and growth factors after cataract surgery, which may induce subclinical or even clinical worsening of diabetic retinopathy and maculopathy[115].

DIABETIC MACULAR EDEMA:

After cataract surgery there will be alteration in the angiogenic factors concentrations which aggravates maculopathy. Following uneventful cataract extraction, OCT imaging has showed increased thickness of retina after uneventful cataract surgery in diabetic patients without retinopathy compared to non-diabetics. In diabetics, trend toward increase in retinal thickness was evident up to 3 months after surgery. ETDRS report number 25 suggested that there is no statistically significant difference in the prevalence of macular edema before and 1 year after surgery in patients without clinically significant macular edema at the time of surgery. Another study found fresh clinically detectable macular edema incidence is 56% in one year after surgery and spontaneous resolution occurred by 6 months and 1 year after surgery without any treatment was 50% and 75% respectively. Other studies reported

that development of CSME in many patients post-operatively may be due to natural course of disease rather than the surgery effect.

Pre-existing CSME showed no spontaneous resolution at the end of one year and most of them showed evidence of deterioration. Reports of severe macular edema seen after cataract extraction is due to deterioration of pre-existing macular edema (not diagnosed or treated due to lens opacity) post-operatively.

It is difficult to distinguish diabetic macular edema from pseudophakic cystoid macular edema (Irvine-Gass syndrome) post-operatively. Fluorescein angiography may help in differentiating these two. Pesudophakic cystoid macular edema shows petaloid pattern with optic disc hyper-fluorescence without any retinopathy.

Need of laser photocoagulation in post-operative diabetic macular edema is controversial[113]. In study conducted by Pollack et al shows that focal laser photocoagulation was needed only in minority of patients who developed post-operative macular edema.

Study conducted by Dowler et al shows spontaneous resolution of post-operative macular edema but not in the pre-existing macular edema, suggesting need of early laser photocoagulation is unnecessary in all cases of post-operative diabetic macular edema[114]. Usually argon laser treatment is not performed until 6 months after cataract surgery.

CYSTOID MACULAR EDEMA:

Even though the visual outcome of cataract surgery is good, diabetic patients may have poor visual outcome compared to non-diabetic patients. Diabetic retinopathy may show rapid acceleration cataract surgery leads to macular changes

like cystoid macular edema. In patients with active proliferative diabetic retinopathy and pre-existing macular edema the worst outcomes may be seen.

In diabetic patients with or without diabetic retinopathy the blood-aqueous barrier is impaired, which leads to inflammation and development of a cystoid macular edema, a process that is exacerbated by cataract surgery. Duration of surgery, wound size and posterior capsular rupture or vitreous loss are the few factors which influences the incidence of cystoid macular edema.

Study conducted by Liu et al showed severely impaired blood aqueous barrier in diabetics with proliferative diabetic retinopathy than patients with non-proliferative retinopathy after phacoemulsification surgery.

Medicare beneficiaries analysis done from 1997 to 2001 showed that rate of development of cystoid macular edema was higher in diabetic patients after cataract surgery.

POSTERIOR CAPSULAR OPACIFICATION:

Posterior capsule opacification (PCO) is a common complication of cataract surgery (20 to 50% five years after surgery). Due to advancement in surgical technique and in IOL technology incidence of PCO is reduced. Proliferation of Lens epithelial cells (LECs) causes PCO. LECs proliferation is affected by many factors like optic-haptic Junction, optic edge design and IOL material. Postoperative inflammation is an another important determinant. Residual LECs are stimulated by surgical trauma and contact with IOL and produces cytokines. LECs affected by these cytokines and leads to collagen formation and fibrous metaplasia. In diabetic patients, impaired blood-aqueous barrier leads to postoperative inflammation. Many believe that incidence of PCO is more common in diabetic patients than non-diabetic patients

but it still remains controversial. Newer diagnostic modalities have facilitated the PCO evaluation.

In study conducted by Hayashi et al showed no difference in the incidence of PCO between diabetic and non-diabetic patients up to 1 year after cataract surgery. At one and half year and later the increased incidence of PCO was seen in the diabetic group. Their results also show the need of laser capsulotomy is more in diabetics compared to non-diabetics. "Ebihara et al using the POCO system (a software for semi objective assessment of PCO) also reported that incidence and severity of PCO is more in diabetics than non-diabetics after cataract surgery[82]."

High incidence of anterior capsular contraction to in diabetic patients with diabetic retinopathy was reported by another study.

MATERIALS AND METHODOLOGY

1.Source of Data:

Patients admitted in _____ _____ in Ophthalmology department

2.Method of Collection of Data:

Study design: Comparative study.

Study period: December 2017 – July 2019

Place of study:

_____

_____

Sample size: 58 non diabetics and 58 diabetic (Type 2) Patients.

3.Inclusion criteria:

1. Patients with type 2 diabetes mellitus.
2. Age group 40- 65 years

4.Exclusion criteria:

1. Patients with traumatic or complicated cataract.
2. Neovascularisation of iris
3. Secondary glaucoma
4. Iridocyclitis
5. Uncontrolled diabetes
6. Posterior segment causes of visual loss in diabetics.

5. Methodology:

Pre-operative evaluation

All patients were admitted to the hospital one day prior to surgery. All these patients underwent pre-operative evaluation and complete ophthalmic examination, including a thorough history with required demographic data. Systemic evaluation was also carried out.

Ophthalmic examination included

1. Best corrected visual acuity
2. Slit lamp examination and grading of cataract done according to LOCS III.
3. Biometry for IOL calculation using which includes Keratometry using keratometer and Axial length measurement using A- Scan and IOLcalculation using SRK II/T formula.
4. Posterior segment evaluation using Indirect ophthalmoscopy, B- Scan and OCT if required.
5. RBS, FBS, PPBS, Hb1Ac

Pre-operative preparation:

One day before surgery, one drop of Gatifloxacin eye drops was instilled 4 times. Pre-op pupillary dilatation was obtained using Tropicamide and phenylephrine 2.5% eye drops and nepafenac eye drops instilled alternatively every 15 minutes, an hour prior to surgery.

PROCEDURE:

ANAESTHESIA:

Anaesthesia and akinesia of the globe was achieved by peri-bulbar block of 8 ml mixture of 2% xylocaine and hyaluronidase (1500 IU). Before starting the

procedure, the eye to be operated was painted with 10% povidone iodine and the same was instilled in the cul-de-sac for 5 mins.

SURGERY:

In all patients small incision cataract surgery with posterior chamber intraocular lens implantation under peri-bulbar anaesthesia was done.

Manual Small Incision Cataract Surgery Technique:

Under aseptic precautions eye was draped, a wire speculum was placed and superior rectus bridle suture was passed and clamped on to the towel. A fornix based conjunctival flap was made. Superficial sclera vessels were cauterized. A 6mm straight incision was made on the scleral 1.5 to 2mm away from the limbus. Sclero-corneal tunnel was made. A side port entry was made with paracentesis knife. Capsule is stained using trypan blue dye through the side port. A continuous curvilinear capsulorhexis was performed. Anterior chamber was entered with angled keratome Hydro-dissection was performed. Nucleus prolapsed into the anterior chamber and delivered out using either sandwich technique. Cortical matter aspired with simcoe cannula, a PMMA IOL was implanted in the capsular bag. Anterior chamber was formed with ringer lactate, side port opening was sealed by stromal hydration. Sub-conjunctival 0.2ml (40mg/ml) Gentamycin and 0.3ml (4mg/ml) Dexamethasone, total 0.5CC was given at end of procedure. Pressure pad was applied at end of surgery.

Post-operative evaluation:

On the first post-operative day, all the patients were subjected to detailed slit lamp examination and fundus examination. Visual acuity was assessed. The patients

were discharged on the second or third post-operative day. On discharge all patients were put on corticosteroid and antibiotic combination eye drops 6 times per day, which was then tapered over a period of 6 weeks. The patients were asked to review at 1 week and 1 month from the date of surgery. Visual acuity was recorded on every visit. At all subsequent visits, patients were subjected to the slit-lamp examination, fundus examination and visual acuity recording. Assessment of anterior chamber inflammation: Aqueous flare and cells were graded by the SUN Working Group Grading Scheme for Anterior Chamber Cells & Flare.

GRADE	CELLS IN FIELD (High intensity, 1x1 mm slit beam)	ANTERIOR CHAMBER FLARE
0	<1	None
+1	1 – 5	
+2	6 – 15	Faint, barely detectable
+3	16 – 25	Moderate (Iris and lens details clear
+4	26 – 50	Marked (Iris and lens details hazy)
+5	>50	Intense (Fibrinoid reaction)

ASSESSMENT OF PCO:

Pupils were dilated and slit lamp bio-microscopy using retro-illumination was performed giving special attention to posterior capsule. PCO grading was done as done by Kucuksumer Yet al., by subjective assessment of the extent and density (assessed by its adverse effect on BCVA) of the lens epithelial cell (LEC) migration on the posterior capsule as follows:

Grade- 0: Posterior Capsule completely clear and no LEC migration.

Grade-I: LEC migration at the periphery with a clear visual axis.

Grade-II: LEC migration onto the visual axis with no drop in BCVA.

Grade-III: LEC migration onto the visual axis with BCVA better than 6/12.

Grade-IV: LEC migration onto visual axis and a BCVA of 6/12 or worse.

STATISTICAL ANALYSIS

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean± standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square ($\chi 2$) test was used for association between two categorical variables. The difference of the means of analysis variables between two independent groups was tested by unpaired t test. If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analyzed using SPSS software v.23.0. and Microsoft office 2007.

RESULTS

The study group consists of 58 eyes of diabetics and 58 eyes of non-diabetics that underwent small incision cataract surgery with posterior chamber intraocular lens implantation under peri- bulbar block. The age and sex wise distribution, glycemic control, preoperative visual acuity, coexistent morbidities, complications of the procedure and final visual outcome were analyzed.

TABLE 1 : DISTRIBUTION OF AGE BETWEEN STUDY GROUPS

Age(yrs)	Diabetic		Non diabetic		p value
	N	%	N	%	
≤40	3	5.2%	0	0.0%	0.093
41-50	10	17.2%	4	6.9%	
51-60	26	44.8%	32	55.2%	
>60	19	32.8%	22	37.9%	
Total	58	100.0%	58	100.0%	

FIGURE 1 : DISTRIBUTION OF AGE BETWEEN STUDY GROUPS

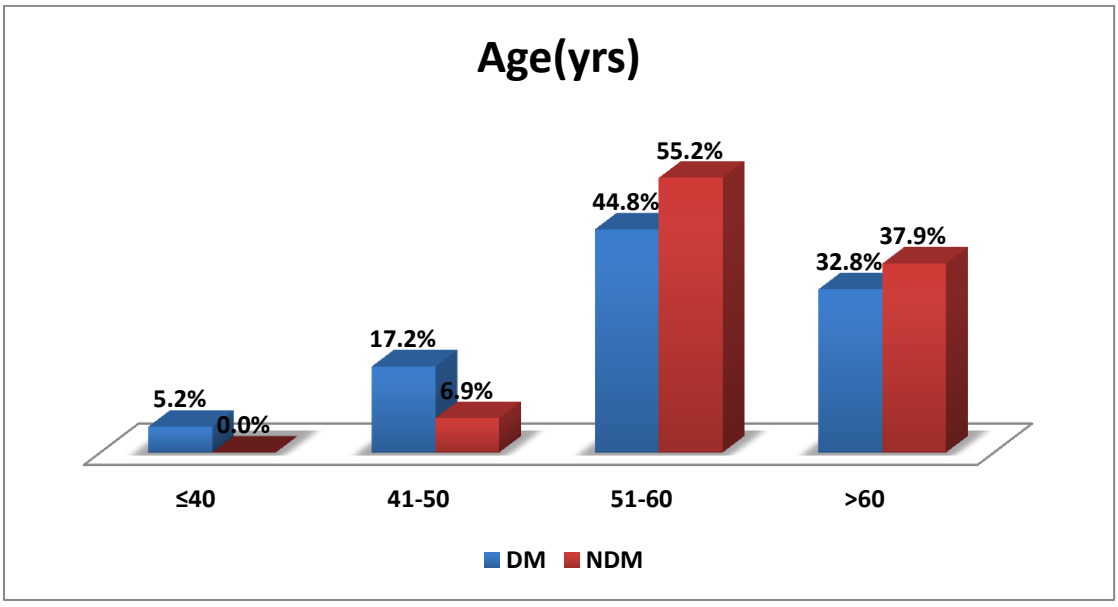
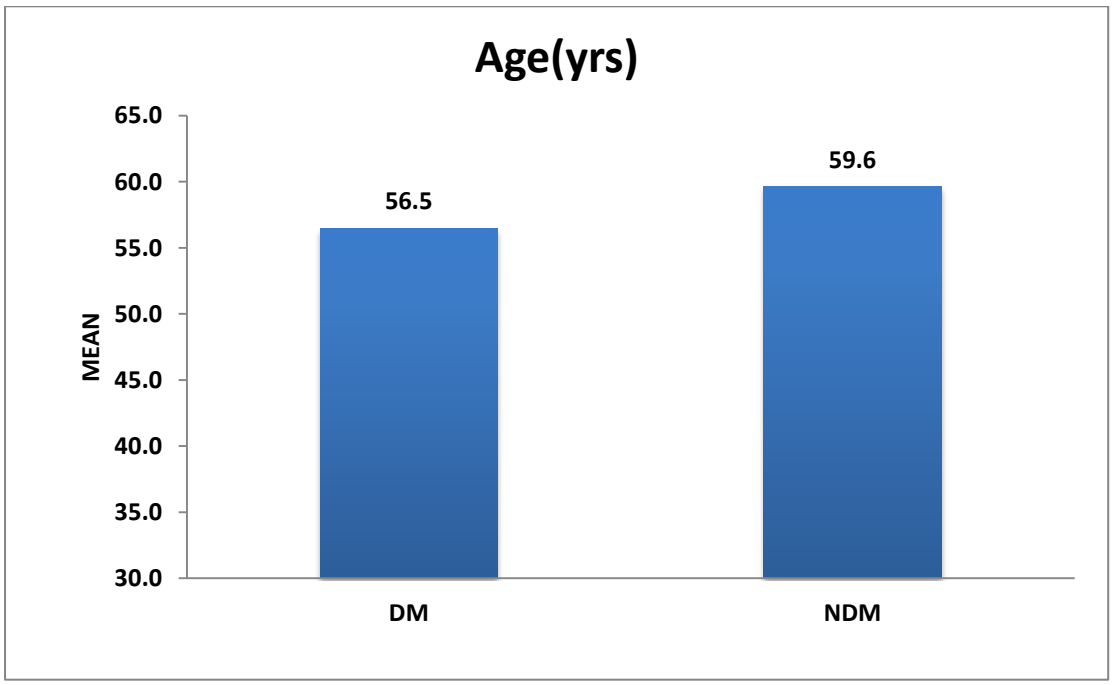


TABLE 2 : MEAN AGE BETWEEN STUDY GROUPS

Age(yrs)	DM		NDM		p value
	Mean	SD	Mean	SD	
	56.5	7.4	59.6	5.2	0.009*

FIGURE 2 : MEAN AGE BETWEEN STUDY GROUPS

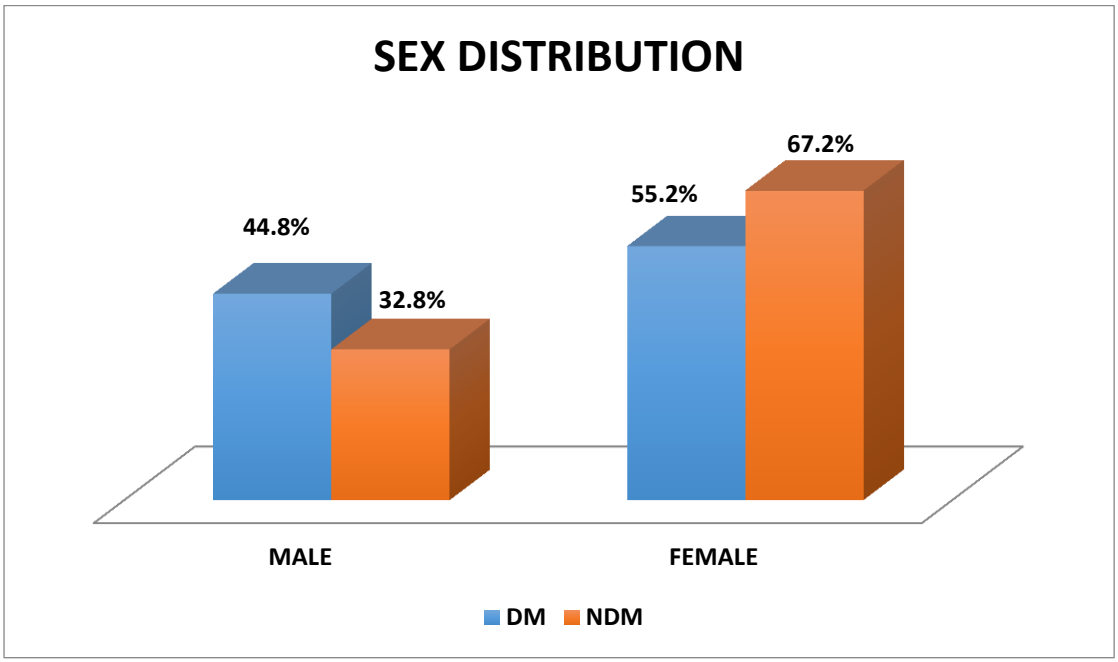


In this study, highest number of patients were in the age group of 51-60 i.e 26 (44.8%) in diabetics and 32 (55.2%) in non-diabetics. In this study patients below 40 and above 65 years of age were excluded. The mean age group of patients in diabetic group was 56.5±7.4 and 59.6±5.2 in non-diabetic group

TABLE 3 : DISTRIBUTION OF SEX BETWEEN STUDY GROUPS

SEX	Diabetic		Non-diabetic		p value
	N	%	N	%	
MALE	26	44.8%	19	32.8%	0.182
FEMALE	32	55.2%	39	67.2%	
Total	58	100.0%	58	100.0%	

FIGURE 3 : DISTRIBUTION OF SEX BETWEEN STUDY GROUPS

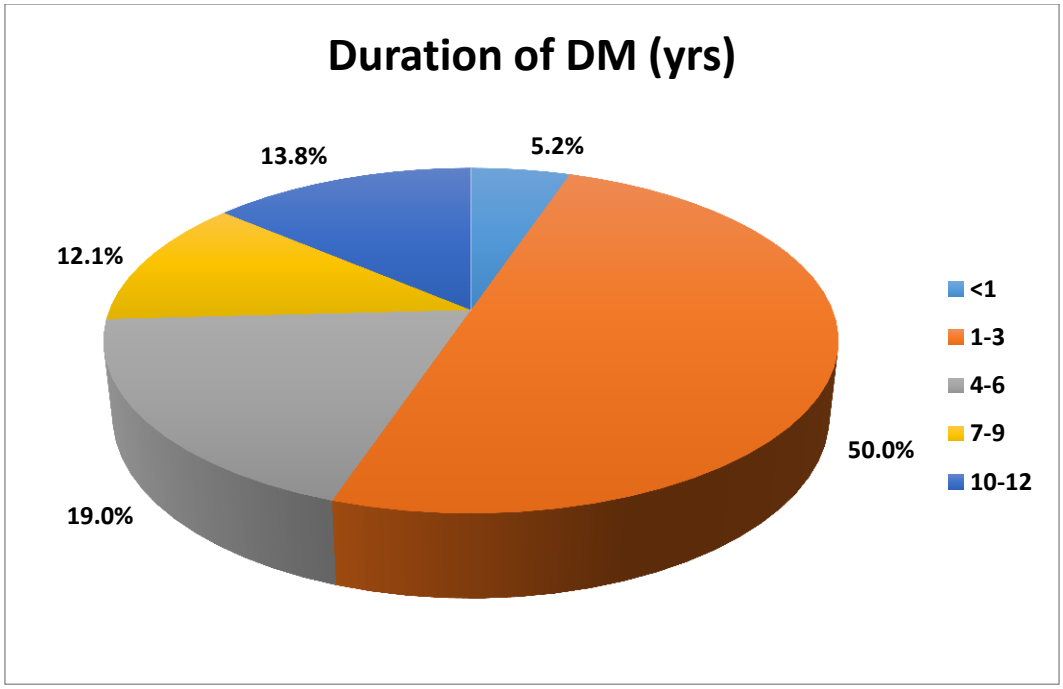


In this study, in diabetic group 26 (44.8%) were males and 32 (55.2%) were females. Among non-diabetic 19 (32.8%) were males and 39 (67.2%) were females

TABLE 4 : DISTRIBUTION OF CASES ACCORDING TO DURATION OF DM

DURATION OF DM (YRS)	N	%
<1	3	5.2
1-3	29	50
4-6	11	19
7-9	7	12.1
10-12	8	13.8
Total	58	100

FIGURE 4 : DISTRIBUTION OF CASES ACCORDING TO DURATION OF DM

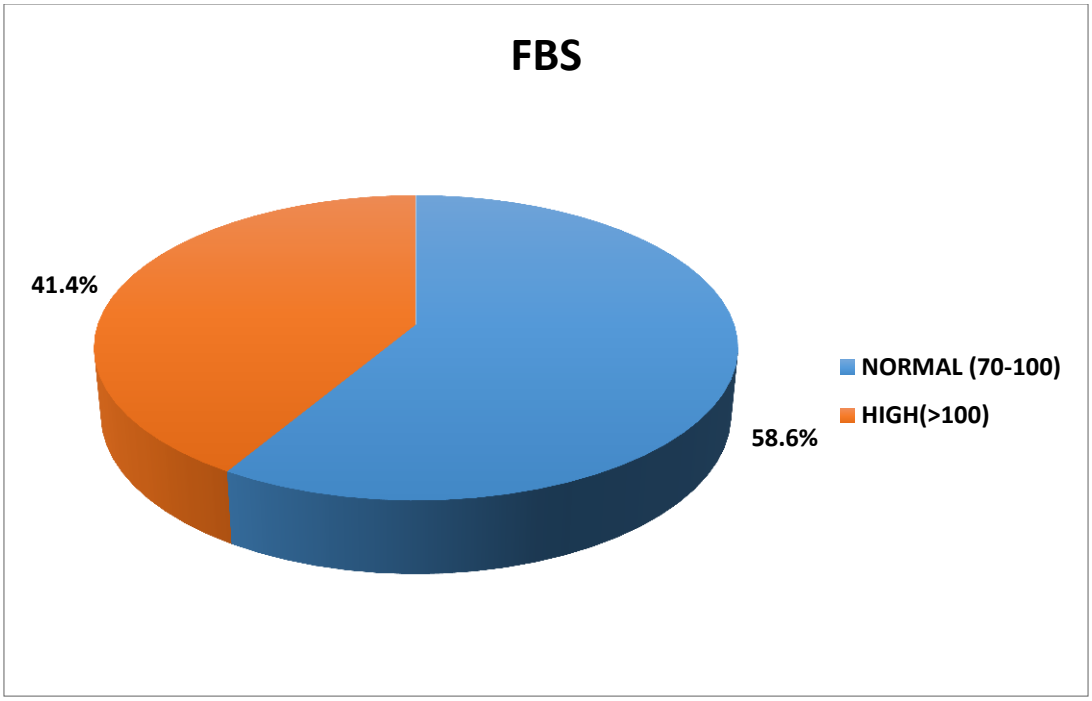


Majority of patients 32 (55.2%) were recently diagnosed diabetics with duration of disease being less than 3 years. There were about 8 (13.8%) patients with duration of disease being more than 10 years

TABLE 5 : DISTRIBUTION OF CASES ACCORDING TO FBS

FBS	N	%
NORMAL (70-100)	34	58.6
HIGH(>100)	24	41.4
Total	58	100

FIGURE 5 : DISTRIBUTION OF CASES ACCORDING TO FBS

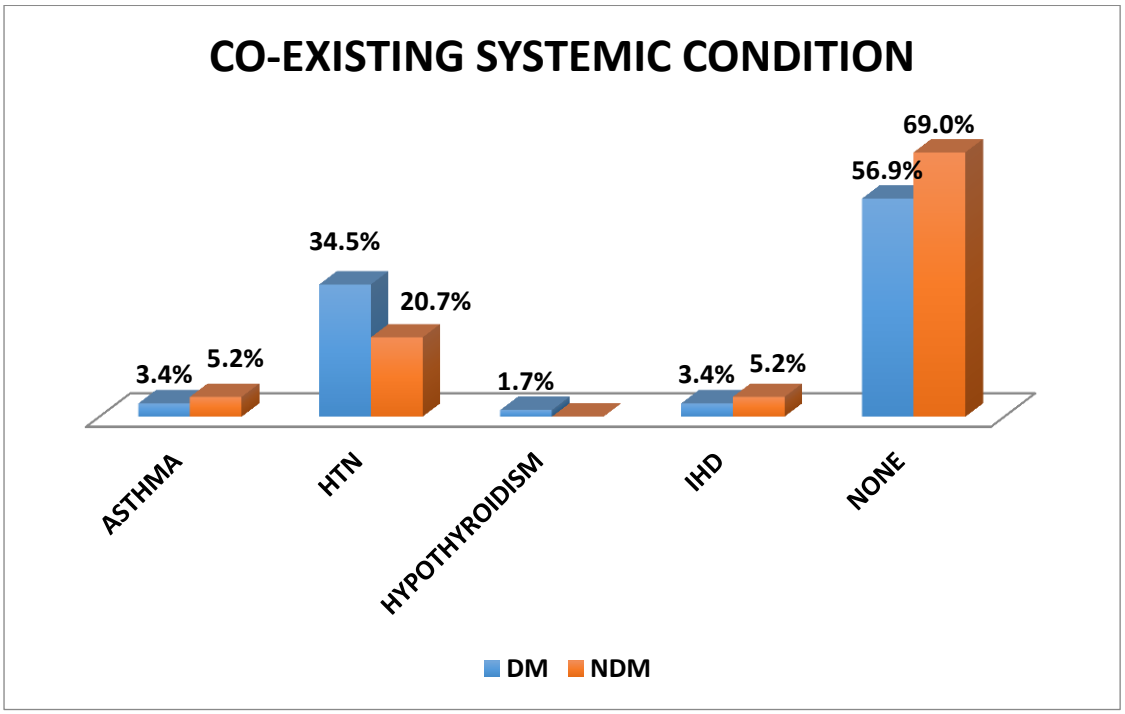


Of 58 patients in the diabetic group, 34 (58.6%) had good glycaemic control (FBS:70-100mg/dl). Remaining 24 (41.4%) patients had high blood sugar levels (>100mg). Their blood sugar levels controlled eventually and they were operated

TABLE 6 : DISTRIBUTION OF CASES ACCORDING TO CO-EXISTING SYSTEMIC CONDITION

CO-EXISTING SYSTEMIC CONDITION	DM		NDM		p value
	N	%	N	%	
ASTHMA	2	3.4%	3	5.2%	0.396
HTN	20	34.5%	12	20.7%	
HYPOTHYROIDISM	1	1.7%	0	0.0%	
IHD	2	3.4%	3	5.2%	
NONE	33	56.9%	40	69.0%	
Total	58	100.0%	58	100.0%	

FIGURE 6 : DISTRIBUTION OF CASES ACCORDING TO CO-EXISTING SYSTEMIC CONDITION

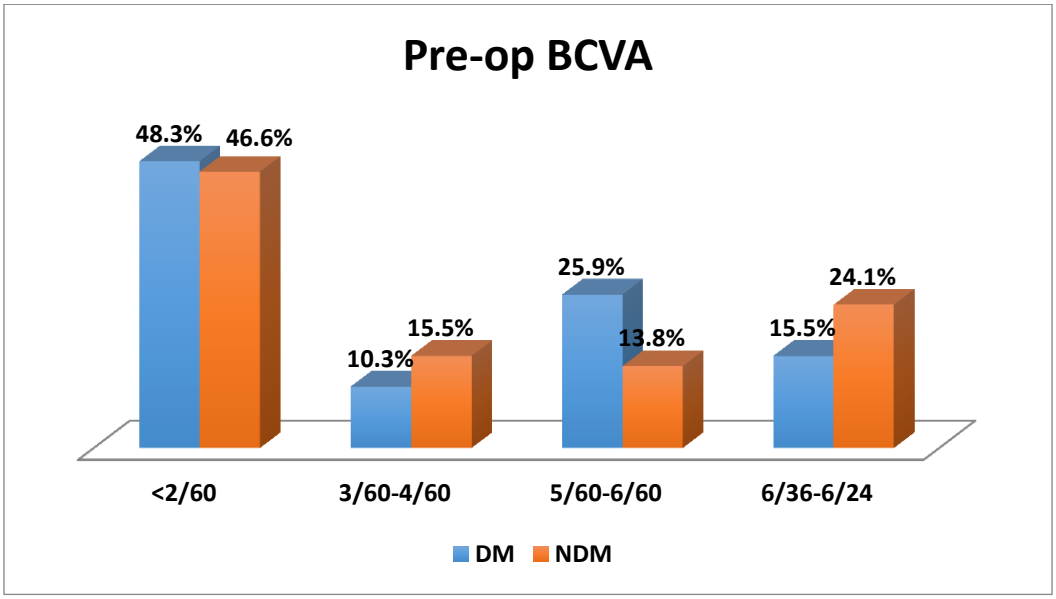


Systemic hypertension though the most frequent co-morbid disease in both the groups, it was more frequent among diabetics as seen in this study, that is 20 (34.5%) compared with 12 (20.7%) of the non-diabetic counter parts. The other co-morbid diseases were asthma which was seen in 2(3.4%) diabetic and 3(5.2%) non-diabetic patients, hypothyroid which was seen in 1(1.7%) diabetic patients and ischemic heart disease which was seen in 2(3.4%) and 3(5.2%) non-diabetic patients.

TABLE 7 : DISTRIBUTION OF PRE-OP BCVA BETWEEN STUDY GROUPS

PRE-OP BCVA	DM		NDM		p value
	N	%	N	%	
<2/60	28	48.3%	27	46.6%	0.28
3/60-4/60	6	10.3%	9	15.5%	
5/60-6/60	15	25.9%	8	13.8%	
6/36-6/24	9	15.5%	14	24.1%	
Total	58	100.0%	58	100.0%	

FIGURE 7 : DISTRIBUTION OF PRE-OP BCVA BETWEEN STUDY GROUPS

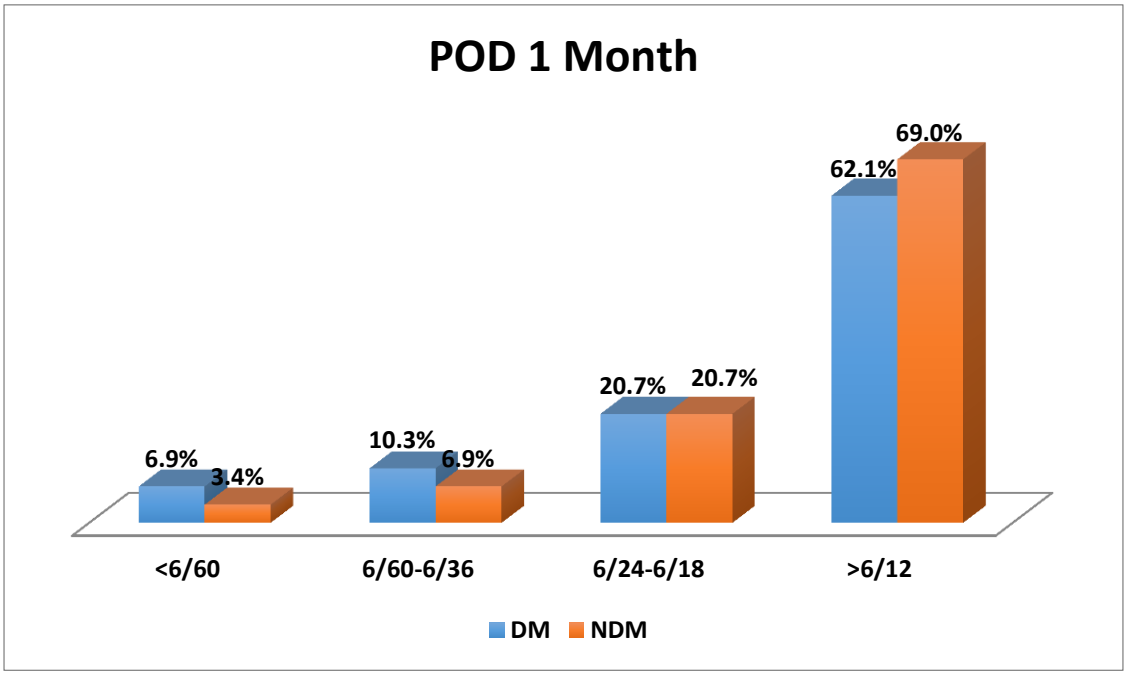


28 (48.3%) in the diabetic group and 27 (46.6%) in the non-diabetic group were having vision of less than counting fingers 2 meters (<2/60). Majority of the patients 49 (84.5%) in diabetic group and 44 (75.9%) in non-diabetic group were having vision of less than 6/60. Only 9 (15.5%) in diabetics and 14(24.1%) in non-diabetics had vision of 6/36-6/24(measured by snellen visual acuity chart). The mean best corrected pre-operative visual acuity in both the groups was calculated in logMAR units. The mean preoperative best corrected visual acuity in the diabetic group was 1.60±0.81 and in non-diabetic group was 1.62±0.87. The p value (>0.05) was not statistically significant

TABLE 8 : DISTRIBUTION OF FINAL VISUAL OUTCOME (POD-1 MONTH) BETWEEN STUDY GROUPS

POD 1 Month	DM		NDM		p value
	N	%	N	%	
<6/60	4	6.9%	2	3.4%	0.735
6/60-6/36	6	10.3%	4	6.9%	
6/24-6/18	12	20.7%	12	20.7%	
>6/12	36	62.1%	40	69.0%	
Total	58	100.0%	58	100.0%	

FIGURE 8 : DISTRIBUTION OF FINAL VISUAL OUTCOME (POD 1 MONTH) BETWEEN STUDY GROUPS

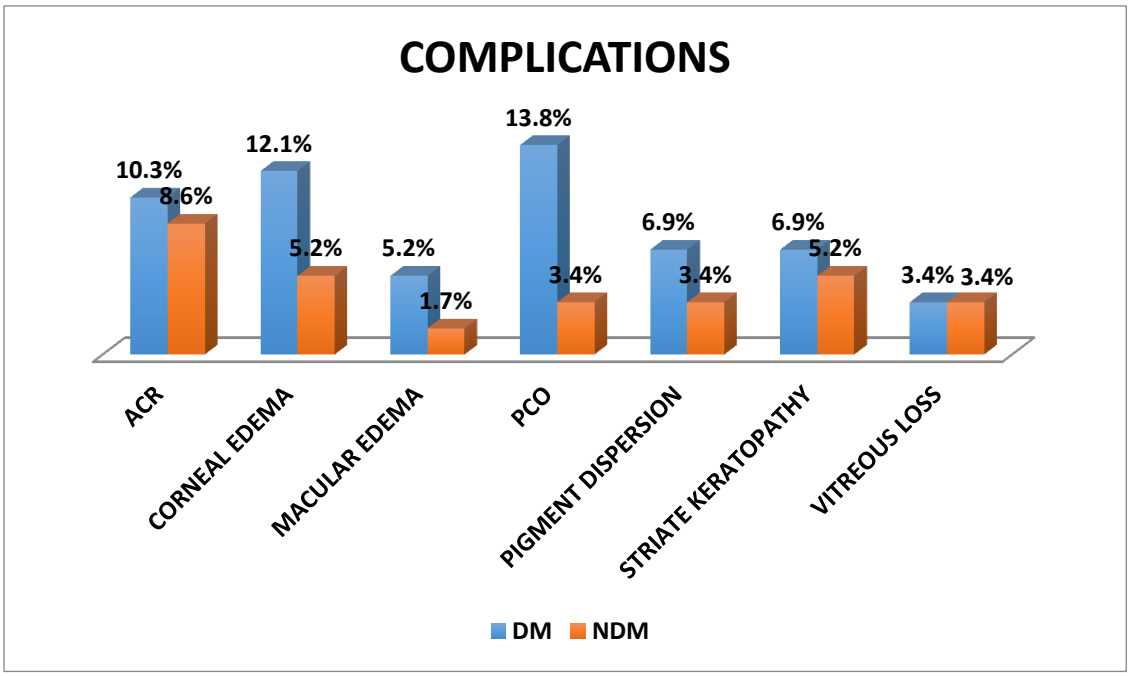


The final visual outcome was recorded using snellen's visual acuity chart and the values were converted to log MAR units for statistical analysis. Majority of the patients 36 (62.1%) in the diabetic group and 40 (69%) in the non-diabetic group had visual acuity of 6/12 or better at the end of 4 weeks of follow up. Only 4 patients in the diabetic group and 2 patients in the non- diabetic group had visual acuity less than 6/60. The mean post-operative best corrected visual acuity in log MAR units in the diabetic group was 0.39±0.32 and in the non-diabetic group was 0.32±0.27 on comparing the post op values in both the groups the p value was (0.23) which was not statistically significant. On comparing the pre-operative and post-operative visual acuity in both the groups the p value (<0.001) was statistically significant

TABLE 9 : DISTRIBUTION OF COMPLICATIONS BETWEEN STUDY GROUPS

COMPLICATIONS	DM		NDM	
	N	%	N	%
ACR	6	10.3%	5	8.6%
CORNEAL EDEMA	7	12.1%	3	5.2%
CYSTOID MACULAR EDEMA	3	5.2%	1	1.7%
PCO	8	13.8%	2	3.4%
PIGMENT DISPERSION	4	6.9%	2	3.4%
STRIATE KERATOPATHY	4	6.9%	3	5.2%
VITREOUS LOSS	2	3.4%	2	3.4%

FIGURE 9 : DISTRIBUTION OF COMPLICATIONS BETWEEN STUDY GROUPS



In this study the development of PCO at the end of 4 weeks in diabetics was 8 eyes (13.8%) compared to 2 eye (3.4%) in non-diabetics, which was higher in diabetics compared to non- diabetics. Corneal edema was found in 7 (12.1%) and 3 (5.2%) of the cases in diabetic and non diabetic groups respectively. In this study, total 6 (10.3%) eyes in the diabetic group and 5(8.6%) eyes in the non-diabetic group had anterior chamber reaction Striate keratopathy was found in 4 (6.9%) of the diabetics compared to 3 (5.2%) in nondiabetics. Pigments over IOL were seen in 4 (6.9%) of the cases in diabetics as compared to 2 (3.4%) in the control group. Cystoid macular

edema was seen in 3 (5.2%) diabetic and 1 (1.7%) non-diabetic patients. Vitreous loss was found in 2 (3.4%) in both diabetic and non-diabetic patients. Out of 59 diabetics complications were seen in 34 (58.6%) patients as compared to 18(30.9%) non diabetics.

DISCUSSION

The study includes cases consisting of 58 diabetic & 58 non diabetic patients (control), who underwent cataract surgery at B.L.D.E. (Deemed To Be University) in Ophthalmology department from December 2017- July 2019. Their visual outcome was compared.

In this study, highest number of patients were in the age group of 51-60 years that is in diabetic 26 (44.8%) & 32 (55.2%) in non-diabetic group. In this study patients below 40 and above 65 years of age were excluded. The mean age group of the patients in diabetic group was 56.5±7.4 and 59.6±5.2 years in control group.

Framingham and other eye studies indicate a 3-4 fold increased prevalence of cataract in patients with diabetes under 65 years and up-to a two-fold excess prevalence in patients above 65 years In this study, in diabetic group 32 (55.2%) were females and 26 (44.8%) were males. Among the non-diabetics, 19 (32.8%) were males & 39 (67.2%) were females. Various studies have proven the prevalence of cataract itself is more common in females than males. In the Framingham eye study also senile lens changes were more common in women. Age related cataract is a bilateral condition, one eye affected earlier than the other.

In these 58 patients in the diabetic group, we assessed the preoperative glycemic control. Patients with extremely high blood sugars and uncontrolled diabetes were excluded from the study.

Glycemic control was assessed using fasting blood sugar levels at the time of admission. Of the 58 patients in the diabetic group, 24 (41.4%) had high blood glucose level (FBS: >100mg/dl).

Their blood sugar was controlled and they were operated. 34 (58.6%) patients had normal blood sugar levels at the time of examination (70-100mg/dl). All 58

diabetic patients were on treatment for type 2 diabetes mellitus with either injection insulin or oral hypo-glycemic agents. Poor control of blood sugar level is associated with increased risk of retinopathy progression, inflammation and infections. Throughout the study it was seen that patients maintain an adequate blood sugar level by giving them insulin therapy whenever necessary.

We also looked at the duration of disease among our diabetic group. Majority of patients 32 (55.2%) were recently diagnosed diabetics with duration of disease being less than 3 years. There were about 8 (13.8%) patients with duration of disease being more than 10 years The risk for cataract formation and diabetic retinopathy is more in patients with longer duration of diabetes and in those with poor metabolic control. The pathways by which hyperglycemia leads to cataract are still uncertain but they are probably due to modification of the lens proteins leading to advanced glycation end products (AGEs) formation or modification of the ATPase pumps, leading to osmotic stress, or both.

Hypertension though the most frequent co-morbid disease in both the groups, its more frequent amongst diabetics as seen in this study, that is 20 (34.5%) compared with 12(20.7%) of the non- diabetic patients. A similar high incidence was seen in study by Onakpoya H Oluwatoyin et al[115], in which hypertension was seen in 60.9% compared with 26.1% in non-diabetic group.

In other studies by Squirrel et al[76], in which 55% had hypertension, Mittra et al, in which 68% had hypertension. By this it can be said that hypertension is a usually accompanying disease along with diabetes mellitus.

The other systemic co-morbidities in our study were ischemic heart disease in 2 (3.4%) in diabetics and 3 (5.2%) in the non-diabetic group. Asthma another

comorbid disease seen in 2 (3.2%) diabetic and in 3 (5.2%) non-diabetic patients. Hypothyroid was another comorbid disease which was seen in 1 diabetic patient.

In this study, majority of the patients had poor pre-operative visual acuity. 28 eyes (48.3%) of the diabetics and 27 eyes (46.6%) of the control patients had vision between counting fingers <2meters (<2/60). Majority of the patients 49 (84.5%) in diabetic group and 44 (75.9%) in non- diabetic group were having vision of less than 6/60. Only 9 (15.5%) in diabetics and 14(24.1%) in non diabetics had vision of 6/36-6/24(measured by snellen's visual acuity chart).The mean best corrected pre-operative visual acuity in both the groups was calculated in logMAR units.

The mean preoperative best corrected visual acuity in the diabetic group was 1.60±0.81 and that in non-diabetic group was 1.62±0.87.

In this study all the patients in both the groups had various grades of immature cataract. In our study, we found that the prevalence of mixed cataract was higher, more than two times of monotype cataract. In the mixed types, the most common type was a combination of nuclear cataract(NC), cortical cataract (CC), and posterior sub-capsular cataract (PSC), followed by the combination of CC and PSC. In the monotype cataracts, the most common cataract CC followed by NC and PSC. The methodology adopted was that of Lens Opacification Classification System (LOCS) III grading.

The prevalence of any cataract ranges from 35% to 48% in the general population; however, in subjects with diabetes, it is higher. The prevalence of NC is around 32% in the general population, but it is lower in subjects with diabetes. PSC is found in 14% of the general population.

All patients underwent cataract extraction by SICS with PCIOL implantation, and all the procedures were done by the same surgeon. On examination of the patients

on post-operative day one, corneal edema was found in 7 (12.1%) and 3 (5.2%) of the cases in diabetic and non-diabetic groups respectively which was considerably higher in diabetics compared to non-diabetics. Striate keratopathy was found in 4 (6.9%) of the diabetics compared to 3 (5.2%) in nondiabetics.

Larsson et al[116] have shown that diabetes has been associated with structural changes in corneal endothelial cells such as polymegathism and pleomorphism. The cornea has been reported to be thicker in eyes of diabetic patients than in eyes of non-diabetic subjects. Cataract extraction and IOL implantation causes trauma to the already compromised corneal endothelium and causes corneal edema.

Lee JS et al[109] showed a decrease in corneal endothelial cell density and the coefficient of variation by cell size significantly increased for high risk proliferative diabetic retinopathy.

Study by Morikuba S et al[117] has shown increase in the corneal thickness was greater on post-op day one among diabetic patients. It also showed that corneal endothelial loss was maximal at 1st week after operation.

It suggested that the corneal endothelium in diabetic patients is under metabolic stress, and weakness against mechanical loads such as cataract surgery than that in non-diabetic patients.

Hence compared with non-diabetic patients, eyes of diabetic patients showed more damage in corneal endothelial cells after cataract surgery and a delay in the post-operative recovery of corneal edema.

In this study the development of PCO in diabetics was 8 (13.8%) compared to 2 (3.4%) in non- diabetics, at the end of 4 weeks, confirming the finding of increase in incidence of PCO in diabetics as shown in previous studies.

Study by Ebihara Y et al[82] also showed significant increase in PCO in diabetic compared to non- diabetic patients.

A study by Hyashi K et al. also showed significant increase in PCO in diabetics after cataract extraction compared to nondiabetics.

Proliferation of Lens epithelial cells (LECs) causes PCO. LECs proliferation is affected by many factors like optic-haptic Junction, optic edge design and IOL material. Postoperative inflammation is an another important determinant. Residual LECs are stimulated by surgical trauma and contact with IOL and produces cytokines. LECs affected by these cytokines and leads to collagen formation and fibrous metaplasia. Thus, the degree of postoperative inflammation may be related to the development of PCO.

Diabetic patients have high incidence of PCO after cataract surgery than non-diabetic patients. PCO interferes with postoperative evaluation of the retina, PRP and even vitreous surgery, which is necessary in some cases. Therefore, it is important to maintain posterior capsule transparency in patients with diabetes.

Pigments over IOL were seen in 4 (6.9%) of the cases in diabetics as compared to 2 (3.4%) in the Non-diabetic group. Previous studies it has been shown that, there is increased pigment dispersion in diabetic patients undergoing cataract extraction and IOL implantation. This may be comparable with: Onakpoya H Oluwatoyin et al[115]showed increase amount of pigment dispersion occurring in diabetic patients i.e 6 in diabetics and 1 in non-diabetic patient.

In this study, total 6 (10.3%) eyes in the diabetic group and 5 (8.6%) eyes in the non-diabetic group had anterior chamber reaction. Diabetic patients had more anterior chamber reaction compared to non-diabetics. Similar observations were made in following studies “Onakpoya H Oluwatoyin et al[115], N D George et al and Mechini

et al reported intraocular inflammation and its sequelae as the most common complication of their study".

Ivancic et al reported inflammatory reaction fibrinous uveitis & PCO as the most common complications of cataract surgery among diabetics.

Longer duration of surgery is associated with increased post-operative inflammation. Fibrinous exudates & posterior synaechiae was not found in our study compared to previous study. None of the patients in our study had anterior segment neovascularization, as reported in previous studies.

In this study cystoid macular edema is seen in 3 (5.2%) diabetic and 1 (1.7%) non-diabetic patients. In diabetics with or without evidence of diabetic retinopathy the blood-aqueous barrier is impaired, it leads to inflammation and development of a cystoid macular edema, a process that is exacerbated by cataract surgery. Duration of surgery, wound size and posterior capsular rupture or vitreous loss are the few factors which influences the incidence of cystoid macular edema. Medicare beneficiaries analysis done from 1997 to 2001 showed that rate of development of cystoid macular edema was higher in diabetic patients after cataract surgery.

2 cases of vitreous loss was seen in both diabetic and non-diabetic in this study.

The final visual outcome was recorded using snellen's visual acuity chart and the values were converted to logMAR units for statistical analysis. Majority of the patients 36 (62.1%) in the diabetic group and 40 (69%) in the non-diabetic group had visual acuity of 6/12 or better at the end of 4 weeks of follow up. Only 4 patients in the diabetic group and 2 patients in the non diabetic group had visual acuity less than 6/60. The mean post-operative best corrected visual acuity in log MAR units in the

diabetic group was 0.39±0.32 and in the non-diabetic group was 0.32±0.27 on comparing the post op values in both the groups the p value was (0.23) which was not statistically significant. On comparing the pre-operative and post-operative visual acuity in both the groups the p value (<0.001) was statistically significant indicating that both the groups had good visual outcomes following surgery. This indicate that cataract surgery in diabetics without retinopathy led to favorable and comparable visual outcomes to that of non-diabetics.

CONCLUSION

This is a comparative study of visual outcomes following small incision cataract surgery with posterior chamber intraocular lens implantation in diabetics and non-diabetics. There were 58 eyes each in the diabetic and non-diabetic group. The pre-operative best corrected visual acuity was compared to the post-operative best corrected visual acuity in both the groups and the P value was statistically significant (p=0.01). The post-operative complications that were observed during the period of this study were posterior capsular opacification, corneal edema, striate keratopathy, anterior chamber reaction, pigment dispersion over IOL, cystoid macular edema and vitreous loss. These were significantly more in the diabetic group when compared to the non- diabetics. None of them were visually disabling and resolved during the course of follow up without any surgical intervention. Therefore, we concluded that small incision cataract surgery in diabetics without diabetic retinopathy yields similar visual outcomes as non-diabetics. There is a higher incidence of post-operative complications among diabetics which can be conservatively managed. The drawbacks of this study were small sample size and shorter duration of follow up.

SUMMARY

This is a comparative study of visual outcomes following small incision cataract surgery with Posterior chamber intraocular lens implantation in diabetics and non-diabetics. Overall 116 patients were included in the study, 58 each in the diabetic and non-diabetic group. For all patients a detailed history was taken with demographic details. A thorough general and ophthalmic evaluation was done which included anterior and posterior segment examination and accurate biometry. A duly signed consent was taken from all patients after explaining the procedure and visual prognosis in detail. All patients underwent small incision cataract surgery at

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_____ in Ophthalmology department. Follow up duration was 4 weeks. Of the 116 patients (58 diabetics and 59 non-diabetics), of which 71 were females and 45 males.

The mean age group of the patients in diabetic group was 56.6.36 ± 7.4 years and 59.6 ± 5.2 years in non-diabetic group. In this study all 58 diabetic patients were on treatment and 58.6% patients had good glycemic control prior to surgery. Mean preoperative best corrected visual acuity in the diabetic group was1.60±0.81 and that in the non-diabetic group was1.62±0.87. Mean post-operative best corrected visual acuity in logMAR units in the diabetic group was 0.39±0.32 and in the non-diabetic group was 0.32±0.27. The difference in pre and post op visual outcome was statistically significant (p=0.001). Post-operative visual acuity of 6/12 or better was achieved in 62.1% eyes in diabetics and 69% among non-diabetics.

Post-operative complications included: posterior capsular opacification, corneal edema, anterior chamber reaction striate keratopathy, pigment dispersion, cystoid macular edema and vitreous loss. The incidence was higher in the diabetic

group. None of the complications were visually disabling and were managed conservatively during the course of follow up.

There was no statistically significant difference in the final visual outcome between the diabetic and non-diabetic group. In conclusion, small incision cataract surgery in diabetics offer favorable and comparable visual outcomes with non-diabetic group, though incidence of post-operative complications remains high among diabetics.

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ANNEXURE – I

ETHICAL CLEARANCE CERTIFICATE

ANNEXURE – II

INFORMED CONSENT FORM

TITLE OF THE PROJECT	:	**COMPARATIVE STUDY OF OUTCOME OF CATARACT SURGERY IN DIABETICS AND NON DIABETICS**
PG GUIDE	:	
PRINCIPAL INVESTIGATOR	:	

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study will help to find and document the visual outcome and intra and post-operative complications of cataract surgery in diabetics

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study to **Dr. VALLABHA. K** in the Department of Ophthalmology who will be available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that _____ may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have been explained about the purpose of this research, the procedures required and the possible risks and benefits, in my own language.

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

Patient's Signature: Witness Signature

Name: Name:

Date: Date:

(Guide) (Investigator)

ANNEXURE – III

PROFORMA

DEPARTMENT OF OPHTHALMOLOGY

Case No : OPD/IPD No: Date :

Name: Age: Sex:

Occupation: Address:

Chief complains:

History of presenting illness: : Patient was apparently alright _____earlier then

H/o diminution of vision Yes/ No

H/o pain Yes/ No

H/o redness Yes/No

H/o watering Yes/No

H/o photophobia Yes/No

H/o foreign body sensation Yes/No

Other complaints:

Past history:

Personal history:

Type of diabetes:

Duration of diabetes:

Treatment history:

GENERAL PHYSICAL EXAMINATION

Pallor:

Icterus: BP:

Cyanosis: PR:

Clubbing:

Edema:

OCULAR EXAMINATION **RE** **LE**

EXTERNAL APPEARANCE

OCULAR MOTILITY

EYELIDS

CONJUNCTIVA

CORNEA

A/C

IRIS

PUPIL

LENS

PRE-OP VISION

Distant Vision

Near Vision

TENSION (By AT/NCT)

SAC

Slit lamp examination

BIOMETRY:-

K1

K2

AXL

IOL

FUNDUS:-

Media-

Disc-

Blood vessel-

Background-

Macula-

LAB INVESTIGATIONS

RBS

FBS-

PPBS-

HIV-

HbsAg-

DIAGNOSIS:-

TREATMENT GIVEN:

OPERATIVE COMPLICATIONS:

POST OP FOLLOW UP- DAY 1

	RE	**LE**
EXTERNAL APPEARANCE		
OCULAR MOTILITY		
EYELIDS		
CONJUNCTIVA		
CORNEA		
A/C		
IRIS		
PUPIL		
LENS		

POST-OP VISION:

Distant Vision

Near Vision

FUNDUS-

Media-

Disc-

Blood vessel-

Background-

Macula-

ONE WEEK:

	RE	**LE**
EXTERNAL APPEARANCE		
OCULAR MOTILITY		
EYELIDS		
CONJUNCTIVA		
CORNEA		
A/C		
IRIS		
PUPIL		

LENS

POST-OP VISION:

Distant Vision

Near Vision

FUNDUS –

Media-

Disc-

Blood vessel-

Background-

Macula-

ONE MONTH:

	RE	**LE**
EXTERNAL APPEARANCE		
OCULAR MOTILITY		
EYELIDS		
CONJUNCTIVA		
CORNEA		

A/C

IRIS

PUPIL

LENS

POST-OP VISION:

Distant Vision

Near Vision

FUNDUS –

Media-

Disc-

Blood vessel-

Background-

Macula-

A-SCAN MACHINE

BIOMETER


ECHORULE
Ultrasonic Biometer
0 1 2 3 Lens
+- 4 5 6 Print
. 7 8 9 Enter Menu
VISION CARE / Phakosystems Inc.
3M
Auto Biometric Mode
Manual Biometric Mode
Diagnostic Mode
SRK I (tm) Calculations
SRK II (tm) Calculations
Binkhorst IOL Calculation

SLIT-LAMP EXAMINATION

NON CONTACT TONOMETRY

KEY WORDS TO MASTERCHART

FBS	Fasting blood sugar level
DM	Diabetes mellitus
Y/N	Yes/No
Pre-op	Pre-operative
POD	Post-operative day
BCVA	Best corrected visual acuity
ACR	Anterior chamber reaction
PCO	Posterior capsular opacification
IMC	Immature cataract
SMC	Senile mature cataract
SIMC	Senile immature cataract
CF	Counting finger
MTR	Meter
PL	Perception of light
HM	Hand movement
HTN	Hypertension
IHD	Ischemic heart disease

MASTER CHART

Sl No	Name	IP No	Age	Sex	DM(Y/N), Duration	Medication	FBS	Co-existing systemic condition	Diagnosis	Pre-op BCVA	POD1	POD 1 Week	POD 1 Month	Complications
1	VITAL SIDRAM BISHI	42659	45	M	Y, 1yr	YES	98	HTN	RE IMC	CF 1MTR	6/12	6/9	6/9	
2	MANAKABAI PANDURANG	41755	60	F	Y, 3yr	YES	120		RE SIMC	CF 2MTR	6/24	6/18	6/18	CORNEAL EDEMA
3	BASAMMA MANAGULI	34103	52	F	Y, 1yr	YES	76		RE SIMC	6/60	6/18	6/12p	6/12	
4	SATAMMA PATIL	32106	55	F	Y, 2yr	YES	102	HTN	RE SIMC	CF 5MTR	6/18	6/12	6/12	PCO
5	BHIMAWWA RUDRAPPA HALLI	12251	45	F	Y, 1yr	YES	98		RE MATURE CATARACT	HM	6/12	6/6p	6/6p	
6	LAXMIBAI MALLIKARJUN	31244	60	F	Y, 2yr	YES	110	HTN	LE SIMC	6/60	6/12p	6/12	6/12	
7	PARVATI SHIVAPPA	29356	50	F	Y, 3yr	YES	88	HTN	RE SIMC	CF 3MTR	6/12	6/9p	6/9	
8	HANAMAWA MADAR	26652	62	F	Y, 4yr	YES	102		RE SIMC	CF 1MTR	6/18p	6/18	6/18	PCO
9	ASIF KHAN	24278	60	M	Y, 5yr	YES	94	HTN	LE SIMC	6/36	6/9	6/6p	6/6p	
10	SANTOSH PATIL	20618	45	M	Y, 1yr	YES	98		RE IMC	6/60	6/18	6/12	6/12	CORNEAL EDEMA, ACR
11	MAHADEV MADHAV	19274	48	M	Y, 1yr	YES	110	HTN	LE IMC	CF 1MTR	6/12p	6/9p	6/9p	
12	CHANDRAWA TULARAM	16520	50	F	Y, 2yr	YES	100		RE SIMC	6/24	6/12	6/9	6/9	
13	CHANDRAKALA DEVENDRA	6206	40	F	Y, 1yr	YES	110		LE IMC	CF 4MTR	6/12p	6/9p	6/9p	
14	NAGAPPA INDI	17097	59	M	Y, 3yr	YES	108	HTN	LE SIMC	6/24	6/9	6/6p	6/6p	
15	LAXMIBAI DEVAPPA	912	62	F	Y, 2yr	YES	90		RE SIMC	CF 1FEET	6/24p	6/24	6/24	CORNEAL EDEMA
16	LAXMAN SHIVAPPA	42023	60	M	Y, 3yr	YES	110	HTN	RE SIMC	CF 5MTR	6/12p	6/9p	6/9p	
17	BOURAWWA BIRADAR	9915	52	F	Y, 1yr	YES	82		LE SIMC	CF 2MTR	6/12p	6/12	6/12	ACR
18	PARASAPPA RAMAPPA	44495	45	M	Y, 1yr	YES	80		LE IMC	6/60	6/12	6/9	6/9	
19	KALLAWWA UPPAR	6903	61	F	Y, 2yr	YES	88		RE SMC	HM	6/24	6/18	6/18	CORNEAL EDEMA
20	IBRAHIMSAB BAGALI	12589	63	F	Y, 5yr	YES	70	ASTHMA	RE SMC	PL +VE	CF 5MTR	6/60	6/60	STRIATE KERATOPATHY
21	AMINABI KOPPA	11817	65	F	Y, 4yr	YES	111	HTN	RE SIMC	6/36	6/9p	6/9	6/9	
22	TARASINGH HIRU	11205	62	M	Y, 3yr	YES	80		LE SMC	HM	6/60	6/36	6/36	STRIATE KERATOPATHY
23	BANGARAWWA HABAGONDE	14976	58	F	Y, 1yr	YES	104		LE SIMC	CF 5MTR	6/18	6/12p	6/12	
24	KAMALA BAGEWADI	5680	65	F	Y, 7yr	YES	110	HTN	LE SIMC	CF-CLOSE TO FACE	CF 3MTR	6/60P	6/60	CORNEAL EDEMA
25	SUNITA JAIN	6156	40	F	Y, 6 months	YES	110	HYPOTHYROIDISM	RE IMC	6/36	6/9p	6/6p	6/6p	
26	RAJU OMBALE	5619	54	M	Y, 3 yr	YES	98		RE SIMC	6/60	6/18	6/12	6/12	PCO
27	KALAPPA MALKAPPA	7763	55	M	Y, 10 months	YES	80		LE SIMC	CF 1FEET	6/24	6/18	6/18	PIGMENT DISPERSION
28	KASTHURIBAI BABUGOUDA	10019	63	F	Y, 10yr	YES	90	HTN	LE SIMC	CF 5MTR	6/12p	6/9	6/9	
29	SONUBAI BADIGER	18581	60	F	Y, 8yr	YES	137	HTN	RE SMC	PL +VE	6/18	6/12	6/12	
30	BASAVANT JADHAV	17934	61	M	Y, 5yr	YES	110		RE SMC	HM	CF-CLOSE TO FACE	CF 3MTR	CF 5MTR	PIGMENT DISPERSION
31	SIDDAPPA NAIKODI	13355	65	M	Y, 6yr	YES	80		LE SMC	HM	6/60	6/36	6/36	MACULAR EDEMA

32	SANGAPPA KAJAGAR	16555	65	M	Y, 9yr	YES	90	IHD	LE SIMC	CF 1MTR	6/18	6/12p	6/12	
33	SIDRAMAPPA IRAPPA	17004	65	M	Y, 7yr	YES	100		LE SIMC	CF 3MTR	CF 1MTR	CF 4MTR	CF 5MTR	MACULAR EDEMA
34	SIDDANAGOUDA BALALASAHEB	9113	60	M	Y, 10yr	YES	68		LE SIMC	CF 1MTR	6/18	6/12p	6/12	PCO
35	YALLAPPA PARAPPA	7848	55	M	Y, 1yr	YES	90		LE SIMC	6/60	6/12	6/9	6/9	
36	BORAMMMA ASARAMPUR	7695	62	F	Y, 3yr	YES	112	HTN	LE SIMC	6/36	6/9p	6/6p	6/6p	
37	SIDDAYYA HIREMATH	6458	50	M	Y, 1yr	YES	80		RE IMC	CF 2MTR	6/18p	6/18	6/18	PCO, ACR
38	ALISAB WALIKAR	6194	58	M	Y, 4yr	YES	90		LE SIMC	6/24	6/12	6/9	6/9	
39	YALLAWWA BHIMAPPA	5671	60	F	Y, 10yr	YES	108	HTN	LE SIMC	6/60	6/18	6/12	6/12	
40	SANGAYYA RAMAYYA	3334	65	M	Y, 12yr	YES	98		LE SIMC	CF 2MTR	6/18p	6/18	6/18	STRIATE KERATOPATHY
41	BASAVA LALSAB	225	65	M	Y, 8yr	YES	80	ASTHMA	LE SIMC	CF 1FEET	CF 3MTR	CF 3MTR	CF 4MTR	VITREOUS LOSS
42	BANGAREWWA B BIRADAR	18753	59	F	Y, 6yr	YES	112	HTN	RE SIMC	6/36	6/9p	6/9	6/9	
43	MAHADEV HALASANGI	15843	54	M	Y, 2yr	YES	74		LE SIMC	CF 4MTR	6/24	6/18p	6/18p	PCO, MACULAR EDEMA
44	SHRISAIL ARAVATTU	15894	42	M	Y, 1yr	YES	106	HTN	RE IMC	6/36	6/9	6/9	6/9	
45	MADIVALAPA DARGA	16126	51	M	Y, 2yr	YES	88		LE SIMC	6/60	6/18	6/12	6/9p	
46	SIDDAMMA BIRADAR	16164	65	F	Y, 4yr	YES	118	IHD	LE SIMC	CF 1FEET	CF 2MTR	CF 3MTR	CF 3MTR	VITREOUS LOSS, ACR
47	LACHABAI RATHOD	16266	65	F	Y, 12yr	YES	114	HTN	LE SIMC	CF-CLOSE TO FACE	CF 3MTR	6/60	6/36	PIGMENT DISPERSION
48	MABUBI MAKANADAR	16001	60	F	Y, 3yr	YES	92		RE SIMC	CF 4MTR	6/12	6/9	6/9	
49	KAMALAMMA NAIKAL	16752	60	F	Y, 11yr	YES	108		RE SMC	HM	CF 5MTR	6/36p	6/36	PIGMENT DISPERSION
50	SAVITRI MALATRAV	16745	65	F	Y, 12yr	YES	98	HTN	LE SIMC	CF 1 1/2 MTR	6/24p	6/24	6/24	CORNEAL EDEMA
51	BANDENAWAZ NADAF	13767	40	M	Y, 11 months	YES	76		LE IMC	6/60	6/18	6/12	6/12	PCO
52	DANABAI RATHOD	33959	55	F	Y, 3yr	YES	120		RE SIMC	CF 3MTR	6/12p	6/9p	6/6p	
53	BHOJU CHANDU CHAVAN	14199	52	M	Y, 4yr	YES	82		RE SIMC	6/60	6/12p	6/9p	6/9p	ACR
54	SHANTABAI PUJARI	16673	62	F	Y, 10yr	YES	124	HTN	LE SMC	PL +VE	6/24p	6/24	6/24	STRIATE KERATOPATHY
55	LALABI CHAVAN	12571	60	F	Y, 7yr	YES	78		LE SIMC	6/60	6/9p	6/6p	6/6p	
56	BORAMMA GILDE	23194	45	F	Y, 4yr	YES	128		RE MATURE CATARACT	HM	6/18p	6/18	6/18	CORNEA EDEMA, ACR
57	NINGAPPA TALWAR	20954	54	M	Y, 8yr	YES	70		LE SMC	PL +VE	6/18	6/12	6/9p	
58	RATNABAI BABURAY	14986	60	F	Y, 10yr	YES	94	HTN	LE SMC	HM	6/24	6/18	6/18	PCO