"A ONE YEAR LONGITUDINAL STUDY TO ASSESS THE CORNEAL ENDOTHELIAL MORPHOLOGY AND CENTRAL CORNEAL THICKNESS IN DIABETIC PATIENTS AND NON-DIABETIC PATIENTS UNDERGOING MANUAL SMALL INCISION CATARACT SURGERY"

BY

REG NO: BK0122001

Dissertation

Submitted to

KAHER, Belagavi, Karnataka

In partial fulfilment
of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

DEPARTMENT OF OPHTHALMOLOGY,

J. N. MEDICAL COLLEGE
BELAGAVI - 590010. KARNATAKA

SEPTEMBER/OCTOBER 2025

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LIST OF ABBREVIATIONS

Sl. No	Abbreviation	Full Form
1	MSICS	Manual small incision cataract surgery
2	CCT	Central corneal thickness
3	ATP	Adenosine triphosphate
4	ECD	Endothelial cell density
5	GAG	Glycosaminoglycan
6	CV	Co-efficient of variance
7	AGE	Advanced glycation end products
8	DNA	Deoxy ribonucleic acid
9	ROS	Reactive oxygen species
10	IL	Interleukin
11	TNF	Tumour necrosis factor
12	IOL	Intraocular lens
13	T2DM	Type 2 Diabetes Mellitus
14	NPDR	Non-proliferative Diabetic retinopathy
15	OPD	Out patient department
16	ANOVA	Anlaysis of variance
17	Et al	Et alia (latin phrase for “and others”)
18	SD	Standard deviation

ABSTARCT

Title: A one year longitudinal study to assess the corneal endothelial morphology and central corneal thickness in diabetic patients and non-diabetic patients undergoing Manual small incision cataract surgery

Introduction: Understanding the impact of MSICS on corneal health, particularly in vulnerable populations such as diabetic patients, is critical for optimizing surgical outcomes and patient care.

Objectives:

1. To compare corneal endothelial cell changes in diabetics and non-diabetic patients who underwent manual small incision cataract surgery,
2. To correlate endothelial cell changes and central corneal thickness in diabetics and non-diabetic patients who underwent manual small incision cataract surgery.

Methodology: A hospital based prospective study was conducted among 35 diabetic patients and 35 non-diabetic controls who were planned for manual small incision cataract surgery. Through a non-contact specular microscope, corneal endothelial cell changes, co-efficient of variance, and central corneal thickness was assessed on preoperative day, followed by post-op day 1, 1 week, 1 month, and 3 month following surgery.

Results: Among diabetic patients, the central corneal thickness increased from pre-operative period (508.4+36.1), to day 1 (548.9+53.4), and subsequently reduced towards normal on one week (527.0+36.6), one month (536.8+38.9), and 3 month

(508.6+38.0). Similar results were noted among non-diabetics (pre-operative:495.1+30.9. 1 day post-op: 531.3+36.6, 1 week post-op: 512.9+24.0, 1 month post-op: 505.6+22.8, 3 month post-op: 495.2+27.7). While the cases were higher among diabetics, the difference was not statistically significant. The co-efficient of variation increased among diabetic group from 0.45+0.04 at pre-operative phase to 0.49+0.09 at 3 months post-operative phase. Similarly, among non-diabetic phase, it increased from 0.45+0.05 at pre-operative phase to 0.47+0.06 at 3 months post-operative phase.

Conclusion: While MSICS proved to be a safe and effective procedure for both diabetic and non-diabetic patients, diabetic corneas exhibited a non-significant delayed recovery and a slightly greater susceptibility to surgical trauma and stress.

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INTRODUCTION

Cataract is the leading cause of reversible blindness, and is the second most common cause of severe visual impairment worldwide.[1] Among population aged 50 years or above, cataract and refractive error constitutes for 55% of blindness and 77% of severe visual impairment worldwide.[1] Surgical management is the mainstay treatment for cataract.[2-3] Among the various techniques available for cataract removal, Manual Small Incision Cataract Surgery (MSICS) remains a popular and cost-effective option due to its affordability, short learning curve, shorter surgical time, and suitability for resource-limited settings.[4-5] In addition, smaller incisions are associated with reduced post-operative astigmatism and glaucoma.[4-5] Understanding the impact of MSICS on corneal health, particularly in vulnerable populations such as diabetic patients, is critical for optimizing surgical outcomes and patient care.[6]

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. An estimated 537 million worldwide suffer from diabetes mellitus and it is expected to increase to 783 million by 2045.[7] With an estimated prevalence of 77 million cases, India ranks second in diabetes mellitus prevalence worldwide.[8] It is of concern as their complications extend to majority of organs.[9-10] Cataract, posterior capsular opacification, diabetic retinopathy and diabetic macular oedema are the common ocular complications in diabetic patients.[11-12] Compared to non-diabetics, the incidence of cataract is 4 times higher among diabetics in younger population (<65 years) and it is two times higher in elderly population (>65 years).[11] Moreover, diabetes mellitus increases the risk of complications following cataract surgery.[12] This includes morphological and functional impairment at corneal endothelium.[13-14]

Hence, understanding the relationship between diabetes mellitus and cataract surgery at anatomical level is of utmost importance.

Cornea is an convex, aspheric, and avascular structural barrier of the eye.[15] They are thin at the centre (551 to 565 μ) and thick at the periphery (612 to 640 μ).[15] The corneal cellular components include epithelial cells (5-7 cell layers), keratocytes and endothelial cells (single layer).[15-16] The monolayer hexagonal corneal endothelium cells play a critical role in maintaining corneal transparency by regulating stromal hydration through an active pump-leak mechanism[15-16] While the endothelial cell density is approximately 3500 cells/mm^2 at birth, they progressively decrease at a rate of 0.6% per year in adulthood.[15-16] Moreover, corneal endothelial cells have limited regenerative capacity.[15-16] Hence, damage or change in these parameters are indicative of morphological or functional stress.[17-18]

The corneal and their endothelial parameters like central corneal thickness and endothelial cell density are particularly important in the context of diabetes mellitus and cataract surgery.[17-18] As diabetes mellitus is a hyperglycaemic state, it is believed to induce oxidative stress, glycation of endothelial proteins, impaired endothelial cell function and microvascular damage, which collectively compromise endothelial cell function.[19-20] Based on previous studies, it is hypothesized that these pathophysiological changes lead to reduced endothelial cell density, central corneal thickness, hexagonal cell ratio and co-efficiency of variance.[20-22] Increased central corneal thickness (CCT) in diabetic patients has been attributed to endothelial dysfunction and increased stromal hydration.[23-24] Moreover, cataract surgeries pose challenges to corneal endothelial health due to mechanical stress from surgical instruments, irrigation fluids, and intra-ocular lens implantation.[25-26] However, long term recovery of endothelial cell following cataract surgery is noted.[27-28] These are

postulated to be due to endothelial cell enlargement.[28] However, there are limited studies on the impact of diabetes mellitus on healing and recovery of endothelial cell following cataract surgeries. Understanding these changes in the context of diabetes is crucial for predicting surgical outcomes and planning follow-up care.

Despite the extensive literature on diabetes and cataract surgery, few studies have focused on the long-term impact of MSICS on corneal endothelial morphology and CCT in diabetic patients. Most studies are limited to short-term follow-ups or fail to include a comparative analysis with non-diabetic patients. This study aims to fill this gap by providing a comprehensive, year-long evaluation of these parameters.

This study aimed to conduct a one-year longitudinal study to assess the corneal endothelial morphology and CCT in diabetic and non-diabetic patients undergoing MSICS. The findings will contribute to the growing body of knowledge on surgical outcomes in diabetic populations and provide insights into postoperative care strategies.

AIMS AND OBJECTIVES

PRIMARY OBJECTIVE:

To compare corneal endothelial cell changes in diabetics and non-diabetic patients who underwent manual small incision cataract surgery

SECONDARY OBJECTIVE:

To correlate endothelial cell changes and central corneal thickness in diabetics and non-diabetic patients who underwent manual small incision cataract surgery

REVIEW OF LITERATURE

The review of literature will be discussed in following subheadings:

A. Cornea

B. Diabetes Mellitus and its effect on cornea

C. Cataract in diabetes mellitus

D. Manual incision cataract surgery

E. Effect of MSICS on cornea

F. Previous studies

G. Lacunae and need for study

A. Cornea

Overview

The cornea is a transparent, avascular structure integral to the optical and protective functions of the eye.[15] Its unique structural and biochemical properties allow it to serve as a refractive medium while maintaining clarity and mechanical strength.[15] Its anatomy and histopathology are foundational to understanding how systemic conditions like diabetes and surgical interventions such as Manual Small Incision Cataract Surgery (MSICS) impact corneal health.

Dimensions and Shape

Diameter: Horizontally 11–12 mm and vertically 9–11 mm.

Curvature: The anterior curvature is 7.8 mm and posterior curvature is about 6.5 mm. Cornea contributes to about 40–44 D of refractive power and accounts for approximately 70% of total refraction.[15]

Corneal Thickness

The **central corneal thickness (CCT)** in normal eyes is found to range from 551 to 565 μ and the peripheral corneal thickness from 612 to 640 μ.[15,30] The corneal thickness is influenced by age, hydration and systemic conditions. The gradual increase in thickness from the central to peripheral cornea is attributed to higher collagen density in the peripheral stroma and anterior stromal rigidity that stabilizes corneal curvature and resists changes in hydration.[15,30] The central corneal thickness (CCT plays) a critical role in maintaining corneal curvature for optimal refraction, and providing structural integrity to withstand intraocular pressure changes.[15,30] It also serves as a biomechanical indicator of endothelial pump function and stromal hydration.[15,30]

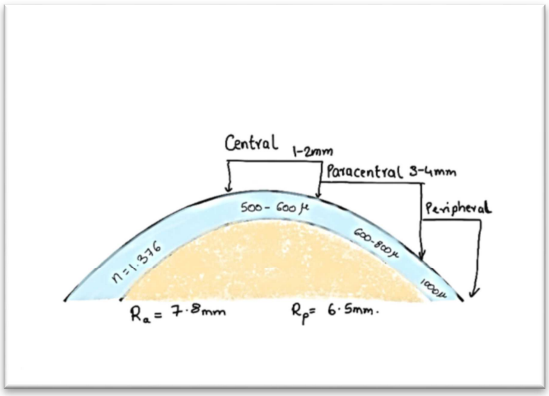


Figure No.1 : Diagrammatic representation of corneal thickness and anterior (Ra) and posterior (Rp) corneal curvature

The role of corneal thickness is critical in management of glaucoma, refractive surgeries (LASIK and photorefractive keratectomy) and corneal pathologies like subclinical keratoconus, Fuchs endothelial dystrophy and pellucid marginal degeneration.[15,30] Moreover, it is an important assessment parameter for cataract surgery and diabetes. Various techniques are used to measure central corneal

thickness. Ultrasound Pachymeter (US), Optical Coherence Tomography (OCT), Topographer (TOPO), Specular Microscope (MS), and Non-contact Tonometer (TONO) are few of the commonly used measures worldwide.[30]

Layers of the Cornea

The cornea is composed of six well-defined layers, each serving unique functional and structural roles:

i. Epithelium

They are made up of non-keratinized, stratified squamous epithelium composed of 5–7 cell layers.[15,31] It is about 50 μ in thickness.They act as a barrier against microbial, chemical, and mechanical insults.[15,31] It also provides a smooth optical surface for light refraction. The epithelial cells regenerate every 7–10 days, maintaining the integrity of the surface.[15,31]

ii. Bowman's Layer

Bowman's membrane is condensation of collagen and proteoglycans. It is a 12 μ structure and is made up of Type I and V collagen as well as proteoglycans. It has no regenerative ability.[15,31] Hence they are prone for scarring upon damage/injury.[15,31]

iii. Stroma

They account for 80–85% of corneal thickness. They are composed of highly organized collagen lamellae (Type I, V, VI) and glycosaminoglycans (GAGS).[15,31] They provide mechanical strength, transparency, and hydration regulation to the cornea.[15,31] Any dysregulation of stromal hydration or collagen arrangement can lead to loss of transparency (Example: corneal oedema).[15,31]

iv. Pre-Descement's membrane

This layer is also known as Dua's layer and was discovered in 2013 by Dr. Harminder Dua. It is an acellular structure located anterior to Descement's membrane and is 6 to 15 µ thick, strong and impervious to air. There are no keratocytes in this layer.[15]

v. Descemet's Membrane

They are the basement membrane for endothelial cells.[15,31] They are secreted throughout life and thicken with age. They provide elasticity and serve as a barrier and scaffold for endothelial cells.[15,31]

vi. Endothelium

They are single layer of metabolically active hexagonal cells that do not regenerate.[15,31] The apical regions of the cells are interconnected by tight junctions and gap junctions, accompanied by interdigitations that connect adjacent cells.[32-34]

Scanning electron microscopy highlights the presence of surface villi and membranous pores. These cells are rich in mitochondria, and hence have high metabolic activity.[32] The corneal endothelium demonstrates passive permeability, permitting the diffusion of large molecules across its barrier.[32] This includes nutrient and ion transport from aqueous humour to stroma of cornea.[33] Endothelial cells maintain corneal clarity by actively pumping excess water out of the stroma via Na+/K+ ATPase pumps and carbonic anhydrase pathways.[15,31] This transport is through pump leak mechanism.[33]

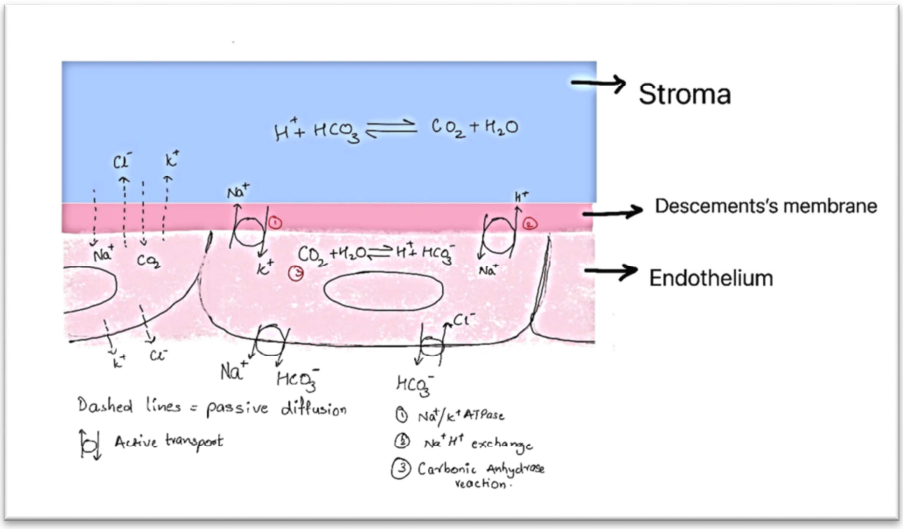


Figure No.2 : Pump leak mechanism of cornea

They maintain corneal transparency by regulating stromal hydration through active ion pumps. Loss of endothelial cell density, particularly in diabetics or post-surgery, can lead to corneal oedema and loss of transparency.[15,31]

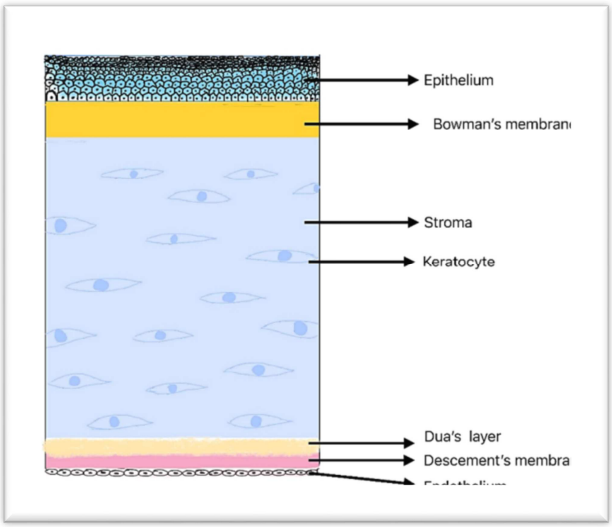


Figure No.3 : Description of layers of cornea

Endothelial cell density: The endothelial cell density (ECD) of the cornea varies with age and is crucial for maintaining corneal transparency.[15] The following points summarize the key aspects of endothelial cell density:

Average ECD Values:

- At birth: Approximately 3500–4000 cells/mm².[15,34]
- In adults: Declines to around 2600 cells/mm² by the eighth decade of life.[15,34]
- Age-Related Decline: The endothelial cell density decreases by about 0.6% per year throughout adulthood.[15,34] This decline is accompanied by an increase in polymegathism (variability in cell size) and pleomorphism (irregularity in cell shape).[15,34]
- A minimum critical ECD (400-500 cells/mm²) is required to maintain these functions.[34] If the ECD drops below this critical threshold, corneal oedema and decompensation may occur.[34]

Biomechanics of cornea

Mechanical Properties of the Cornea

i. Viscoelastic Behaviour: The cornea exhibits viscoelasticity, balancing immediate elastic responses with time-dependent viscous deformation.[35]

ii. Elasticity: Immediate resistance to deformation and critical for maintaining shape.[35]

iii. Viscosity: Dissipates energy under sustained or repetitive stress and hence reducing damage.[35]

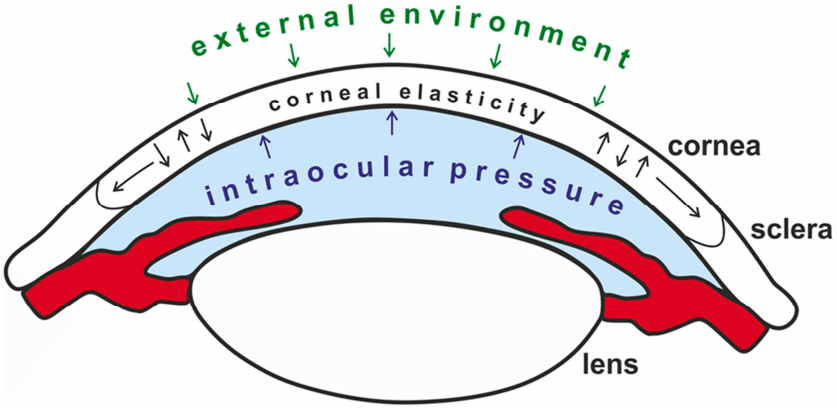


Figure No.4 : Diagrammatic representation of internal and external environment impacting corneal elasticity[36]

Anisotropy and Heterogeneity

i. Anisotropy: Mechanical properties differ along different meridians of the cornea.[35] The superior-inferior axis may exhibit different stiffness compared to the nasal-temporal axis.[35]

ii. Heterogeneity: Central cornea is mechanically stronger, while the periphery is more flexible to accommodate intraocular pressure (IOP) fluctuations.[35]

Hydration and Biomechanics

Proper hydration is critical for maintaining stromal thickness and transparency.[35]

Overhydration: Weakens stromal strength and increases susceptibility to deformation.

Dehydration: Leads to stiffening and reduced flexibility.[35]

Biomechanical Changes with Aging

Ageing is associated with increased collagen crosslinking (non-enzymatic) enhances stiffness but reduces flexibility.[35] In addition, decrease in GAG content impairs

hydration regulation and shock absorption. Ageing is also associated with reduced keratocyte density which stromal repair and maintenance.[35]

Assessment of cornea using non-contrast specular microscope

Specular microscopy is a non-invasive imaging technique used to evaluate the corneal endothelium by utilizing the principle of specular reflection.[37] This optical principle involves the reflection of light from the smooth endothelial surface at specific angles, which enables high-resolution imaging of cell boundaries.[37]

Figure No.5 : Specular microscope

Specular light reflex is based on the principle that angle of incident beam is equal to the angle of the reflected beam.[37] The endothelial cells have a refractive index greater than 1.336 value for the aqueous humor,which allows reflection of light at endothelial-aqueous interface.[37] This reflected light is captured and analysed to create an image of the endothelial cell layer. This allows real time visualization and assessment of endothelial cells.[37]

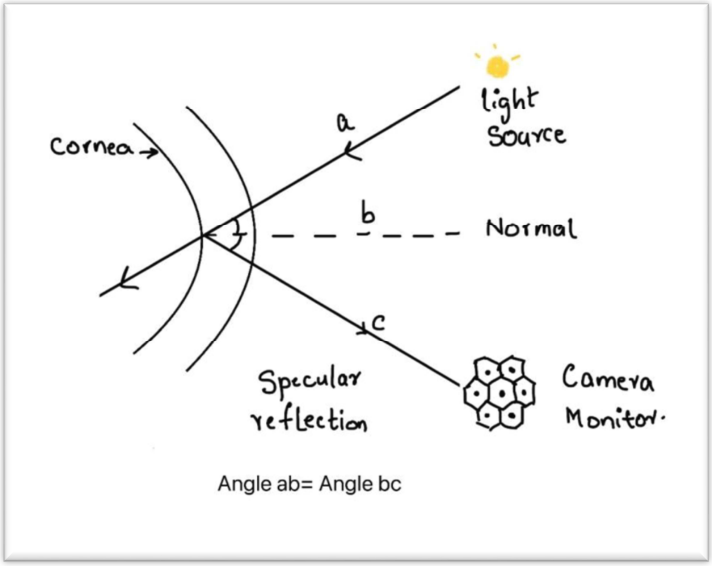


Figure No.6 : Principle of specular microscope (a: incident light ray, c= reflected light ray, ab: angle of incident, bc: angle of reflection)[37]

The endothelial cell morphology analysis includes cell area (square micrometre), endothelial cell density (cells/mm^2), polymegathism (in co-efficiency of variance), and pleomorphism (% of hexagonal cells).[37]

- The cell density is determined by the following equation:

$$\text{Cell density} = \frac{10^6}{\text{Average cell area}}$$

- The coefficient of variation (CV) is derived by the equation:

$$CV = \frac{\text{SD cell area}}{\text{Mean cell area, } \mu m^2}$$

While low cell density (<1000 cells/mm^2) can lead to corneal decompensation during intraocular surgery, and higher co-efficiency of variance (>0.4%) is called polymegathism.[37]

A specular microscope measures central corneal thickness by analysing the reflection of light from the corneal endothelium by calculating the distance between the anterior

corneal surface and the reflecting surface of the endothelial cells.[37] The specular microscope model used in this study is Rexxam SPM 700.

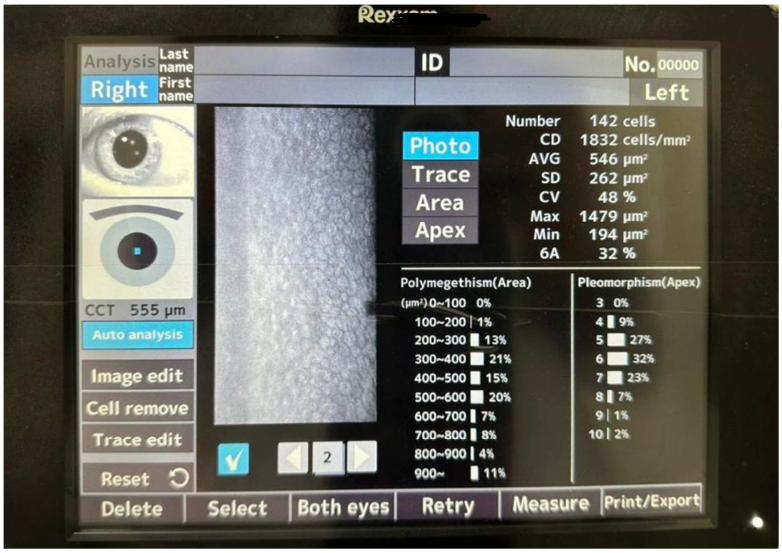


Figure No.7 : Pictorial representation of a specular microscopy measurement of corneal morphology (CCT: Central corneal thickness, CD: Endothelial cell density, CV: Co-efficiency of variance, 6A: Hexagonal cell percentage)[38]

B. Diabetes Mellitus and its effect on cornea

Overview

Diabetes mellitus is a chronic non-communicable disease characterized by elevated blood glucose levels due to decreased insulin secretion or insulin resistance.[39] The number of individuals living with diabetes has increased drastically, rising from 200 million in 1990 to 830 million in 2022.[39] This growth is disproportionately seen in low- and middle-income countries.[39] Diabetes significantly contributes to morbidity and mortality worldwide.[39] It leads to both micro and macrovascular complications with complications such as blindness, kidney failure, heart disease, stroke, and amputations.[39]

In 2022, 14% of adults aged 18 years and older were living with diabetes.[39] Moreover, more than half of them were not taking medications.[39] They directly attribute to 1.6 million deaths worldwide.[39] In India, an estimated 77 million population were affected by diabetes mellitus in 2019, and it is estimated to increase to 134 million by 2045.[8]

Impact of Disease on Cornea

Diabetes mellitus is a hyperglycaemic state that alters stromal stiffness due to excessive non-enzymatic collagen crosslinking.[35] In addition, endothelial dysfunction exacerbates hydration imbalance, reducing mechanical integrity.[35] Diabetic keratopathy is characterized by reduced corneal epithelial sensitivity, recurrent corneal ulceration, and delayed wound healing.[19] The hyperglycaemia state has distinctive effects on various corneal parts. It is also associated with structural and functional alterations in the corneal epithelium, stroma, and endothelium.[19]

Following are the major changes noted:

Diabetic Corneal Neuropathy

Neuropathy results from chronic hyperglycaemia damaging the trigeminal nerve. Due to hyperglycaemia state, accumulation of Advanced Glycation End Products (AGEs) and oxidative stress contribute to nerve Fiber degeneration.[19] This leads to reduced corneal innervation.[19] Immunocyte accumulation and mitochondrial damage exacerbate the condition. Chronic nerve damage impairs epithelial healing and predisposes the cornea to ulceration and perforation.[19] This can also affect healing following surgery. They are characterised by photophobia, irritation, and pain, though some patients remain asymptomatic.[19\

Corneal Epithelium Abnormalities

Hyperglycaemia-induced overexpression of matrix metalloproteinases (MMPs) leads to basement membrane degradation.[19] In addition, it can lead to accumulation of AGEs at the epithelial-stromal interface, disrupting cell adhesion and basement membrane integrity. The AGEs are particularly distributed on the basement membrane laminin.[19] Moreover, reduced neurotrophic factors impair epithelial homeostasis and wound healing. Key investigative findings include reduced density of basal epithelial cells, and increased cell size variability (polymegathism).[19]

Corneal Stroma Alterations

Structural changes following diabetes mellitus include increased central corneal thickness (CCT) due to stromal swelling, particularly in advanced diabetic neuropathy.[19] Moreover, disorganized collagen fibrils and abnormal crosslinking mediated by AGEs lead to reduced corneal transparency.[19]

At a cellular level, there are altered keratocyte density and activation in response to oxidative stress and hyperglycemia.[19] Also, chronic inflammation impairs stromal matrix remodeling.[19]

Tear Film and Lacrimal Functional Unit (LFU) Abnormalities

Diabetes mellitus can lead to reduced tear production due to lacrimal gland dysfunction and neuropathy.[19] Increased tear film osmolarity triggers inflammatory cascades, leading to dry eye syndrome (DES).[19]

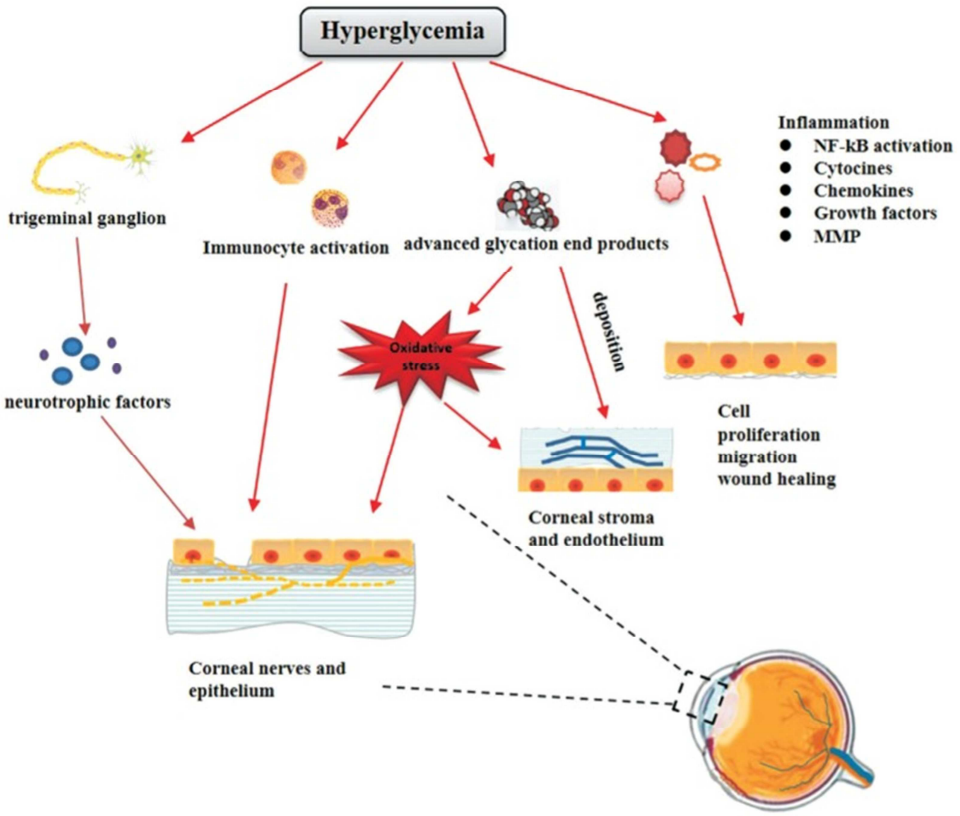


Figure No.8 : Pathophysiology of diabetes mellitus on cornea[19]

Corneal Endothelium Abnormalities

i. Role of Advanced Glycation End Products (AGEs): Accumulation of AGEs IN Descemet's Membrane led to endothelial cell adhesion and structural integrity.[19]

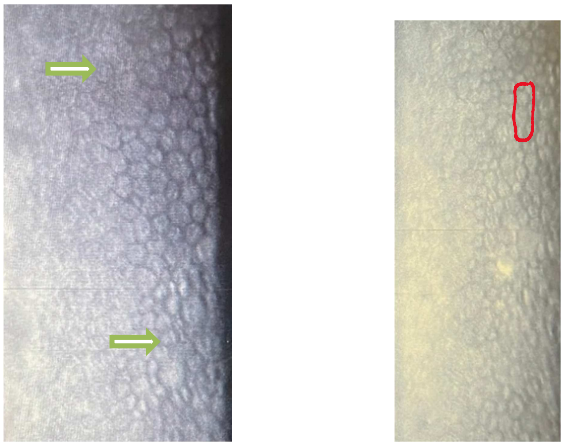
ii. Mitochondrial Dysfunction: AGEs induce mitochondrial oxidative stress, impairing energy-dependent processes like ionic transport.[19]

iii. Oxidative Stress: Chronic hyperglycaemia promotes reactive oxygen species (ROS) production, damaging endothelial DNA, proteins, and lipids. ROS disrupt endothelial tight junctions, increasing permeability and reducing barrier integrity.[19]

iv. Inflammatory Mediators: Elevated levels of pro-inflammatory cytokines (e.g., IL-6, TNF-α) in the aqueous humour of diabetic patients exacerbate endothelial cell apoptosis and dysfunction.[19]

v. Structural changes in the endothelial cell like thickened Descemet's membrane, interferes with nutrient and ion exchange between the aqueous humour and stroma.[19]

These lead to thickened central corneal thickness, reduced endothelial cell density, polymegathism, and pleomorphism.[19]



a **b**

Figure No.9 : specular microscopy of endothelial cell changes in diabetes mellitus (a: shows polymegathism, b.: shows pleomorphism)

C. Cataract in diabetes mellitus

Overview

Cataract is a clinical condition defined as the progressive loss of lens transparency, resulting in impaired vision. This loss of clarity is caused by structural and biochemical changes in the lens fibres and proteins, including aaggregation of

crystallin proteins, ddisruption of lens Fiber architecture, and ooxidative stress and accumulation of advanced glycation end-products (AGEs).

Cataract is the leading cause of reversible blindness, and is the second most common cause of severe visual impairment worldwide.[1] Cataracts significantly affect quality of life and productivity, particularly in older adults. Among population aged 50 years or above, cataract and refractive error constitutes for 55% of blindness and 77% of severe visual impairment worldwide.[1] Compared to non-diabetics, the incidence of cataract is 4 times higher among diabetics in younger population (<65 years), and it is two times higher in elderly population (>65 years).[11] Moreover, diabetes mellitus increases the risk of complications following cataract surgery.[12] This includes morphological and functional impairment at corneal endothelium.[13-14]

Anatomy and Function of the Lens

The crystalline lens is a transparent, biconvex, avascular structure located behind the iris and anterior to the vitreous body.[40-41] Its primary functions include focusing light onto the retina for clear vision (light refraction), and adjusting refractive power for near and distant vision (accommodation).[40-41]

The lens is enclosed within an elastic capsule and suspended by zonular fibres from the ciliary body.[40-41] It originates from surface ectoderm during embryogenesis.[40-41] Lens fibres are continuously produced by anterior epithelial cells, with older fibres compacted into the lens nucleus.[40-41] This lifelong growth makes the lens susceptible to age-related changes.[40-41]

Pathophysiology

The primary mechanisms of cataract formation include:

i. Age-Related Changes: Reduced efficiency of nutrient and antioxidant transport from the aqueous humour, and decreased enzymatic redox activity and crystallin protein solubility, leading to protein aggregation.[40-41]

ii. Oxidative Stress: ROS generated by environmental factors (e.g., UV light) and metabolic processes damage lens proteins and lipids. Oxidative damage impairs lens transparency and induces cross-linking of lens crystallins.[40-41]

Risk Factors

1. Non-Modifiable:

i. Aging: The most significant factor with lens opacity increasing progressively with age.[40-41]

ii. Genetic predisposition and congenital anomalies.[40-41]

2. Modifiable:

i. Poor glycaemic control in diabetics: Promotes osmotic stress through the polyol pathway.[40-41]

ii. UV light exposure: Increases ROS generation in the lens.

iii. Smoking: Elevates oxidative stress.

iv. Nutritional deficiencies: Lower antioxidant levels exacerbate lens damage.[40-41]

Classification of Cataract

Cataracts are classified based on their anatomical location, aetiology, morphology, and clinical grading systems.

1. Anatomical Classification

i. Nuclear Cataract: Involves the central part of the lens (nucleus). These are characterised by myopic shift in early stages due to changes in lens refractive index.[40-41] Advanced stages (brunescent cataracts) lead to hardening and significant colour changes (yellow to brown). They are common in age-related cataract.[40-42]

ii. Cortical Cataract: It affects lens cortex, presenting as spoke-like or wedge-shaped opacities extending from the periphery.[40-41] They are primarily due to osmotic imbalance due to electrolyte dysregulation, and may present as glare, halos, and reduced peripheral vision. They interfere with contrast sensitivity.[40-42]

iii. Subcapsular Cataract (PSC): They are located just beneath the lens capsule.[40-42] They can be anterior or posterior subcapsular cataract. They are commonly noted in prolonged steroid use, diabetes mellitus, and radiation exposure. They present with profound glare sensitivity, especially in bright light, and affects near vision disproportionately.[40-42]

iv. Capsular cataract: it involves the capsule.[40-42]

v. Polar cataract: involves capsule and the superior part of the cortex in the polar region.[40-42]

iv. Mixed Cataracts: They involve features of multiple types, commonly seen in advanced cases.[40-42]

2. Morphological Classification

Morphological features provide insights into disease progression and severity:

i. Lamellar Cataracts: Affect specific layers of the lens.

ii. Cupuliform Cataracts: Cup-shaped opacities, often linked to trauma or specific systemic diseases.

3. Etiological Classification

i. Age-Related Cataracts: The most prevalent type, caused by cumulative oxidative stress and protein denaturation. These can be Nuclear sclerotic, Cortical, Posterior subcapsular, or mixed.

Stages of senile cataract:

i. Stage of Lamellar Separation: Patients experience blurred vision, frequent changes in prescription glasses due to rapid changes in the refractive index of the lens, and painless, progressive, gradual vision loss. It is detected during a slit-lamp examination but not visible to the naked eye.[42]

ii. Incipient Stage: Lens opacities appear as black spots against a red reflex background during ophthalmoscopy.[42] The iris shadow becomes visible on the lens surface. In this stage the patients experience, Polyopia and see colored halos. Patients experience glare in sunlight and progressive vision reduction.[42]

iii. Immature Cataract: The iris shadow is present on the lens, and lens opacities appear as black spots against a red background on ophthalmoscopy.[42] While cortical cataracts present with reduced night vision and impaired day vision, nuclear cataracts present with "Second sight" or a myopic shift.[42]

iv. Mature Cataract: Immature cataract progress into mature cataract characterised by absence of iris shadow, and absence of fundal glow.[42] The present with significant visual acuity reduction, possibly limited to light perception (PL positive).[42] Cortical cataract may appear as pearly white, and nuclear cataract show coloured appearance.[42]

v. Hyper mature Cortical Cataract (Morgagnian Cataract): The lens cortex becomes liquefied, and the nucleus settles within the capsular bag.[42]

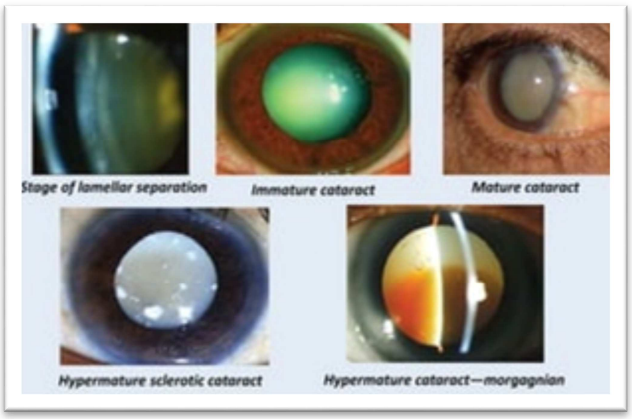


Figure No.10 : Stages of senile cataract

ii. Congenital and Paediatric Cataracts: They are present at birth or in early life, and are associated with associated with genetic abnormalities (e.g., galactosemia) or intrauterine infections (e.g., rubella).[40-42]

iii. Traumatic Cataracts: Develop due to mechanical injuries, radiation, or chemical exposure. Their unique clinical features include "rosette cataracts" from blunt trauma.

iv. Secondary Cataracts: Result from systemic diseases or medications:

- Diabetes mellitus: Promotes osmotic stress and oxidative damage.
- Corticosteroids: Increase risk of posterior subcapsular cataracts.

- Uveitis: Chronic inflammation accelerates lens opacity.[40-42]

Indications for Surgery

"Guidelines for the Management of Cataract in India" published by Vision 2020: The Right to Sight India provides guidelines for management of cataract cases.[43]

Criteria and Timing Cataract surgery: Cataract Surgery is recommended for patients with uncorrected Snellen visual acuity of 6/24 or less. Exceptions include patients with significant visual disability affecting quality of life despite better acuity.[43] However, advances in surgical techniques have lowered the threshold for surgical intervention due to better outcomes.[43]

Timing of surgery varies based on severity, urgency, and associated conditions:

i. Surgery within a month for bilateral cataract with a fellow eye vision >6/60.

ii. Immediate surgery if visual acuity is limited to finger counting close to the face.[43]

Contraindications: Surgery is deferred for patients unlikely to achieve a two-line improvement in vision or those with significant systemic or ocular risks.[43]

Preoperative Assessment: Comprehensive ophthalmic evaluation, including:

i. Best-corrected visual acuity (BCVA).

ii. Slit-lamp examination to assess lens opacification.

iii. Optical coherence tomography (OCT) for detecting macular oedema or retinal pathology.[43]

Surgical techniques:

i. Phacoemulsification: Ultrasound energy is used to emulsify the lens, followed by IOL implantation. It is preferred due to its minimally invasive nature and quicker recovery.[43]

ii. Manual Small-Incision Cataract Surgery (MSICS): Cost-effective alternative in resource-limited settings.[43]

iii. Femtosecond Laser-Assisted Cataract Surgery (FLACS): Enhances precision in capsulorrhexis and lens fragmentation. Improves outcomes in complex cases.[43]

D Manual Small-Incision Cataract Surgery (MSICS)

MSCIS is the preferred cataract surgery technique in high load, resource limited settings.[44] A cross-sectional, questionnaire-based observational study was conducted among 278 ophthalmologists worldwide.[44] They noted that peribulbar anaesthesia was the most commonly used (84.17%). Among the surgical techniques, majority (77.3%) preferred capsulorrhexis before corneoscleral tunnel incision.[44] Frown-shaped incisions (52.15%) were favoured for minimizing astigmatism.[44] Among instrumentation, continuous curvilinear capsulorrhexis was used by 93.9% of surgeons.[44] Anterior chamber maintainers were routinely used by 81.3%, and non-foldable intraocular lenses (IOLs) were the preferred choice (62.9%). Among post-operative practices, subconjunctival antibiotic-steroid injections were given by 59.7% of surgeons, and stromal hydration was the most common method to close side ports.[44]

Pre-operative steps:

i. Start mydriatic drops 30-60 minutes before surgery: Tropicamide 1% combined with Phenylephrine 2.5%.[45]

ii. Add cycloplegics (e.g., atropine) in cases of posterior synechiae or poor dilation.[45]

iii. Local Anaesthesia Preparation (peribulbar or retrobulbar block): Use 2% lidocaine or a mixture of lidocaine and bupivacaine with hyaluronidase for diffusion.[45]

iv. Sterile preparation by: cleaning the periocular area with povidone-iodine solution (5% for the eye and 10% for the skin). This is followed by draping the patient to isolate the operative field. Use a lid speculum to expose the eye.[45]

Manual small incision cataract surgery (procedure)

Conjunctival Peritomy: A fornix-based conjunctival flap is created to expose the sclera.

Scleral Tunnel Incision: A self-sealing scleral tunnel incision is made approximately 1.5–2 mm posterior to the limbus.

Paracentesis: One or two side-port incisions are created for instrument access.

Capsulotomy: An anterior capsulotomy is performed to access the lens.

Hydro dissection: Balanced salt solution is injected to separate the lens nucleus from the cortex.

Nucleus Delivery: The nucleus is prolapsed into the anterior chamber and extracted using an wire vectis or phacosandwich technique.

Cortical Cleanup: Residual cortical material is aspirated to prepare for IOL placement.

IOL Implantation: An intraocular lens is implanted into the capsular bag.

Wound Closure: The scleral tunnel is checked for watertightness; sutures are placed if necessary.

Conjunctival Repositioning: The conjunctival flap is repositioned and secured.

Modified manual small incision cataract surgery technique for phacoemulsification-trained surgeons (operative procedure):

i. Conjunctival Peritomy and Scleral Tunnel Creation: Limbal conjunctival peritomy is performed spanning 3-4 clock hours.[46] Scleral Tunnel is prepared by making an incision starting 2 mm posterior to the limbus, followed by preparing 6-7 mm scleral tunnel, advancing anteriorly into the mid-posterior corneal

stroma.[46] Care should be taken to ensure a "funnel-shaped" configuration where the internal opening is larger than the external to facilitate nucleus delivery.[46]

ii. Large Corneal Wound Creation: Using a 2.65 mm keratome, a clear corneal tunnel (CCT) is created at 90° to the scleral tunnel.[46] This is followed by mimicking a typical phacoemulsification incision to allow intuitive manipulation of instruments.[46] The clear corneal tunnel should ensure adequate space for IOL insertion and removal of cortical matter.[46]

Iiii. Capsulotomy: Stain the anterior capsule with Trypan blue to enhance visibility, especially in cases with poor red reflex.[46] This is followed by performing a continuous curvilinear capsulorrhexis (CCC) using a bent 26G needle or capsulotomy forceps.[46]

iv. Hydro dissection and Nucleus Delivery: Hydro dissection is performed by using a balanced salt solution to separate the nucleus from the cortex. This ensures free mobility of the nucleus.[46] Inject viscoelastic beneath the nucleus to protect the posterior capsule and endothelium. This helps in prolapse of nucleus into the anterior chamber.[46]

Nucleus Delivery is achieved by applying controlled pressure on the posterior lip of the scleral tunnel.[46] Care needs to be taken to avoid inserting instruments behind the nucleus during delivery to minimize risks.[46]

v. Irrigation and Aspiration of Cortical Matter: Using a Simcoe cannula or an irrigation/aspiration, probe to remove cortical material. Perform through the corneal wound to maintain anterior chamber stability.[46]

vi. Intraocular Lens (IOL) Insertion:

Insert the IOL through the corneal tunnel. Foldable IOLs can be implanted using an injector system and non-foldable IOLs may require larger tunnel incisions. Ensure proper positioning of the IOL in the capsular bag.[46]

vii. Wound Closure and Conjunctival Peritomy Closure: Hydrate the edges of the corneal and scleral tunnels to achieve a watertight seal.[46] Use a single safety suture if the scleral tunnel is large, or wound integrity is compromised.[46] Oppose the conjunctival edges without sutures or secure with cautery if necessary.

Postoperative Care

i. Administer topical moxifloxacin 0.5% and dexamethasone 0.1%:

ii. Antibiotics: Four times daily for 4 weeks.

iii. Steroids: Six times daily for 1 week, then tapered to four times daily for 4 weeks.

iv. Educate the patient about post-op care and symptoms of complications like pain, redness, or sudden vision loss.

E. Effect of MSICS and diabetes on cornea

Biomechanics in Cataract Surgery

Surgical Impact: Manual small incision cataract surgery (MSICS) induces mechanical stress, particularly on the endothelium and stroma.[35]

Postoperative Changes: Following cataract surgery, increased central corneal thickness (CCT) may be noted due to from transient edema.[35] Progressive loss of endothelial cells can reduce corneal stiffness over time.[35]

Impact of diabetes mellitus on cataract surgery

Hyperglycaemia-induced myopic shifts and corneal changes are common among diabetic patients, and these can influence cataract surgery outcomes.[49] Diabetic corneas exhibit delayed recovery of biomechanical properties due to pre-existing endothelial and stromal dysfunction.[35] A systematic review by Kelkar A et al noted that Cataract surgery in diabetics can lead to Diabetic retinopathy (DR) progression.[49] In addition, it increases risks of macular oedema (ME), vitreous haemorrhage, and tractional retinal detachment. In addition, postoperative visual outcomes are often influenced by glycaemic control and pre-existing ocular conditions like diabetic maculopathy.[49] The review noted that Diabetics increase the risk of corneal endothelial cell loss, intraoperative hyphaema, and photic retinopathy. Moreover, increased inflammatory cytokines post-surgery may exacerbate retinopathy.[49]

G. Previous studies

Pre-operative:

A prospective clinical study done by Storr-Paulsen et al noted that T2DM has no effect on corneal endothelial cell density or morphology, provided glycaemic control is maintained.[22] However, CCT increased among diabetic cases. It was also noted that, among poor glycaemic control patients, corneal endothelial cell density and CV were low compared to non-diabetic subjects.[22]

A study done by Pandey S et al noted that T2DM patients exhibited significantly reduced endothelial cell density (ECD) (2447.32 ± 269.89/mm²), hexagonality (6A) (45.03 ± 6.71%), average cell area (413.97 ± 50.19 µm²), coefficient of variance (CoV) (39.84 ± 15.59%), and standard deviation of cell size (167.05 ± 77.91).[21]

However, there was no significant difference in CCT between diabetic and non-diabetic patients.[21] The study also noted that longer diabetes duration (>10 years) correlated with decreased ECD and increased CoV.[21] In addition, Poor glycaemic control (HbA1c >10%) significantly reduced ECD and increased average cell area ($p < 0.001$).[21] Moreover, as the severity of DR progressed from non-proliferative (NPDR) to proliferative (PDR), ECD further declined, and average cell area increased significantly ($p < 0.001$).[21] This was in contrast to study by Storr-Paulsen et al.[22]

A prospective observational study including 1188 eyes (592 diabetic, 596 control) by Jha A et al noted increased CCT (522.1 ± 36.6 μm in diabetics vs 514.9 ± 37.1 μm in controls; p=0.001), reduced endothelial cell density (2484.5 ± 299.5 cells/mm² in diabetics vs. 2555.9 ± 258.2 cells/mm² in controls; p=0.017), increased CV (40.3 ± 6.1 in diabetics vs 37.2 ± 6.1; p<0.001 in controls), and decreased Hex (39.9 ± 5.2% in diabetics vs 44.6 ± 6.0% in controls; p<0.001).[50] The study also noted that poor glycaemic control showed a clear negative impact on corneal health. In addition, increasing severity of DR linked to progressive corneal changes. However, the study was cross-sectional in nature, and hence progressive changes couldn't be determined.[50]

A case-control study by Papadakou P et al endothelial sell density was significantly lower among T2DM patients.[51] However, there were no significant different among other parameters between diabetics and non-diabetics. In addition, duration of T2DM, poor glycaemic control, and progressive retinopathy influenced endothelial cell density.[51] Hence contrasting evidence are noted as per current literature, and further studies are recommended.

Post small incision cataract surgery

A prospective observational study by Kudva AA et al conducted among 106 patients (54 diabetics, 52 nondiabetics) with senile uncomplicated cataracts noted that diabetics exhibited significantly higher endothelial loss at 1 and 3 months postoperatively (27.5% vs. 18.3% in controls, $p<0.001$).[14] They also noted that diabetics showed consistently thicker corneas (CCT) postoperatively compared to nondiabetics, with significant differences at 1 month ($p = 0.0012$). Similarly, diabetics exhibited significantly higher postoperative CV and reduced hexagonality indicating greater endothelial cell variability and stress.[14] The results emphasize the need for appropriate monitoring of corneal parameters in diabetics undergoing cataract surgery to mitigate potential complications.[14]

A prospective observational study by Morikubo S et al among 93 eyes from patients with type 2 DM and 93 eyes from nondiabetic individuals undergoing phacoemulsification and intraocular lens implantation were analyzed.[52] The study noted that both groups exhibited increased corneal thickness immediately after surgery, with gradual recovery.[52] At 1 month, the diabetic group had a significantly higher residual increase in CCT (+1.6%) compared to the nondiabetic group (+0.04%; $p = 0.03$). Moreover, delayed recovery of corneal oedema was evident in diabetics. Postoperative endothelial cell loss was significantly greater in the diabetic group (7.9% in diabetics vs. 3.6% in nondiabetics at 1 week of follow up, $p = 0.04$).[52] While polymegathism was noted in both groups, there was no significant difference between the groups. · While the study was a well-matched group study, with standardized surgical techniques and robust data collection, the main limitation of the study was its short follow up (1 month).[52]

A cohort study by Zeba Ahmed et al among 60 diabetic patients and 60 age-matched non-diabetic controls who underwent manual small-incision cataract surgery (MSICS) noted that there was significant difference in endothelial cell density between diabetics and non-diabetics in both pre-operative and post-operative phase.[53] Endothelial cell loss was significantly higher in diabetics (3.76%) than in non-diabetics (2.02%). Diabetics exhibited consistently higher CV (indicative of increased polymegathism) postoperatively compared to non-diabetics.[53] However, no significant differences were noted between diabetics and non-diabetics in pre- or postoperative hexagonality. One of the major limitations of the study was its shorter follow up (8 weeks).[53]

A prospective comparative study by Raut NG et al among 100 diabetics and 100 non-diabetics who underwent uneventful MSICS noted that post-operative endothelial cell loss was significantly higher among diabetics compared to non-diabetics (Diabetics: 22.01 ± 10.49% vs Non-Diabetics: 16.64 ± 6.72%, $p < 0.0001$).[54] Similarly, CCT was significantly higher among diabetics during post-operative follow up (Diabetics: 527.64 ± 14.34 µm vs Non-Diabetics: 523.13 ± 15.36 µm, $p = 0.0326$).[54] However, no significant differences between groups across follow-ups were noted for CV and hexogenicity. These findings emphasize the need for thorough preoperative evaluations, intraoperative precautions, and close postoperative monitoring to optimize outcomes in this high-risk group.[54]

Lacunae and need for study

Cataract surgery-induced damage to the corneal endothelium is multifactorial, influenced by systemic health, ocular conditions, surgical techniques, and postoperative care. Advances in surgical methods, viscoelastic agents, and

medications have improved outcomes, but protecting endothelial cells remains crucial. Providing evidence-based information to the patients on the effects of cataract surgery among diabetes is important for comprehensive care of the patient. Moreover, preventive and promotive care of the patients can be enhanced by this information. However, there are limited studies on the effect of small incision cataract surgery on endothelial properties among diabetics. But there are limited studies on this, and there is a lacuna of evidence on them. Hence the study is a necessity.

MATERIALS AND METHODS

Source of data: Newly diagnosed and already diagnosed patients of diabetes with cataract attending the ophthalmology OPD of KLES Dr. Prabhakar kore Hospital and MRC, Belagavi.

Study Design: One-year Longitudinal study

1.1 Study Period: The study was conducted between April 2023 and April 2024.

Sampling technique: Convenience sampling

1.2 Inclusion Criteria:

- Patients aged between 50 years to 80 years
- Patient with senile uncomplicated cataract (immature and mature)
- Patients willing to undergo Manual Small Incision Cataract Surgery.

1.3 Exclusion Criteria:

- Patients with corneal opacity
- Contact lens users.
- Patients having corneal dystrophies
- Diagnosed cases of age-related macular degeneration.
- Having history of any previous ocular surgery.
- Having any other systemic illness
- Known cases of glaucoma Known/unknown cases of uveitis
- Any history of trauma.

Sample Size:

$$N = \frac{(Z_{1-\alpha/2} + Z_{1-\beta)}^2 .(SD_1^2+SD_2^2)}{-(\bar{x}_1-x_2)^2} \quad {}^{*20\%}\text{attrition}$$

$$N = \frac{(1.96 + 1.64)^2 .(4.76^2+5.26^2)}{(42.83-38)^2} \quad {}^{*20\%}\text{attrition}$$

$N = 27.95\ {}^{*20\%}\text{attrition}$

N= 35 in each group

TOTAL n= 70

Sample size is calculated with 95% confidence interval and power with 20% attrition.

Where $Z_{1-\alpha/2}$ is 1.96, $Z_{1-\beta}$ is 1.64, SD1 is 4.76, SD2 is 5.26

$\bar{x}_1$ is 42.83 and X_2 is 38.

Therefore n = 70 i.e 35 in each group.

Study procedure:

After obtaining informed consent from subjects attending KLEs Dr. Prabhakar Kore Hospital, Belagavi, their demographic details, investigations and clinical history, anterior segment examination by slit lamp bio-microscopy, fundus examination by indirect ophthalmoscope were be assessed for compliance with inclusion and exclusion criteria. The patients who fulfilled the inclusion criteria underwent a random blood glucose test to uncover the undetected diabetes. All diabetic patients whether known or recently diagnosed underwent serum glycosylated haemoglobin, (HbA_1c) test. Through a non-contact specular microscope, corneal endothelial cell changes and central corneal thickness on preoperative day was performed and documented.

Pre-operative ocular dilatation was performed using tropicamide plus eye drops. All surgeries were performed by single surgeon under peribulbar anaesthesia prepared using 5 ml of 2% lignocaine with 150 units of hyaluronidase. Surgery was using 6mm scleral incision. All the steps of a standard manual small incision cataract were carried out and nucleus were prolapsed in anterior chamber after performing capsulorrhexis following which nucleus were delivered using wire Vectis or phacosandwich technique. Single polymethyl methacrylate intraocular lens was implanted after proper cortical aspiration. After this stromal hydration were done. Subconjunctival injection of gentamycin and dexamethasone were given.

Post-operative assessment of corneal endothelial cell changes and central corneal thickness were done using non-contrast specular microscope at day 1, 1 week, 1 month and 3 months.

Statistical analysis

Data was entered using statistical software R version 4.2.2 and Microsoft Excel. Frequency tables will be used to represent categorical variables. Continuous variables were measured using mean, median, range, and standard deviation. Chi-Square test was used to assess the association between attributes. Normality of variable was assessed using Shapiro Wilk test. If the data is normal, then parametric test will be used. Otherwise, non-parametric test will be used. Two sample t test / Mann Whitney U test will be used to compare the means/distributions between the groups. One-way repeated measures of ANOVA/ Friedman test is used to compare the means/distributions over different time points. Pairwise t test/Pairwise Wilcoxon test can be used as post hoc analysis. Appropriate tables and figures will be generated. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

Table No.1: Distribution of participants according to group

Group	Frequency	Percentage
Diabetic group	**35**	**50**
Non diabetic group	**35**	**50**
Total	**70**	**100**

The study included 70 participants who were planned for cataract surgery. The participants were divided into diabetic group (35, 50%) and non-diabetic group (35, 50%).

Table No.2 : Glycosylated haemoglobin status of diabetic patients

Glycosylated Hb variable	HbA1c (%)
Minimum	**6.5**
Maximum	**7.6**
Mean	**7.0**
SD	**0.31**

The mean HbA1C ranged from 6.5-7.6%, with mean of 7.0±0.31.

Table No.3: Distribution of participants according to age group

Age group	Diabetic n (%)	Non diabetic n (%)	Total N (%)	P value
50-60 years	13 (37.1)	15 (42.9)	28 (40)	**0.273**
61-70 years	18 (51.4)	12 (34.3)	30 (42.9)	
71-80 years	4 (11.4)	8 (22.9)	12 (17.1)	
Total	**35**	**35**	**70**	

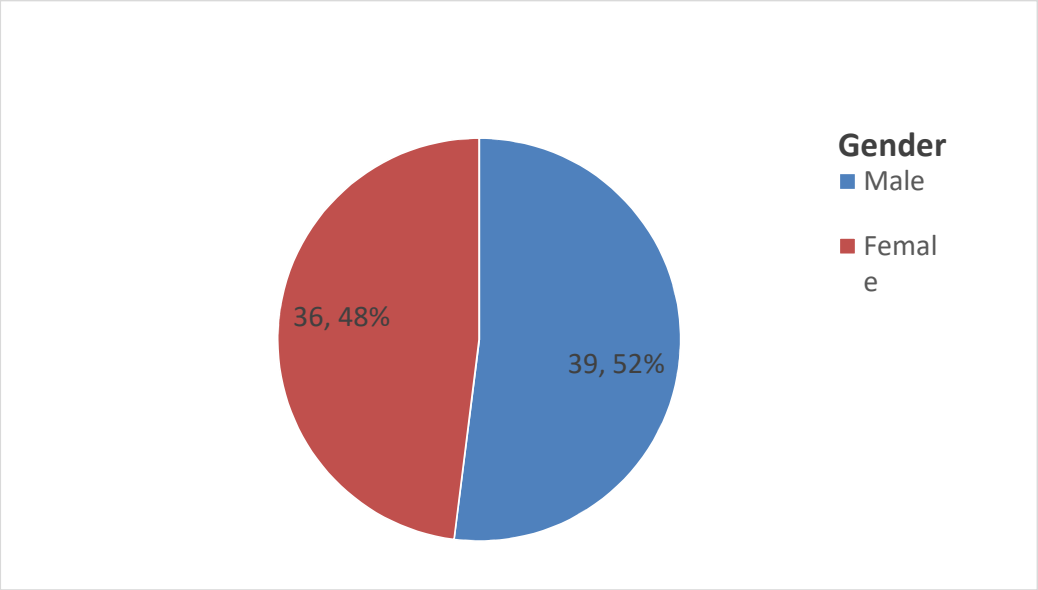
The study included 70 participants in the age range of 50 - 80 years, with mean of 64.1+7.0 years. They were equal distribution of participants among diabetic group (35, 50%) and non-diabetic group (35, 50%). The mean age of participants among diabetic and non-diabetic group was 63.6+6.3 years and 64.5+7.7 years, respectively. This difference was not statistically significant (p=0.590).

Overall, majority of participants belonged to the age group of 50-60 years (42.9%), followed by 50-60 years (40%), and 71-80 years (17.1%). On further evaluation, majority of participants among diabetic group were in the age group of 61-70 years (51.4%), and in comparison, majority of participants in the non-diabetic group belonged to 50-60 years (42.9%). However, this difference was not statistically significant (p-0.273). Hence, both group were comparable in terms of age of participants.

Table No.4 : Distribution of participants according to gender

Gender	Diabetic n (%)	Non-diabetic n (%)	Total n (%)	P value
Male	19 (54.3)	20 (57.1)	39 (55.7)	0.810
Female	16 (35.7)	15 (42.9)	31 (44.3)	
Total	35	35	70	

Graph 1: Distribution of participants based on gender



Majority of participants were male (55.7%), followed by females (44.3). On further evaluation, males were higher among diabetic group (54.3%) and non-diabetic group (57.1%). Hence both groups were comparable in gender (p=0.810).

Table No.5 : Distribution of participants based on pre-operative diminution of vision

Diminution of vision	Diabetic n (%)	Non-diabetic n (%)	Total n (%)	P value
Right eye	26 (74.3)	26 (74.3)	52 (74.3)	1.00
Left eye	29 (82.9)	24 (68.6)	53 (75.7)	0.265

74.3% of the participants complained of diminution of vision of right eye, and 75.7% of left eye. There was no significant difference in diminution of vision between the groups ($p<0.05$).

Table No.6 : Distribution of participants based on duration of diminution of vision

Duration of diminution of vision	Diabetic %	Non-diabetic %	Total %	P value
Up to 3 months	11.4	20.0	15.7	0.757
4-6 months	22.9	25.7	24.3	
7-9 months	8.6	2.9	5.7	
9-12 months	51.4	45.7	48.6	
>12 months	5.7	5.7	5.7	

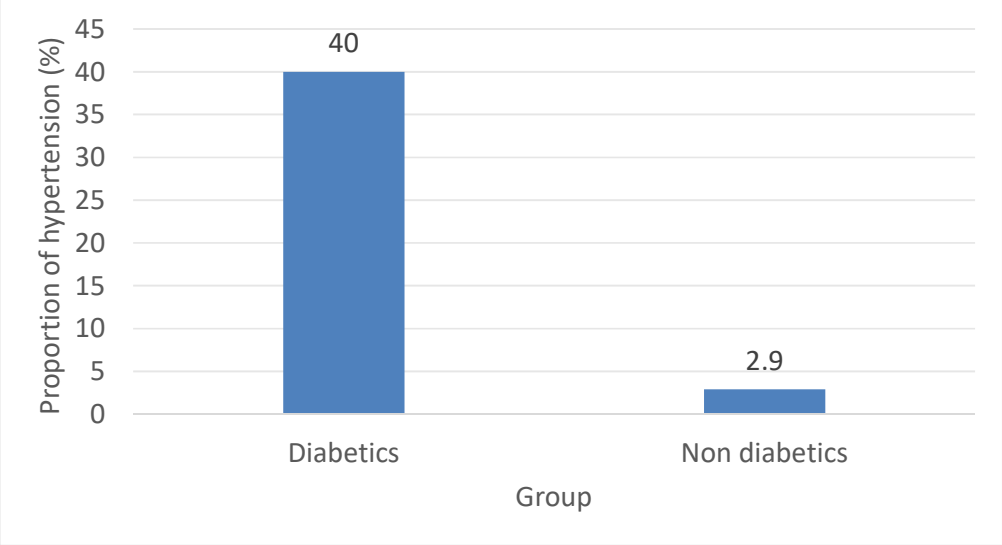
Majority of the participants had vision defect for 9-12 months (48.6%), followed by 4-6 months (24.3%). and less than 3 months (15.7%). Only 5.7% had diminution of vision for more than a year. There was no pre-operative difference in diminution of vision between the groups ($p<0.05$).

Table No.7 : Distribution of participants based on presence of hypertension

Hypertension	Diabetic n (%)	Non-diabetic n (%)	Total n (%)	P value
Hypertensive	14 (40)	1 (2.9)	15 (21.4)	**0.000***
Non hypertensive	21 (60)	34 (77.1)	55 (78.6)	
Total	35	35	70	

* Statistically significant

Graph 2: Comparison of hypertension between the groups



40% of participants in diabetics group were hypertensive, and in comparison, only 2.9% in non-diabetic groups were hypertensive. This difference was statistically significant (p=0.00).

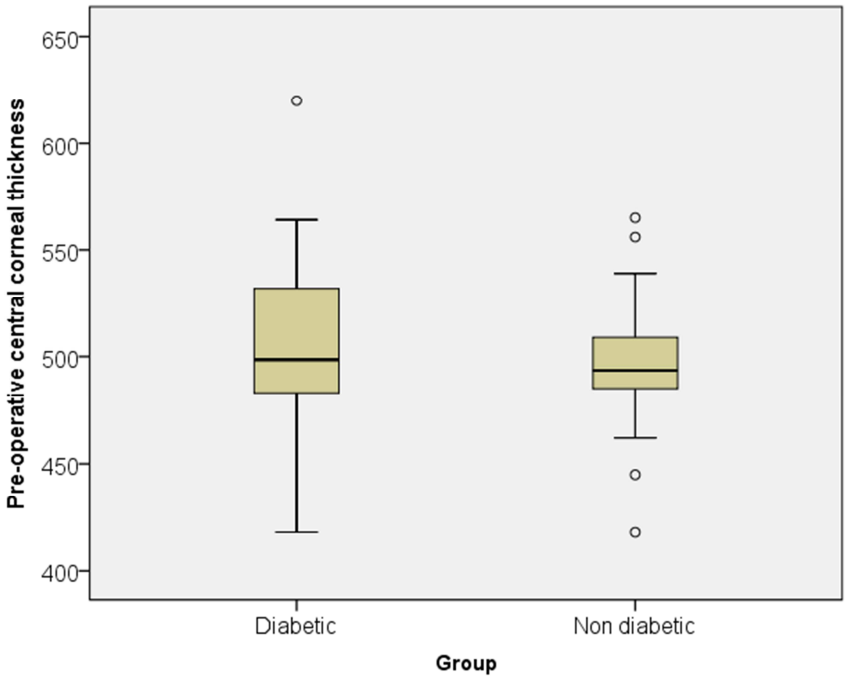
There was no history of ocular trauma or glaucoma among participants in both groups.

CENTRAL CORNEAL THICKNESS

Table No.8 : Comparison of pre-operative central corneal thickness between the groups

Central corneal thickness (μm)	**Diabetic**	**Non-diabetic**	**P value**
Mean±SD	508.4±36.1	495.1±30.9	0.104
Range	418 - 620	418-565	-

Graph 3: Boxplot comparing mean pre-operative central corneal thickness between the groups

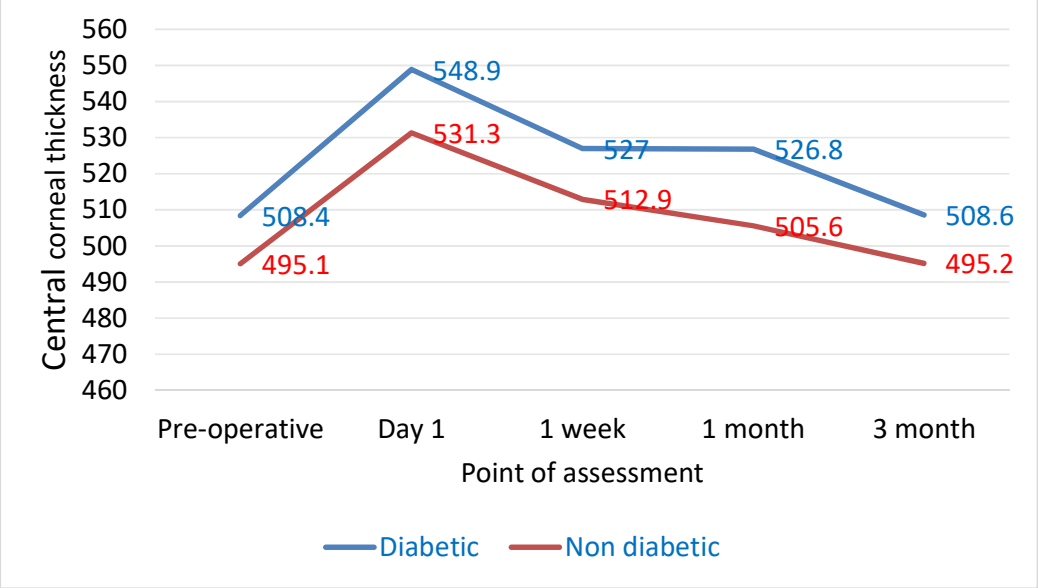


Among diabetic group, the pre-operative central corneal thickness ranged from 418-620 μm, with mean of 508.4±36.1 μm. In comparison, the thickness ranged from 418-565 μm among non-diabetic group, with mean of 495.1±30.9 μm. However, this difference was not statistically significant (p=0.104).

Table No.9 : Comparison of central corneal thickness across the study period between the groups

Central Corneal thickness	Pre-operative	Post-operative			
	Pre-op	**Day 1**	**1 week**	**1 month**	**3 months**
Diabetic Mean±SD	508.4±36.1	548.9±53.4	527.0±36.6	526.8±38.9	508.6±38.0
Range	418 - 620	469 - 681	460 - 630	465 - 681	438 - 618
Non diabetic Mean±SD	495.1±30.9	531.3±36.6	512.9±24.0	505.6±22.8	495.2±27.7
Range	418 - 565	463 - 611	465 - 585	460-577	423 - 552

Graph 4: Comparison of central corneal thickness between the groups

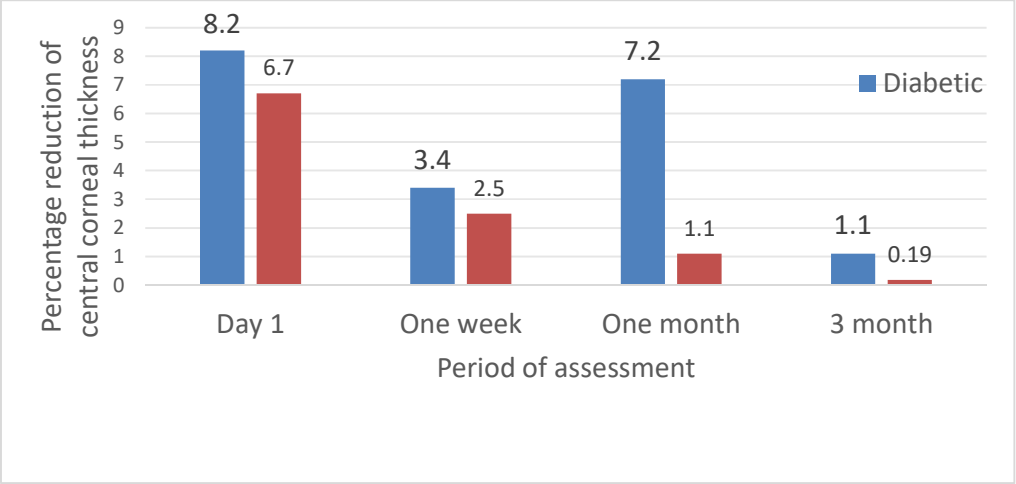


Among diabetic group, the mean central corneal thickness increased to 548.9±53.4 at day one following surgery. This reduced to 527.0±36.6 at one week, and to pre-operative thickness (508.6±38.0) at 3 months. Similarly, among non-diabetic group, it increased at day 1 (531.3+36.6), and subsequently reduced close to pre-operative phase at 3 months of follow up 495.2+27.7.

Table No.10 : Percentage change in central corneal thickness compared to pre-operative stage

Percentage reduction of Central corneal thickness	Post-operative			
	Day 1	1 week	1 month	3 months
Diabetic				
Mean±SD	8.0±6.0	4.0±6.6	5.9±7.0	0.23±4.6
Median (%)	8.2%	3.4%	7.2%	1.0%
Range (%)	20.7 - +7.1	14.8 - +9.6	18.2 - 9.0	8.8 - +8.2
Non diabetic				
Mean±SD	7.1±4.0	3.5±4.7	1.8±5.1	0.06±2.3
Median (%)	6.7%	2.5%	1.1%	0.19%
Range (%)	16 - -0.31	15.7 - -5.6	16.7 - - 10.7	4.5 - 6.8

Graph 5: Comparison of percentage increase of central corneal thickness (median) between diabetic and non-diabetic



Among diabetic patients, the central corneal thickness increased following the procedure by 8.2% at day 1 after surgery. However, the increase was only 1% at 3 months following the procedure.

Among non-diabetic patients, the central corneal thickness increased by 6.7% following the procedure. However, it improved to 2.5% at 1 week, 1.1% at 1 month, and 0.19% at 3 months of procedure.

Table No.11 : Comparison of median percentage increase of central corneal thickness between the groups

Phase of study	Percentage reduction of central corneal thickness (%)		P value
	Diabetic	Non-diabetic	
Day 1	8.2	6.7	0.307
One week	3.4	2.5	0.526
One month	7.2	1.1	0.003
3 months	1.0	0.19	0.823

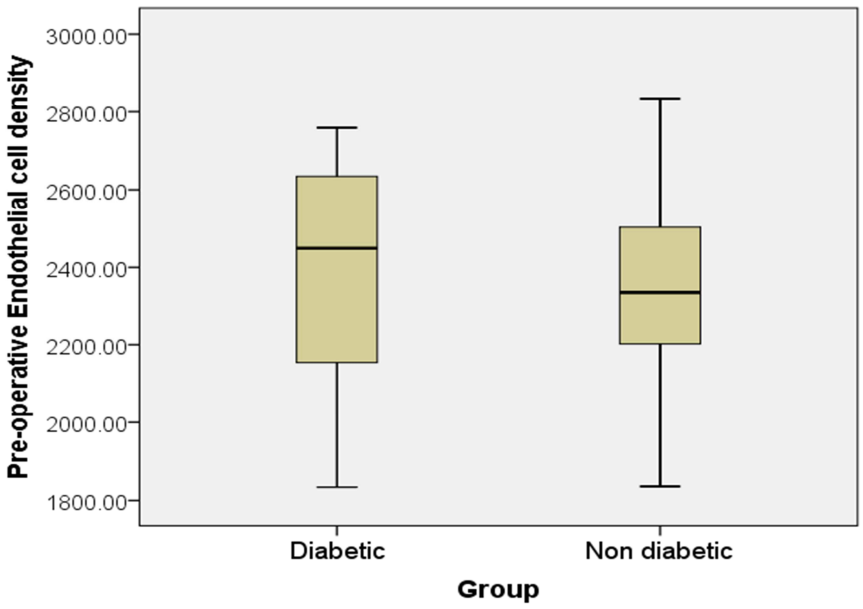
The study noted that there was no significant difference in change in central corneal thickness at post-operative day one (8.2% in diabetes vs 6.7% in non-diabetes, p=0.307), one week (3.4 in diabetes vs 2.5% in non-diabetes, p-0.526), and 3 months (1.0% in diabetes vs 0.19% in non-diabetes, p=0.823) between the groups. However, higher change was noted among diabetics (7.2%) when compared to non-diabetics (1.1%) at one month of follow up. This difference was statistically significant (p=0.003).

ENDOTHELIAL CELL DENSITY

Table No12 .: Comparison of pre-operative endothelial cell density between the groups

Endothelial cell density (µm)	Diabetic	Non-diabetic	P value
Mean±SD	2383±278	2342±236	0.514
Range	1834 - 2759	1836 - 2718	-

Graph 6: Boxplot comparing mean pre-operative endothelial cell density between the groups

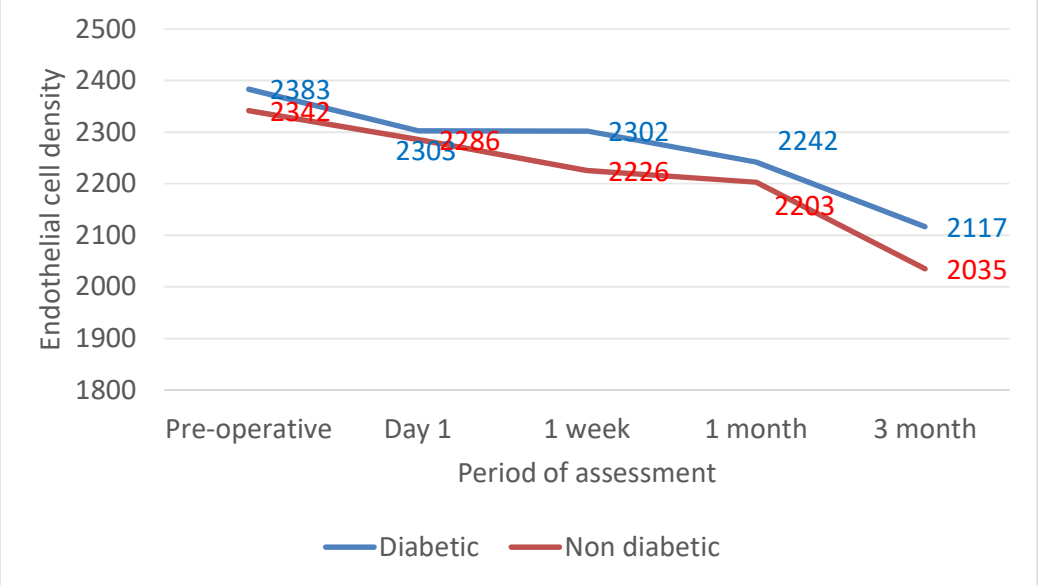


Among diabetic group, the pre-operative endothelial cell density ranged from 1834 - 2759 µm, with mean of 2383±27 µm. In comparison, the density ranged from 1836 - 2718 µm among non-diabetic group, with mean of 2342±236 µm. However, this difference was not statistically significant (p=0.514).

Table No.13 : Comparison of endothelial cell density across the study period between the groups

Endothelial cell density	Pre-operative	Post-operative			
	Pre-op	Day 1	1 week	1 month	3 months
Diabetic					
Mean±SD	2383±278	2303±326	2302±218	2242±197	2117±216
Range	1834-2759	1613-2894	1624-2718	1528-2663	1624-2599
Non diabetic					
Mean±SD	2342±236	2286±326	2226±248	2203±257	2035±185
Range	1836-2718	1775-2917	1834-2715	1624-2835	1524-2553

Graph 7: Comparison of endothelial cell density between the groups

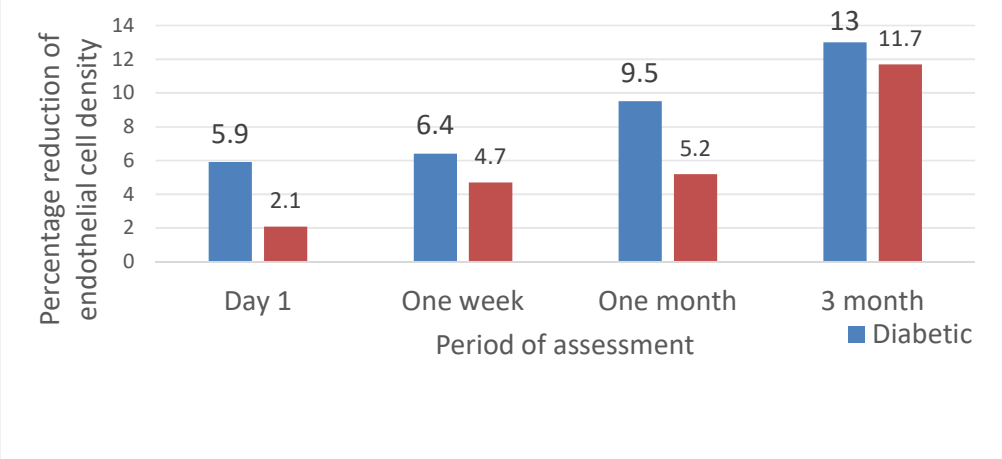


Among diabetic group, the endothelial cell density decreased across study period with mean at post-operative day 1, one week, one month, and 3 months of 2303+326, 2302+218, 2242±197, and 2117±216, respectively. Among non-diabetic group, it decreased from 2286±326 (day 1) to 2035+185 (3 months).

Table No.14 : Percentage change of endothelial cell density between the groups

Percentage change of Endothelial cell density	Post-operative			
	Day 1	**1 week**	**1 month**	**3 months**
Diabetic Median (%)	5.9	6.4	9.5	13
Non diabetic Median (%)	2.1	4.7	5.2	11.7

Graph 8: Comparison of percentage endothelial cell density loss (median) between diabetic and non-diabetic group



There was variable change in endothelial cell density among both groups. Among diabetics, there was minimal median reduction (5.9%) at day 1 post-operative period, followed by subsequent reduction of 6.4%, 9.5%, and 13% at one week, one month, and 3 months, respectively. However, among non-diabetic groups, endothelial cell density reduced by 2.1%, 4.7%, 5.2%, and 11.7% at post-operative day 1, one week, one month, and 3 months, respectively.

Table No.15 : Comparison of median change in endothelial cell density between the groups

Phase of study	Percentage change (median) of endothelial cell density		P value
	Diabetic	Non-diabetic	
Day 1	5.9	2.1	0.738
One week	6.4	4.7	0.865
One month	9.5	5.2	0.593
3 months	13	11.7	0.375

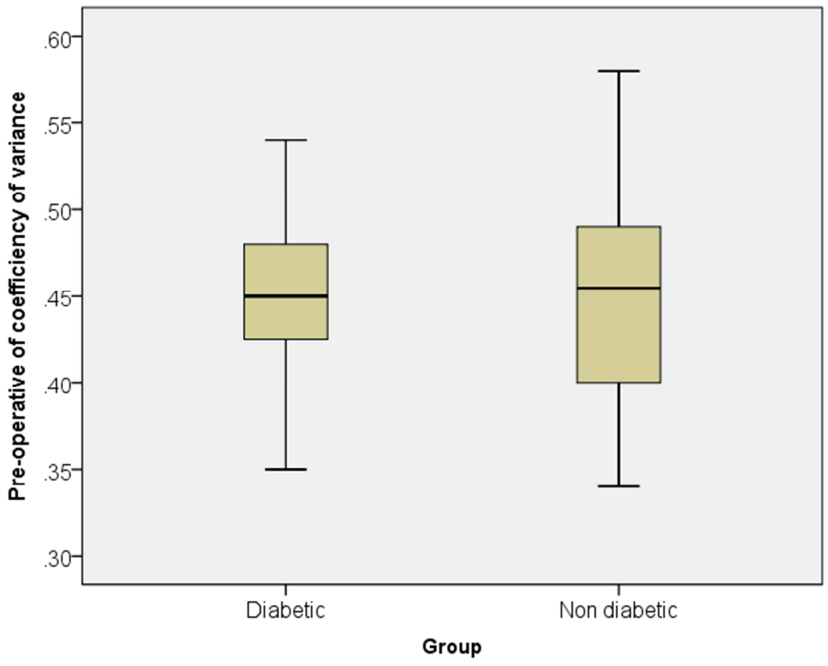
The study noted that there was no significant difference in change in endothelial cell density at post-operative day one (5.2% in diabetes vs 0.09% in non-diabetes, p=0.630), one week (1.4 in diabetes vs 3.1% in non-diabetes, p-0.851), one month (9.5% in diabetes vs 4.8% in non-diabetics, p=0.391) and 3 months (6.3% in diabetes vs 8.2% in non-diabetes, p=0.445) between the groups.

CO-EFFICIENT OF VARIANCE

Table No.16 : Comparison of pre-operative co-efficient of variance between the groups

Co-efficient of variance	Diabetic	Non-diabetic	P value
Mean±SD	0.45±0.04	0.45±0.05	0.870
Range	0.35-0.54	0.34-0.58	

Graph 9: Boxplot comparing mean pre-operative endothelial cell density between the groups

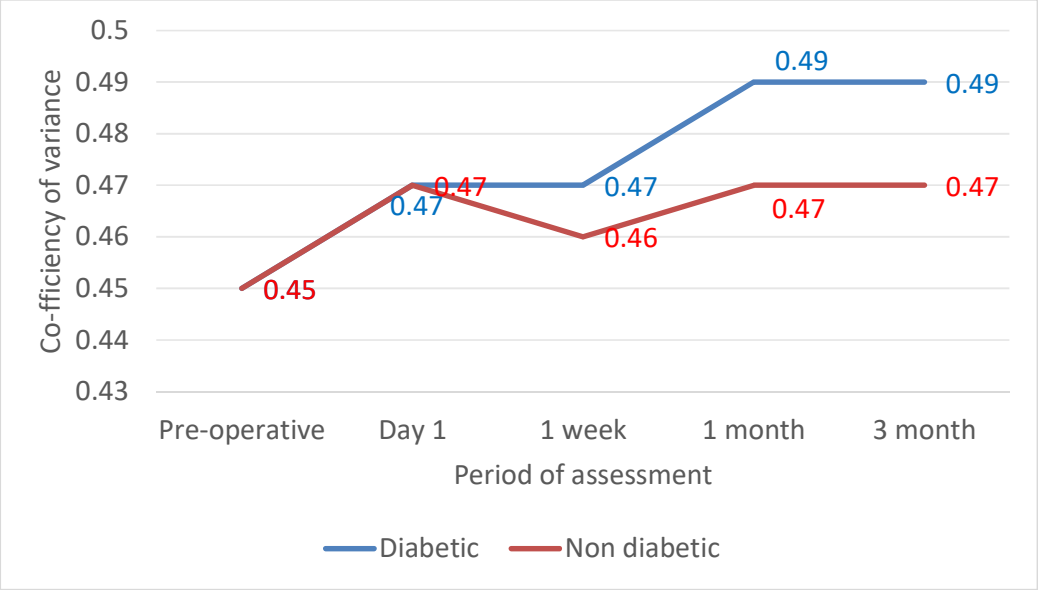


The mean pre-operative co-efficiency of variance was similar among patients with diabetes (0.45±0.04) and without diabetes (0.45±0.05).

Table No.17 : Comparison of Co-efficiency of variance across the study period between the groups

Co-efficiency of variance	Pre-operative	Post-operative			
	Pre-op	Day 1	1 week	1 month	3 months
Diabetic					
Mean±SD	0.45±0.04	0.47±0.06	0.47±0.06	0.49±0.08	0.49±0.09
Range	0.35 - 0.54	0.37 - 0.84	0.40 - 0.72	0.36 - 0.85	0.36 - 0.86
Non diabetic					
Mean±SD	0.45±0.05	0.46±0.07	0.46±0.04	0.47±0.06	0.47±0.06
Range	0.34 - 0.58	0.36 - 0.60	0.39 - 0.59	0.34 - 0.72	0.39 - 0.61

Graph 10: Comparison of Co-efficiency of variance between the groups

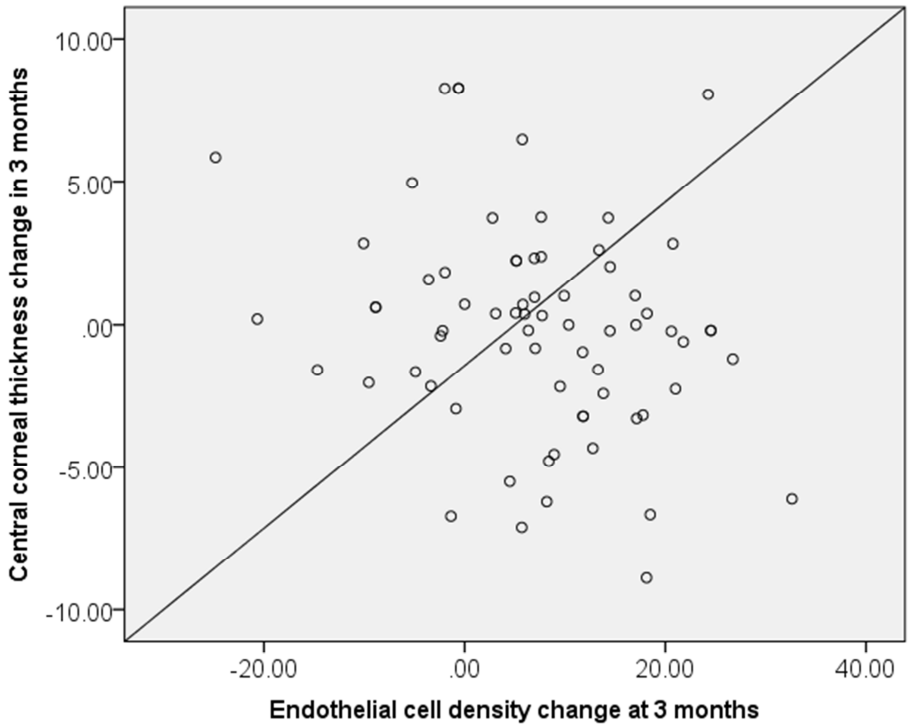


CORRELATION

Table No.18 : Correlation between change in endothelial cell density loss and reduction of central corneal thickness

Phase	Correlation	P value	Inference
Pre-operative (Baseline)	0.416	0.000	Positive, moderate, and significant
Day 1 (% change)	0.008	0.947	Positive, negligible, and non-significant
One week (% change)	0.288	0.016	Positive, low, but significant
One month (% change)	0.196	0.104	Positive, Negligible, and non-significant
3 months (% change)	-0.071	0.558	Negative, low, and non-significant

Graph 11: Correlation between change in corneal thickness and endothelial cell change at 3 months of follow up



The study showed positive, negligible, and non-significant correlation between changes in central corneal thickness and endothelial cell density at day 1 of post-operative period (rho=0.008, p=0.947). However, this correlation was positive, low, and significant at 1 month of follow up (rho=0.288, p=0.016). At 3 months of follow up, there was negative, negligible, and non-significant correlation between the two parameters (p=-0.071, p=0.558).

DISCUSSION

The present study aimed to evaluate the effects of Manual Small Incision Cataract Surgery (MSICS) on corneal endothelial health and central corneal thickness (CCT) in diabetic and non-diabetic patients. Cataract surgery is a well-established intervention for vision restoration. However, there are concerns regarding its impact on corneal endothelial integrity, particularly in individuals with diabetes.[55-57] The discussion section elaborates on key findings, their implications, and comparisons with existing literature. It also discusses on the future implications of the result.

The study included 70 participants, equally distributed into diabetic and non-diabetic groups. The mean age of the participants was 64.1±7.0 years, with no statistically significant difference between the two groups. Males constituted the majority (55.7%), and the gender distribution was comparable across groups. A significant proportion of participants were housewives, businessmen, and farmers, indicating the effect of diabetes and cataract across different occupational group. The comparable demographic characteristics between the diabetic and non-diabetic groups ensure homogeneity between the group, and thereby reduces their effect on the overall treatment outcome. The findings align with previous studies that have shown a higher prevalence of cataract development in the elderly, with diabetes being a contributing factor to early onset.[58-59] Vyas U et al in Ahmedabad noted that 44.4% of diabetes patients had early onset cataract.[59] A community-based study in Saudi Arabia by Alabdulwahhab KM et al. noted that 35% of diabetic cases had senile cataract.[60]

With 727 million elderly population worldwide constituting for 13% of world population, there is a need to focus on research on this population.[61-63] Moreover,

studies show upward projection of diabetes mellitus and cataract surgeries in the near future.[64-66]

Among the diabetic group, the mean HbA1c was 7.0±0.31%, which is indicative of well-controlled diabetes. However, this may be due to pre-operative control measures and patient compliance on course for preparation of cataract surgery. However, hypertension was significantly higher among diabetes (40%) compared to non-diabetes (2.9%). These are co-morbid conditions due to multifactorial causes, but mainly attributed to the maladaptive changes in the autonomic nervous system and vascular endothelial system, secondary to diabetes mellitus.[67-68] While studies on rat (Li X et al) have demonstrated disruption of endothelial cell barrier dysfunction and pump function, the impact of systemic hypertension on these parameters are unclear.[69] Similarly, study by Ollivier FJ et al. noted that chronic ocular hypertension (laser induced) didn't show significant change in central corneal thickness.[70]

In this study, the baseline central corneal thickness (CCT) was slightly higher in diabetic patients (508.4±36.1 µm) compared to non-diabetic patients (495.1±30.9 µm), indicative of a mean difference of 13.3µm. However, the difference was not statistically significant (p=0.104). This was similar to a previous study done by Luo XY et al[23], where CCT was 5.4 µm thicker in diabetes cases compared to non-diabetics. In contrast, a study in Singapore noted that diabetic patients had a significantly higher baseline CCT compared to non-diabetics (547.2 µm vs. 539.3 µm, mean difference of 8.9 µm, $p<0.001$).[24] Overall, studies have noted higher pre-operative CCT among diabetes patients. High advanced glycosylated end products (AGE's) deposition in the corneal collagen fibres (cross-linkages) due to chronic metabolic stress is postulated to the increased corneal thickness.[71-72]

Similarly, preoperative endothelial cell density (ECD) was slightly higher in diabetics (2383±278 cells/mm²) than non-diabetics (2342±236 cells/mm²) with no significant difference between the groups. Similarly, Kudva AA et al,[14] and Storr-Paulsen A et al[22] noted no significant difference between the group. In contrast, Pandey S et al reported decrease in endothelial cell density among diabetes patients.[21,] However, the absence of a significant baseline difference indicates that both groups were comparable in terms of corneal health prior to MSICS.

Following MSICS, a transient increase in CCT was observed in both the groups, increasing on postoperative day 1 (548.9±53.4 µm in diabetics vs. 531.3±36.6 µm in non-diabetics) which was followed by subsequent reduction towards normal. Previous studies also noted similar changes for both MSICS and phacoemulsification procedures.[14,74,75.76,77,78] This increase can be attributed to surgical trauma, inflammatory response, and corneal edema.[76] Over time, CCT gradually reduced towards preoperative levels at three months post-surgery. This is attributed to slower recovery of compromised endothelial pump (Na^+-K^+ ATPase pump).[26.79]

Endothelial cell loss is a key concern following cataract surgery, as these cells do not regenerate. The study found a progressive decline in ECD post-operatively, with a higher percentage loss in diabetics (13%) compared to non-diabetics (11.7%) at three months follow-up. Although the difference was not statistically significant, it indicates that diabetic corneas are slightly more susceptible to surgical trauma. Similarly, higher ECD loss following MSICS was noted among diabetics were noted in studies from Mangalore (27.5% in diabetics vs 18.3% in non-diabetics)[14], and Egypt[74] (21.3% in diabetics vs 6.5% in non-diabetics).

Endothelial cell loss is a well-established postoperative outcome, with previous studies reporting similar findings. The increased susceptibility of diabetic patients can be attributed to surgical trauma, chronic hyperglycemia-induced endothelial dysfunction, oxidative stress, and impaired wound healing.[80-81]

Endothelial cell loss is a permanent process due to loss of regeneration of lost cells. This is followed by compensatory mechanism of increase in size of endothelial cell to bridge the gap. This leads to change in shape and size of endothelial cells, resulting in increased co-efficient of variation in cell size.[25,80-81] In our study, an increase in the coefficient of variation was observed in both groups, indicating postoperative polymegathism. This suggests endothelial stress and compensatory mechanisms to maintain corneal clarity. While polymegathism was more pronounced in diabetics (0.45 ± 0.04 at pre-operative to 0.49+0.09 at 3 months post-op) compared to non-diabetics (0.45+0.05 pre-operative to 0.47+0.06 at 3 months post-op), the difference was not statistically significant. Steady increase in polymegathism following cataract surgery with comparative higher changes among diabetics compared to non-diabetics were also noted in previous studies.[14,82,83] Our study further strengthens these findings. In addition, decrease in percentage of hexagonal cells (suggestive of change in shape) was also noted in previous studies.[14,22,74,76]

A moderate, significant positive correlation was observed between endothelial cell loss and CCT changes at baseline, suggesting that patients with greater endothelial cell loss exhibited more significant corneal swelling. However, this correlation diminished over time. These results suggest that corneal edema resolves as endothelial function stabilizes.

The findings of this study have important clinical implications. While MSICS is a safe procedure for both diabetic and non-diabetic patients, the slight delay in recovery and greater susceptibility to endothelial cell loss in diabetics highlight the need for careful perioperative management. Preoperative assessment of endothelial health, intraoperative protective measures (e.g. use of viscoelastic substances) and close postoperative monitoring are essential in diabetic patients undergoing cataract surgery.

CONCLUSION

This study assessed the effects of Manual Small Incision Cataract Surgery (MSICS) on corneal endothelial health in diabetic and non-diabetic patients. We can conclude that both the groups experienced a gradual reduction in endothelial cell density following surgery, but no significant difference was observed between them. An increase in the polymegathism (coefficient of variation) was noted in both groups, without significant intergroup differences. This study also correlates endothelial cell changes and central corneal thickness (CCT) in diabetic and non-diabetic patients. Based on our study, we conclude that, while CCT increased during the first postoperative day, it subsequently decreased over time. However, the changes in CCT did not significantly differ between diabetic and non-diabetic patients. It is inferred that corneal edema resolves as endothelial function stabilizes. These findings indicate the While MSICS proved to be a safe and effective procedure for both diabetic and non-diabetic patients, diabetic corneas exhibited a delayed recovery and a slightly greater susceptibility to surgical trauma and stress.

SUMMARY

A one year longitudinal study was conducted in KLEs Dr. Prabhakar Kore Hospital, Belagavi among 35 diabetic patients and 35 non-diabetic patients planned for manual incision cataract with a aim of comparing corneal endothelial cell changes in diabetics and non-diabetic patients who underwent manual small incision cataract surgery. The study assessed corneal parameters like central corneal thickness, endothelial cell density, and co-efficient of variation at pre-operative, post op day 1, 1 week, 1 month, and 3 month following surgery. Following are the salient results of the study:

General characteristics: The study included 70 participants in the age range of 50 - 80 years, with mean of 64.1+7.0 years. The mean age of participants among diabetic and non-diabetic group was 63.6+6.3 years and 64.5+7.7 years, respectively.

CCT: While the pre-operative CCT was higher among diabetics compared to non-diabetics, this difference was not statistically significant (508.4$\pm$36.1μm in diabetics vs 495.1$\pm$30.9μm in non-diabetics, p=0.104). Among diabetics, post-operatively, the CCT increased at day 1 (548.9+53.4μm) and subsequently decreased over 3 months (508.6$\pm$38.0μm) towards normal. Similar results were noted among non-diabetics (increased to 531.3+36.6μm at day 1 and steadily decreased to 495.2+27.7μm at 3 months). There was no significant difference in CCT change between the groups across the study period ($p<0.05$).

Endothelial cell density (ECD): They were comparable between the group at pre-operative phase (2383$\pm$27 μm in diabetics vs 2342$\pm$236 μm, p=0.514). The ECD loss occurred in both groups with decreased in ECD over period of time. At 3 months of follow up, ECD loss was non-significantly higher among diabetics compared to non-diabetics (13% in diabetics vs 11.7% in non-diabetics, p=0.375). At 3 months of

follow up, ECD loss was non-significantly higher among diabetics compared to non-diabetics (13% in diabetics vs 11.7% in non-diabetics, p=0.375).

Co-efficient of variation: At pre-operative phase, the mean pre-operative co-efficiency of variance was similar among patients with diabetes (0.45±0.04) and without diabetes (0.45±0.05). The co-efficient of variation increased among diabetic group from 0.45±0.04 at pre-operative phase to 0.49+0.09 at 3 months post-operative phase. Similarly, among non-diabetic phase, it increased from 0.45±0.05 at pre-operative phase to 0.47±0.06 at 3 months post-operative phase.

The study concluded that, while MSICS proved to be a safe and effective procedure for both diabetic and non-diabetic patients, diabetic corneas exhibited a delayed recovery and a slightly greater susceptibility to surgical trauma and stress.

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ANNEXURE – I - INFORMED CONSENT FORM

"A one year longitudinal study to assess the corneal endothelial morphology and central corneal thickness in diabetic patients and non-diabetic patients undergoing Manual small incision cataract surgery."

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

Introduction: There is a part of eye known as cornea. This cornea is made of many layers of cells, the innermost membrane is known as endothelial cell layer. These cells help in maintaining hydration, thickness and transparency of cornea. This cell count in this layer let us know the hydration of the cornea.

We are doing this study to know the changes in this cell layer in patients with raised blood sugar levels and normal healthy subjects who are undergoing cataract surgery.

Explanation of procedure: In this study we will do regular examination where cataract will be graded and routine investigations like checking blood sugar levels, cell count on non-contact specular microscope will be done. After the proper collection of investigations and satisfactory examination, patient will undergo regular cataract surgery. Cell count will be measured by non-contact specular microscope on day 1 after surgery, and on further regular follow ups after 1 week, 1 month and 3 months later.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be part of routine investigations, thus no extra amount to be paid by the **Participant.**

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**A one year longitudinal study to assess the corneal endothelial morphology and central corneal thickness in diabetic patients and non-diabetic patients undergoing Manual small incision cataract surgery**". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE – II - PROFORMA

Hospital ID: Study ID:

NAME: _____

AGE: Years GENDER: MALE / FEMALE

ADDRESS: _____

OCCUPATION: _____

CONTACT | | | | | | | | | | NUMBER:

DATE OF EXAMINATION: / /

CHIEF COMPLAINTS:

1. DIMINUTION OF VISION: YES/ NO

   RE: DURATION _____ Days/Months/Years LE: DURATION _____ Days/Months/Years

   GRADUAL / SUDDEN WITH PAIN / PAINLESS.

IS THE PATIENT ELIGIBLE FOR STUDY? ☐ YES ☐ NO

HAS INFORMED CONSENT BEEN GIVEN? ☐ YES ☐ NO

PAST HISTORY:

1. DIABETES: PRESENT/ ABSENT

   DURATION OF DIABETES: _____ Months/Years

   ANY MEDICATION: YES /NO

2. HYPERTENSION: PRESENT/ ABSENT

3. OCULAR TRAUMA: PRESENT /ABSENT

4. SPECTACLE USE: YES/ NO IF YES, DURATION: _____

5. H/O GLAUCOMA: YES/ NO

IF YES, DURATION: _____

ANY MEDICATION: YES /NO

6. DRUG HISTORY: _____

7. H/O OCULAR SURGERY: YES / NO

OCULAR EXAMINATION:

a. **ANTERIOR SEGMENT:** RIGHT EYE

LEFT EYE

VISUAL ACUITY

PINHOLE

WTH GLASSES

NEAR VISION

WITH GLASSES

EXTRA OCULAR MOVEMENTS

EYELIDS

CONJUNCTIVA

CORNEA

ANTERIOR CHAMBER

IRIS

PUPIL

LENS

b. POSTERIOR SEGMENT: RIGHT EYE

LEFT EYE

GLOW

MEDIA

DISC

C:D

B/V

B/G

MACULA

c. **REFRACTION:**

	SPH	**CYL**	**AXIS**	**SPH**	**CYL**	**AXIS**	
V							**V**
NV							**NV**

d. INTRA OCULAR PRESSURE:

OD:

OS:

e. NASOLACRIMAL DUCT PATENCY:

OD:

OS:

f. DIAGNOSIS:

g.

DIABETIC / NON- DIABETIC							
Sr. No	Parameter	Ip/Op No.	Pre-Operative	Post Op 1 Day	Post Op 1 Week	Postop 1 Month	Post Op 3 Months
Date							
1.	CENTRAL CORNEAL THICKNESS						
2.	ENOTHELIAL CELL DENSITY						
3.	COEFFICIENT OF VARIANCE						

NOTE (IF ANY):

NAME OF PRINCIPAL INVESTIGATOR:

SIGNATURE OF PRINCIPAL INVESTIGATOR:

NAME OF GUIDE:

SIGNATURE OF GUIDE:

ANNEXURE – III – PHOTOGRAPHS

Photograph 1: Corneal endothelial morphology assessment on specular microscope

Photograph 2: Anterior segment assessment on slit lamp biomicroscope

ANNEXURE – IV

MASTER CHART

SL NO.	AGE (IN YEARS)	GENDER	OCCUPATION	DIMINUTION		HYPERTENSION (IF YES, DURATION)	DIABETES MELLITUS YES/ NO AND DURATION	HbA1C (%)	BCVA		ANTERIOR		IOP (in mmHg)		FUNDUS		DIAGNOSIS	CENTRAL CORNEAL					ENDOTHELIAL CELL DENSITY					COEFFICIENT OF VARIANCE				
				OD	OS				OD	OS	OD	OS	OD	OS	OD	OS		PREOPERATIVE	POST OP DAY 1	POST OP 1 WEEK	POST OP 1 MONTH	POST OP 3 MONTHS	PREOPERATIVE	POST OP DAY 1	POST OP 1 WEEK	POST OP 1 MONTH	POST OP 3 MONTHS	PREOPERATIVE	POST OP DAY 1	POST OP 1 WEEK	POST OP 1 MONTH	POST OP 3 MONTHS
1	70	FEMALE	HOUSEWIFE	YES	-	NO	NO	-	6/18	6/9	GREYISH	PCIOL	20.9	20.5	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	493	518	499	508	488	2279	2277	1943	2322	2053	43%	44%	46%	39%	39%
2	68	FEMALE	HOUSEWIFE	YES	YES	NO	NO	-	6/18	6/18	GREYISH	GREYISH	16.4	13.2	mormal	normal	LEFT EYE SENILE IMMATURE CATARACT	483	509	499	492	487	2200	2024	2493	2411	2110	47%	44%	39%	38%	42%
3	60	MALE	FARMER	YES	YES	NO	NO	-	6/18	CF 1/2 Mtr	GREYISH	GREYISH	16.6	11.6	SMALL DISC	SMALL DISC	LEFT EYE SENILE IMMATURE CATARACT	506	534	493	499	507	2588	2532	2297	2389	1953	40%	46%	43%	50%	47%
4	68	MALE	BUISNESSMAN	YES	YES	NO	NO		6/12	CF 1 Mtr	GREYISH	BROWN CATARACT	14	15.8	NORMAL	DETAILS NOT MADE OUT	LEFT EYE BROWN CATARACT	516	535	506	523	518	2008	2134	2277	2335	1961	46%	45%	44%	50%	42%
5	58	MALE	BUISNESSMAN	YES	NO	NO	NO		HMCF	6/12	PEARLY WHITE	PCIOL	13.4	14.2	DETAILS NOT MADE OUT	NORMAL	RIGHT EYE SENILE MATURE CATARACT	565	611	533	507	537	2255	2521	2547	2440	2143	51%	39%	41%	34%	55%
6	62	FEMALE	HOUSEWIFE	NO	YES	NO	NO		6/6	CF 2 M	PCIOL	GREYISH	15.7	19.6	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	462	496	493	465	483	2254	2092	2147	1968	2053	45%	45%	43%	47%	46%
7	67	MALE	TRADER	YES	YES	NO	NO		CF2.5 Mtr	6/60	GREYISH	GREYISH	12.2	10.4	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	485	518	518	534	483	2163	2294	2208	1907	2053	49%	44%	42%	52%	46%
8	58	MALE	BUISNESSMAN	NO	YES	NO	NO		6/6	CF1Mtr	PCIOL	GREYISH	12.5	13.2	NORMAL	HAZY VIEW	LEFT EYE SENILE IMMATURE CATARACT	497	535	506	505	509	2655	2687	2395	2586	2288	44%	42%	47%	45%	46%
9	58	MALE	BUISNESSMAN	NO	YES	NO	NO		6/18	6/18	GREYISH	GREYISH	13.5	13	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	536	589	543	528	534	2411	2697	2685	2214	2267	46%	42%	42%	50%	46%
10	57	FEMALE	HOUSEWIFE	YES	YES	NO	YES, 5 YEARS	6.7	6/9	6/12	GREYISH	GREYISH	19	20.8	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	531	549	543	534	564	2469	2715	2689	1907	2260	46%	48%	42%	52%	46%
11	67	FEMALE	HOUSEWIFE	YES	NO	NO	NO		6/36	6/6	GREYISH	GREYISH	17.2	16.6	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	418	471		465	438	2503	1624	2595	2354	2293	50%	72%	47%	45%	58%
12	60	MALE	FARMER	YES	YES	NO	NO		CF 1 Mtr	CF2Mtr	GREYISH	GREYISH	16.2	13.9	HYPEREMIC DISC	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	474	499	479	478	475	1920	1775	1834	1872	1642	43%	48%	50%	46%	40%
13	80	MALE	ELECTRICIAN	NO	YES	NO	NO		6/9	6/36	GREYISH	GREYISH	18.1	10.1	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	505	570	543	523	513	2835	2122	2214	2200	2004	51%	47%	50%	47%	47%
14	50	FEMALE	FARMER	NO	YES	NO	NO		6/12	6/36	GREYISH	GREYISH	13.9	17.1	NORMAL	HAZY VIEW	LEFT EYE SENILE IMMATURE CATARACT	556	598	549	550	552	2553	2967	2715	2553	2003	40%	44%	44%	47%	45%
15	71	FEMALE	FARMER	YES	NO	NO	NO		6/24	6/24	GREYISH	PCIOL	18.6	19.9	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	489	521	528	492	486	2412	2172	2214	2221	2015	35%	40%	44%	49%	41%
16	52	MALE	FARMER	YES	YES	NO	NO		6/12	6/18	GREYISH	GREYISH	17	15.6	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	539	556	549	530	528	2718	2997	2715	2538	2324	48%	50%	47%	51%	60%
17	70	FEMALE	BUISNESSMAN	NO	YES	NO	NO		6/9	CF 2 Mtr	PCIOL	GREYISH	13.3	19.8	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	507	509	511	577	518	2440	2418	2427	2423	2208	34%	36%	45%	49%	42%
18	72	MALE	RETIRED	YES	NO	NO	NO		HMCF	6/18	NEAR MATURE	GREYISH	19.6	18.7	DETAILS NOT MADE OUT	NORMAL	RIGHT EYE SENILE MATURE CATARACT	445	490	499	502	446	2301	2059	2019	1872	1827	49%	51%	50%	53%	46%
19	60	FEMALE	HOUSEWIFE	YES	NO	NO	NO		6/9	6/6	GREYISH	GREYISH	14.2	16.9	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	506	534	493	499	507	2588	2531	2297	2289	1953	40%	46%	43%	50%	47%
20	62	MALE	FARMER	YES	YES	NO	YES, 1 YEAR	6.9	6/18	CFCF	GREYISH	HYPERMATURE	18.8	14.1	MODERATE DIABETIC RETINOPATHY	DETAILS NOT MADE OUT	LEFT EYE HYPERMATURE CATARACT	495	540	534	532	539	2741	1613	1897	2179	2244	43%	47%	46%	49%	42%
21	70	MALE	TAILOR	YES	YES	NO	NO		6/24	6/24	GREYISH	GREYISH	17.2	16	NORMAL	NORMAL	LEFT EYE POSTERIOR SUBCAPSULAR CATARACT	494	556	538	508	462	2315	2073	2018	1834	2182	50%	46%	44%	47%	46%
22	72	MALE	RETIRED POLICE	YES	YES	NO	NO		CF1Mtr	CF2Mtr	GREYISH WHITE	GREYISH	11.8	14.3	DETAILS NOT MADE OUT	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	518	542	532	524	513	2470	1872	2179	2033	2298	46%	53%	51%	49%	47%
23	70	MALE	SHOP OWNER	YES	YES	NO	NO		6/36	6/24	GREYISH	GREYISH	14.2	16	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	479	556	493	490	461	2190	2073	2105	2090	2023	37%	46%	41%	37%	40%
24	60	MALE	SHOPKEEPER	YES	YES	NO	YES, 5 YEARS	7	CF1Mtr	CF2Mtr	PEARLY WHITE	GREYISH	10.4	15.9	DETAILS NOT MADE OUT	FEW HARD EXUDATES+	RIGHT EYE SENILE MATURE CATARACT	557	621	607	577	544	2619	2309	2360	2423	2437	48%	43%	44%	49%	45%
25	57	MALE	FARMER	YES	NO	NO	NO		CF 1/2 Mtr	6/12	GREYISH	GRADE 1 NASAL PTERYGIUM WITH PCIOL	15.2	15.2	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	485	521	519	500	506	2388	2172	2102	2579	2083	46%	60%	48%	44%	43%
26	52	FEMALE	HOUSEWIFE	YES	NO	NO	NO		C FCF	6/6	PEARLY WHITE	PCIOL	15.9	11.6	DETAILS NOT MADE OUT	NORMAL	RIGHT EYE SENILE MATURE CATARACT	523	607	585	529	528	2509	2680	2473	2378	2214	55%	44%	51%	52%	50%
27	60	FEMALE	FARMER	YES	YES	NO	NO		CF 2Mtr	CFCF	GREYISH	GREYISH	17.8	21.1	NORMAL	HAEMORRHAGE+	RIGHT EYE SENILE IMMATURE CATARACT	488	524	519	495	499	2322	2433	2312	2316	1834	39%	41%	42%	43%	54%

28	54	MALE	TAILOR	YES	YES	NO	YES, 7 YEARS	7.1	CF2Mtr	CF2Mtr	GREYISH	GREYISH	17.1	20.5	HAZY VIEW, NORMAL	HAZY VIEW, NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	498	510	513	523	514	2686	2503	2298	2335	2369	42%	40%	47%	44%	45%
29	60	FEMALE	HOUSEWIFE	YES	YES	NO	YES, 10 YERS	7.4	CF2Mtr	CF2.5Mtr	GREYISH WHITE	GREYISH	20.3	20.3	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	476	518	522	521	508	1913	2208	2368	2105	1939	52%	42%	43%	47%	47%
30	63	MALE	CLERK	NO	YES	NO	NO		6/9	CFCF	PCIOL	PEARLY WHITE	14.3	12	NORMAL	DETAILS NOT MADE OUT	LEFT EYE SENILE MATURE CATARACT	485	519	513	507	493	2204	2102	2298	2195	2104	42%	42%	45%	47%	41%
31	58	MALE	TRADER	NO	YES	NO	YES, 10 YEARS	7	6/18	CF3Mtr	GREYISH	GREYISH	17.3	20.6	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	483	469	472	530	443	2131	2223	2115	2182	2118	51%	42%	43%	52%	49%
32	68	MALE	FARMER	YES	YES	NO	YES, 1 YEAR	6.6	6/18	HMCF	GREYISH	PEARLY WHITE	12.1	9.9	NORMAL	DETAILS NOT MADE OUT	LEFT EYE SENILE MATURE CATARACT	533	550	543	528	521	2469	2143	2214	2342	2234	46%	51%	50%	48%	36%
33	68	FEMALE	HOUSEWIFE	YES	YES	NO	YES, 6 YEARS	7.4	CF 1/2 Mtr	6/18	GREYISH WHITE	GERYISH	16.3	18.6	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARCT	509	585	560	549	524	2535	2473	2599	2215	2295	47%	51%	49%	46%	49%
34	71	MALE	SHOPKEEPER	YES	NO	NO	YES, 1 YEAR	6.8	6/24	6/6	GREYISH	GREYISH	18.6	18.5	HAZY VIEW, NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	544	630	530	598	544	2759	2519	2232	2473	1592	43%	58%	52%	85%	86%
35	70	FEMALE	FARMER	YES	NO	NO	YES, 1 MONTH	7.2	HMCF	6/6	PEARLY WHITE	GREYISH	17.3	17.3	DETAILS NOT MADE OUT	NORMAL	RIGHT EYE SENILE MATURE CATARACT	483	549	534	535	484	2053	2288	2267	2121	2048	46%	58%	46%	46%	53%
36	72	MALE	SHOPKEEPER	NO	YES	YES	YES, 8 YEARS	6.9	6/6	CF 1/2 Mtr	GREYISH	BROWN CATARACT	14.6	12.2	NORMAL	DETAILS NOT MADE OUT	LEFT EYE BROWN CATARACT	507	521	483	584	505	2449	2186	2118	2098	2004	36%	50%	51%	53%	51%
37	80	MALE	RETIRED	YES	YES	NO	NO		CFCF	HMCF	GREYISH WHITE	PEARLY WHITE	18.1	13.8	DETAILS NOT MADE OUT	DETAILS NOT MADE OUT	LEFT EYE SENILE MATURE CATARACT	487	495	499	492	491	2060	2774	1881	1789	1915	58%	54%	51%	49%	60%
38	52	FEMALE	HOMEMAKER	NO	YES	NO	YES, 4 YEARS	7.2	6/9	6/18	GREYISH	GREYISH	21.1	20.7	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	506	573	578	564	504	2327	2221	2263	2341	2255	42%	61%	42%	40%	43%
39	59	FEMALE	HOUSEWIFE	NO	YES	YES	YES, 10 YEARS	7.5	6/9	6/12	PCIOL	GREYISH	17.3	20.6	MODERATE NPDR	MODERATE NPDR	LEFT EYE SENILE IMMATURE CATARACT	493	532	504	524	479	2650	2298	2255	2315	2100	35%	50%	43%	49%	61%
40	63	FEMALE	FARMER	YES	NO	YES	NO		6/36	6/12	GREYISH	GREYISH	11.3	11.2	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	418	463	484	488	423	2503	2411	1836	1836	1834	48%	47%	47%	52%	54%
41	70	MALE	RETIRED	YES	YES	NO	YES, 1 YEAR	6.5	6/12	6/36	PCIOL	GREYISH WHITE	17.3	17.3	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	620	651	630	681	618	2475	2646	2519	2222	2284	42%	47%	58%	67%	40%
42	65	MALE	BUISNESSMAN	YES	YES	NO	YES, 4-5 MONTHS	6.5	6/24	6/24	GREYISH	GREYISH	14.6	13.4	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	523	562	514	578	555	2719	2341	2270	2263	1832	44%	40%	41%	42%	48%
43	70	FEMALE	HOUSEWIFE	YES	YES	NO	YES, 10-15 YEARS	6.9	6/18	CF 3 Mtr	GREYISH	GREYISH	14.2	16.6	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	499	532	521	484	498	1834	2244	2247	2236	2213	54%	40%	40%	47%	48%
44	80	MALE	BUISNESSMAN	NO	YES	NO	YES, 6 MONTHS	7	6/36	HMCF	GREYISH	PEARLY WHITE	18.3	18.7	NORMAL	DETAILS NOT MADE OUT	LEFT EYE SENILE MATURE CATARACT	496	530	532	521	483	2427	2282	2271	2186	2102	45%	52%	43%	50%	43%
45	60	FEMALE	HOUSEWIFE	NO	YES	NO	NO		6/9	6/60	GREYISH	GREYISH	14.5	18.1	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	473	505	492	483	488	2233	2133	2178	2053	1836	45%	44%	44%	46%	52%
46	75	MALE	RETIRED	YES	YES	NO	NO		6/18	CF 3 Mtr	GREYISH	GREYISH	8.7	7.3	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	515	552	527	514	516	2094	2222	1988	2094	1961	43%	50%	41%	45%	46%
47	65	FEMALE	HOUSEWIFE	YES	YES	YES, 10 YEARS	YES, 10 YEARS	6.7	6/12	CF 1 Mtr	GREYISH	GREYISH	20.7	22.5	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	492	552	533	505	502	2178	2553	2547	2004	2386	44%	45%	41%	51%	40%
48	63	MALE	BUISNESSMAN	YES	YES	YES, 4-5 MONTHS	YES, 3 YEARS	7.4	6/24	CF 1.5 Mtr	GREYISH	GREYISH WHITE	14.6	14.6	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	456	514	521	539	443	1936	2670	2447	2663	2131	49%	41%	47%	48%	49%
49	70	MALE	RETIRED	YES	YES	NO	NO		CF 1/2 Mtr	CF2Mtr	GREYISH	GREYISH	16.1	17.2	DETAILS NOT MADE OUT	GRADE 1 HYPERTENSIVE RETINOPATHY	RIGHT EYE SENILE IMMATURE CATARACT	489	521	528	492	486	2481	2172	2214	2221	2015	35%	40%	44%	49%	47%
50	65	MALE	SHOPKEEPER	NO	YES	YES	YES, 4 YEARS	7.2	6/12	CFCF	PCIOL	GREYISH	14	9.8	NORMAL	DETAILS NOT MADE OUT	LEFT EYE SENILE IMMATURE CATARACT	446	469	512	501	453	1993	2322	2071	2397	1728	40%	37%	53%	48%	50%
51	67	FEMALE	HOUSEWIFE	YES	NO	YES	YES, 15 YEARS	7.4	CF 1 Mtr	6/12	GREYISH	GERYISH	13.8	17.7	HAZY VIEW	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	454	480	460	478	479	2199	2894	2439	1971	2100	48%	37%	42%	54%	61%
52	60	FEMALE	HOUSEWIFE	YES	NO	NO	NO		6/36	6/6	GREYISH	GREYISH	18.1	14.4	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	509	542	508	493	490	2502	2594	1939	2501	2144	46%	40%	47%	41%	46%
53	59	FEMALE	HOUSEWIFE	YES	YES	NO	NO		CF 2.5Mtr	CF2 Mtr	GREYISH	GREYISH	15.1	16.4	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	504	524	513	498	496	2336	2315	2071	2213	2255	43%	49%	53%	48%	42%
54	65	FEMALE	HOUSEWIFE	YES	NO	YES, 1 YEAR	YES, 2 YEARS	7.2	6/36	6/24	GREYISH	GREYISH	21.2	16.9	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	492	534	539	528	527	2108	2532	2718	2214	1988	45%	46%	48%	50%	41%
55	72	MALE	RETIRED	YES	NO	NO	NO		6/24	6/18	GREYISH	PCIOL	10.5	9.1	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	488	491	481	471	483	1836	1915	1921	1624	1524	52%	60%	59%	72%	61%
56	71	MALE	RETIRED	NO	YES	NO	NO		6/24	6/18	CORNEAL OPACITY	GREYISH	ERROR	12.5	DETAILS NOT MADE OUT	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	488	499	509	491	479	1872	1834	2024	1971	1832	52%	54%	47%	50%	50%
57	60	FEMALE	HOUSEWIFE	YES	YES	YES, 2-3 YEARS	YES, 7 YEARS	7.6	6/9	CF1MT	GREYISH	GREYISH	21.5	20.3	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	544	602	507	495	499	2456	1650	2252	2407	2092	45%	60%	50%	44%	46%
58	53	FEMALE	HOUSEWIFE	YES	YES	NO	NO		6/12	CF1.5Mtr	GREYISH	GREYISH	21.7	17.6	NORMAL	NORMAL	LEFT EYE SENILE IMMATURE CATARACT	506	586	501	46	509	2588	2335	2377	2501	2024	40%	41%	48%	39%	44%

59	68	MALE	TRADER	YES	NO	NO	YES, 10 YEARS	7.5	CF 3 Mtr	6/24	GREYISH	GREYISH	16	17	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	510	548	543	547	521	2255	2444	2685	2255	2115	51%	44%	48%	55%	50%
60	62	MALE	FARMER	YES	YES	NO	YES, 1 YEAR	6.7	6/18	CFCF	GREYISH	BROWN CATARACT	18.8	14.1	NORMAL	DETAILS NOT MADE OUT	LEFT EYE HYPERMATURE CATARACT	495	540	535	521	528	2741	1613	2271	2247	2234	43%	114%	43%	40%	48%
61	68	MALE	SHOPOWNER	YES	YES	YES, 7 YEARS	YES, 12 YEARS	7.1	6/24	6/18	GREYISH	GREYISH	14	11.3	GRADE 1 HYPERTENSIVE RETINOPATHY	GRADE 1 HYPERTENSIVE RETINOPATHY	RIGHT EYE SENILE IMMATURE CATARACT WITH GRADE 1 HYPERTENSIVE RETINOPATHY	521	484	471	475	479	2427	1836	1624	1834	1838	40%	47%	72%	54%	48%
62	62	FEMALE	HOUSEWIFE	YES	YES	NO	YES, 8 YEARS	7.2	6/18	6/18	GREYISH	GREYISH	14.4	15.3	NORMAL	NORMAL	RIGHT EYE SENILE IMMATURE CATARACT	483	564	534	531	465	2053	2759	2267	2447	1996	46%	54%	52%	50%	59%
63	58	MALE	BUISNESSMAN	YES	YES	YES, 2-3 YEARS	YES, 6 YEARS	6.7	HMCF	6/9	PEARLY WHITE	GREYISH	17.9	20.3	DETAILS NOT MADE OUT	NORMAL	RIGHT EYE SENILE MATURE CATARACT	544	602	507	497	531	2673	1650	2252	1997	2469	46%	60%	50%	47%	46%
64	62	FEMALE	RETIRED	YES	YES	NO	YES, 5-6 YEARS	7.4	6/18	6/18	GREYISH	GREYISH	14.2	15.3	MILD NPDR	MILD NPDR	RIGHT EYE SENILE IMMATURE CATARACT WITH MILD NPDR	456	513	521	539	471	1960	2670	2447	2657	1624	50%	41%	47%	48%	72%
65	60	FEMALE	HOUSEWIFE	YES	NO	NO	YES, 8 YEARS	7.6	6/12	6/9	GREYISH	GREYISH	19.2	21	MODERATE NPDR	MODERATE NPDR	RIGHT EYE SENILE IMMATURE CATARACT WITH MODERATE NPDR	564	681	546	564	531	2469	2178	2501	1990	1978	43%	46%	40%	36%	46%
66	75	MALE	RETIRED	YES	YES	YES	YES, 10 YEARS	7.2	CF 1/2 Mtr	6/12	PEARLY WHITE	PCIOL	20.2	19.5	DETAILS NOT MADE OUT	GRADE 1 HYPERTENSIVE RETINOPATHY	RIGHT EYE SENILE MATURE CATARACT	507	521	483	584	507	2449	2186	2118	2098	2031	36%	50%	51%	53%	46%
67	55	FEMALE	SHOPKEEPER	YES	YES	NO	YES, 8-10 YEARS	6.7	6/18	CF 1/2 Mtr	GREYISH	BROWN CATARACT	17.3	8.5	MODERATE NPDR	DETAILS NOT MADE OUT	LEFT EYE BROWN CATARACT	498	510	513	523	514	2686	2503	2298	2335	2369	42%	40%	47%	44%	45%
68	55	MALE	SHOPKEEPER	NO	YES	YES	YES, 10-12 YEARS	7.1	6/12	HMCF	GREYISH	PEARLY WHITE	12.2	12.5	MODERATE NPDR	DETAILS NOT MADE OUT	LEFT EYE SENILE MATURE CATARACT	564	659	586	542	560	2759	2575	2335	2599	2394	44%	54%	41%	40%	47%
69	64	FEMALE	HOUSEWIFE	NO	YES	YES	YES, 2 YEARS	6.9	6/6	CF1Mtr	GREYISH	GERYISH WHITE	19.5	16.8	MILD NPDR	MILD NPDR	LEFT EYE SENILE IMMATURE CATARACT WITH MILD NPDR	483	469	472	530	443	2338	2223	2115	2182	2118	51%	42%	43%	52%	49%
70	56	MALE	BUISNESSMAN	NO	YES	YES	YES, 3 YEARS	7.1	6/12	6/36	GREYISH	GREYISH	13.1	14.6	MODERATE NPDR	MODERATE NPDR	LEFT EYE SENILE IMMATURE CATARACT WITH MODERATE NPDR	533	550	543	528	521	2469	2143	2214	2234	2202	46%	51%	50%	48%	36%