A ONE YEAR PROSPECTIVE STUDY TO COMPARE THE PREVALENCE OF DRY EYE DISEASE AND ASSESSMENT OF CORNEAL NERVE SENSITIVITY BETWEEN DIABETICS AND NON-DIABETICS AT KLE'S DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI.

BY

REG. NO: BK0120002

Dissertation

Submitted to the

KLE Academy of Higher Education and Research, Belagavi, Karnataka

In partial fulfilment

Of the requirements of the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELAGAVI, KARNATAKA

JUNE/JULY 2023

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

BELAGAVI, KARNATAKA

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"A ONE YEAR PROSPECTIVE STUDY TO COMPARE THE PREVALENCE OF DRY EYE DISEASE AND ASSESSMENT OF CORNEAL NERVE SENSITIVITY BETWEEN DIABETICS AND NON-DIABETICS AT KLE'S DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"** is a bonafide and genuine research work carried out by **REG. NO: BK0120002**

Dr. SHIVANAND C. BUBANALE.
MS (OPHTHALMOLOGY) F.I.G.O
Professor & Head of Department
Department of Ophthalmology
J.N. Medical College,
Nehru Nagar,
Belagavi -590010

Date: 27-12-2022
Place: Belagavi

PROFESSOR & HEAD
Dept. of Ophthalmology
Jawaharlal Nehru Medical College,
BELGAUM

Dr. (Mrs) N.S. MAHANTSHETTI M.D.
Principal
Nehru Nagar,
Belagavi -590010

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

Date: 27.12.2022
Place: Belagavi

ii

Undertaking

"I, **REG. NO: BK0120002,** hereby declare that the information and the data mentioned in my dissertation entitled **""A ONE YEAR PROSPECTIVE STUDY TO COMPARE THE PREVALENCE OF DRY EYE DISEASE AND ASSESSMENT OF CORNEAL NERVE SENSITIVITY BETWEEN DIABETICS AND NON-DIABETICS AT KLE'S DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"** belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original-one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University".

Date: 27. 12. 2022 **REG. NO: BK0120002**

Place: Belagavi

PLAGIARISM CERTIFICATE

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(Recognized by Medical Council of India, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MHRD (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 13-12-2022.

ACCEPTANCE LETTER

The softcopy of thesis entitled: **" ONE YEAR PROSPECTIVE STUDY TO COMPARE THE PREVALENCE OF DRY EYE DISEASE AND ASSESSMENT OF CORNEAL NERVE SENSITIVITY BETWEEN DIABETICS AND NON DIABETICS AT KLE'S DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI "** has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **08%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER * BELAGAVI * J. N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BK0120002.
Postgraduate Student,
2020-21 Batch,
Department of Ophthalmology,
J. N. Medical College, Belagavi.

ABBREVIATIONS

Dry Eye Disease	=	DED
Diabetes Mellitus	=	DM
Tear Film and Ocular Surface Society	=	TFOS
Dry Eye Workshop	=	DEWS
Mucins	=	MUC
Lacrimal Functional Unit	=	LFU
Vascular Endothelial Growth Factors	=	VEGF
Mitogen Activated Protein Kinases	=	MAPK
Tumor Necrosis Factor	=	TNF
Interleukin	=	IL
Matrix Metallo Proteinase	=	MMP
Ocular Dryness and Disease Severity	=	ODISSEY
Japan Dry Eye Society	=	JDES
Asia Dry Eye Society	=	ADES
Ocular Surface Disease Index questionnaire	=	OSDI
Standard Patient Evaluation of Eye Dryness Questionnaire	=	SPEED
Impact of Dry Eye in Everyday Life	=	IDEEL
Tear Film Breakup Time	=	TBUT

Milli osmoles	=	mOsm
Sjogren's Syndrome	=	SS
Rheumatoid Factor	=	RF
Anti nuclear antibodies	=	ANA
Hyaluronic Acid	=	HA
Flurometha lone	=	FML
Lymphocyte Function-Associated antigen	=	LFA
Intercellular adhesion molecule	=	ICAM
Essential Fatty Acid	=	EFA
Transforming growth factor	=	TGF
Immunoglobulin A	=	IgA
Epithelial Growth Factor	=	EGF
Autologous Blood Serum	=	ABS
World Health Organization	=	WHO
Impaired glucose tolerance	=	IGT
Diabetic Retinopathy	=	DR
Non Proliferative Diabetic Retinopathy	=	NPDR
Proliferative Diabetic Retinopathy	=	PDR
Diabetic Macular Edema	=	DME
Advanced glycation end products	=	AGE's

Early treatment Diabetic Retinopathy Study	=	ETDRS
Neovascularization of the disc	=	NVD
Neovascularization elsewhere	=	NVE
Diabetic neuropathy	=	DN
Reactive oxygen species	=	ROS
Dry eye syndrome	=	DES
Insulin-like Growth Factor Binding Protein	=	IGFBP

ABSTRACT

Primary objective:

To compare the prevalence of dry eye disease and to assess corneal nerve sensitivity in diabetic patients in comparison to non-diabetic patients.

Secondary objective:

To study associations of dry eye disease in diabetic patients with diabetic retinopathy in comparison to patients without diabetic retinopathy.

Methodology:

The present study was a prospective comparative study done from 1st January 2021 to 31st December 2021 in KLE'S Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Considering the prevalence of Dry Eye Disease to be 54.3%, with a percentage of maximum errors as 5% at 95% confidence level, the sample size was calculated to be 381, but for the ease of calculations the sample size taken was 400.

All patients, attending the Ophthalmology OPD, meeting the inclusion and exclusion criteria were enrolled in the study after taking informed and written consent. All the demographic data was noted in a predesigned proforma.

The patients eligible for the study were divided into 2 groups,

Group A: Diabetic individuals.

Group B: Non-diabetic individuals.

A detailed clinical history including symptoms of DED, duration of diabetes etc were elicited from all the patients enrolled in the study.

For assessing the symptoms of DED, Standard Patient Evaluation of Eye Dryness (SPEED) questionnaire was used.

Ocular examination included evaluation of visual acuity, eye lids, conjunctiva, lacrimal apparatus, cornea, anterior chamber, fundus examination.

For tear film evaluation, TBUT and Schirmer's II basal tear secretion, was done.

Corneal nerve sensitivity was tested using cotton wisp.

Results:

Considering the SPEED score, Schirmer II values, TBUT values and DEWS II diagnostic criteria, mild Dry Eye Disease was seen in 23% diabetic and 22.25% non-diabetic group, moderate Dry Eye Disease in 45.75% diabetic and 9.75% non-diabetic group and severe Dry Eye Disease in 2% diabetic and 1.75% non-diabetic group. Corneal Nerve Sensitivity was reduced more in diabetic group and Corneal Nerve Sensitivity was reduced to a greater extent in patients with higher degree of Dry Eye Disease. Moderate Dry Eye Disease was more common within all grades of Diabetic Retinopathy.

Conclusion:

Prevalence of Dry Eye Disease (DED) is more common in the patients suffering from Diabetes Mellitus (DM). Corneal Nerve Sensitivity was found to be reduced, more in the diabetic patient group than the non-diabetic patient group. Also, diabetic patients who have evidence of Diabetic Retinopathy (DR) were found to have more severe forms of ocular surface disease.

Key words: Dry eye, Diabetes, Retinopathy

TABLE OF CONTENTS

SL.NO	CONTENTS	PAGE NO.
1	INTRODUCTION	1-2
2	OBJECTIVE	3
3	REVIEW OF LITERATURE	4-53
4	MATERIALS AND METHODS	54-60
5	RESULTS	61-83
6	DISCUSSION	84-89
7	CONCLUSION	90
8	BIBLIOGRAPHY	91-101
9	ANNEXURES	
	Annexure I: Ethical Clearance Letter	102
	Annexure II: Consent form	103-106
	Annexure III: Proforma	107-111
	Annexure IV: Photographs	112-116
	Annexure V: Key to Master Chart	117
	Annexure VI: Master Chart	118-129

LIST OF FIGURES

SL NO.	FIGURE	PAGE NO.
1	Distribution of conjunctival goblet cells	6
2	Micro-structure of corneal epithelium	7
3	Lacrimal Functional Unit	8
4	Layers of the tear film	9
5	Tear film interface	11
6	Corneal innervation	12
7	Lipid layer of tear film	14
8	a) Structure of eyelid b) Apposed eyelids.	14
9	The blink cycle.	16
10	Classification of Dry Eye Disease	19
11	Pathophysiology of Dry Eye Disease	21
12	Immune response in Dry Eye Disease	21
13	TFOS DEWS II Management Protocol	28

14	Classification of tear substitutes.	32
15	Punctal plugs	34
16	WHO Classification of DM	39
17	Etiopathogenesis of Diabetes Mellitus	40
18	Effects of Hyperglycemia on Lacrimal Functional Unit.	50
19	Effect of hyperglycemia on corneal epithelium	52
20	Distribution of eyes according to dry eye grade.	75

LIST OF TABLES

SL NO.	TABLE	PAGE NO.
1	Age wise distribution of Diabetics and Non-Diabetics	61
2	Gender wise distribution of Diabetics and Non Diabetics	62
3	Distribution of Diabetics and Non-Diabetics based on Treatment of Diabetes Response to the proforma provided including the SPEED Questionnaire	63
4	Distribution of patients based on SPEED questionnaire and score	64
5	Distribution of patients based on frequency of symptoms	67
6	Distribution of patients based on severity of symptoms	68
7	Distribution of patients based on SPEED Score	69
8	Distribution of patients based on Schimer's II Test	71
9	Distribution of patients based on TBUT	72
10	Distribution of patients based on grade of DED	74
11	Distribution of Dibetics and Non Diabetics with respect to DED	76
12	Distribution of patients based on grade of DR	78
13	Distribution of patients with different grades of DR over severity of	80

	DED	
14	Distribution of CNS with respect to Diabetics and Non Diabetics	82
15	Distribution of CNS with respect to severity of DED	83

LIST OF GRAPHS

SL NO.	GRAPH	PAGE NO.
1	Distribution of gender over groups.	62
2	Distribution of Grittiness over groups.	65
3	Distribution of Burning over groups.	65
4	Distribution of Irritation over groups.	66
5	Distribution of Eye fatigue over groups	66
6	Distribution of frequency over groups.	67
7	Distribution of severity over groups.	68
8	Distribution of Dry eye based on speed score over groups.	70
9	Distribution of Dry eye based on Schirmer's test result over groups.	71
10	Distribution of Dry eye based on TBUT result over groups	73
11	Distribution of Dry eye grade over groups.	77
12	Distribution of subjects in diabetic group with respect to ETDRS Grade.	79

13	Distribution of grade of diabetic retinopathy according to severity of dry eye in diabetic patients who had dry eye.	81
14	Distribution of CNS with dry eye.	83
15	Mean plot of SPEED score over groups.	85
16	Mean plot of Schirmer's test result over groups.	86
17	Mean plot of TBUT test result over groups.	87

LIST OF PHOTOS

SL NO.	PHOTOS	PAGE NO.
1	Patient undergoing Schirmer's II Test	112
2	Schirmer's II test	112
3	Schirmer's II Test showing severe Dry Eye Disease	113
4	Slit lamp examination in cobalt blue filter to assess TBUT	113
5	TBUT under cobalt blue filter	114
6	Assessment of corneal nerve sensitivity using cotton wisp	114
7	Moderate NPDR	115
7	Severe NPDR	115
8	PDR	116
9	CSME	116

INTRODUCTION

Dry eye disease (DED) is a multifactorial ocular surface disorder, caused due to a vicious cycle of tear film instability, hyperosmolarity and chronic inflammation. It leads to serious ocular discomfort due to irritation and visual disturbances leading to reduced quality of life and decreased efficiency at work.[1] Globally, around 5-34% of population has been diagnosed with some form of dry eye.[2] Although epidemiological information on the prevalence of dry eye disease in India is limited due to broad variability in its definition and lack of a single diagnostic gold standard test, studies in India have shown a prevalence of 18.4% to 54.3% across various geographical domains.[3] Dry eye disease started as a disease affecting mainly the geriatric population, but in the past few years it is found to be seen amongst the younger population as well.

There are multiple etiological factors associated with dry eye disease, Diabetes Mellitus (DM) being one of the veiled co-morbidities. The prevalence of DM has been steadily on the rise since the past few decades. International Diabetes Federation gave an estimate that nearly 500 million people are diagnosed with DM at present.[4] Owing to our ethnicity, Indians are at an increased risk of developing DM in comparison to other groups. In 2019 it was projected that approximately 77 million Indians were living with diabetes, the prevalence being 8.9% among adults.[5]

One of the consequences of chronic DM is autonomic neuropathy which occurs as a part of diabetic neuropathy.[6] The clinical manifestations associated with diabetes with respect to DED are possibly due to exocrine dysfunction of lacrimal gland, decreased sensitivity to stimulatory signals or abnormality in the production of tears proteins. Recent studies have shown that insulin resistance or deficiency and

long-standing hyperglycaemia can lead to a reduction in the meibomian gland epithelial cells and goblet cells resulting in a deficient tear film.[7]

Abnormalities in corneal nerve structure and function has been noted in chronic diabetics. The corneal nerves undergo changes such as tortuosity and decrease in the corneal nerve density as seen in various animal-based studies.[8] These structural anomalies lead to decreased corneal nerve sensitivity. Reduced corneal sensitivity thereby causes a reduction in blink rate which thus leads to excess evaporation of the tear film further increasing the ocular discomfort. By simply extrapolating the data from previous studies, it is possible that the severity of DED and the decreased corneal nerve sensitivity will be higher in patients with severe stages of diabetic retinopathy as well.

These manifestations are a result of various complex mechanisms and their appearance simultaneous. Previous studies have given a framework to correlate the occurrence of DM and DED but there are very few studies which highlight the relation between the severity of both these entities.

This study considers the severity of diabetic retinopathy, corneal nerve sensitivity and severity of DED and aims to establish a sense of mindfulness while assessing any patient diagnosed with DM. This not only develops a practice of early diagnosis but also gives a chance to preserve the vision in the long run.

There are very few studies which evaluate the relation between DED and DR.

The precise role of tear film abnormalities and their association with DM is not well defined. Hence this study is aimed to bring to light the relationship between DED, corneal nerve sensitivity and DM and to emphasize the need to integrate dry eye evaluation in routine practice.

OBJECTIVES

Primary objective:

To compare the prevalence of dry eye disease and to assess corneal nerve sensitivity in diabetics in comparison to non-diabetics.

Secondary objective:

To study associations of dry eye disease in diabetic patients with diabetic retinopathy in comparison to patients without diabetic retinopathy.

REVIEW OF LITERATURE

The first widely accepted definition of Dry Eye Disease (DED) was published in 1995 in accordance to various clinical trials in DED from the National Eye Institute. This report defined dry eye as "Dry eye is a disorder of the tear film due to tear deficiency or excessive evaporation, which causes damage to the interpalpebral ocular surface and is associated with symptoms of ocular discomfort.''[9]

The elementary view that DED is simply a disease of the tear fluid has evolved into a rounded opinion as a disruption in the complex interaction of tear film, the ocular surface and the lacrimal apparatus. The next major development was in the year 2007, published in the report of the Tear Film and Ocular Surface Society (TFOS) of the International Dry Eye Workshop (DEWS). This report expanded the scope of DED and reinvigorated the research towards various ocular surface diseases.[10]

The TFOS DEWS II was then published online in The Ocular Surface journal.

TFOS DEWS II defines dry eye disease as the following: "Dry eye is a multi-factorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.[11]

This definition recognised loss of tear film homeostasis as an important key factor for the characteristic features of DED and neurosensory anomalies as a contributing etiology for DED. The adaptation of this definition indicates that DED is a part of a larger spectrum of disorders of the ocular surface.[10]

Anatomy of the Ocular Surface

Thoft and Friend suggested that cornea, conjunctiva, lacrimal glands, and lids function as an integrated entity known as the ocular surface. The anatomical ocular surface comprises of the mucosa that lines the globe and palpebral surfaces, the corneoscleral limbus, the corneal epithelium, and the tear film.[12]

These constituents are interconnected through a continuous epithelium, as well as the nervous, vascular, immune and endocrine systems.

- **Conjunctiva:**

The conjunctiva is a thin, transparent tissue that outlines the inner surface of the eyelids. It blends with the epithelium of the eyelid at the eyelid margin and the corneal epithelium at the limbus.[13] The conjunctival epithelium is a stratified non-keratinizing epithelium, around 5 layers in depth. It is formed of cuboidal and columnar cells, interspersed with mucus producing goblet cells. The distribution of goblet cells within the epithelium is specific to each species. In humans, the density of goblet cells is highest in the nasal region.[14]

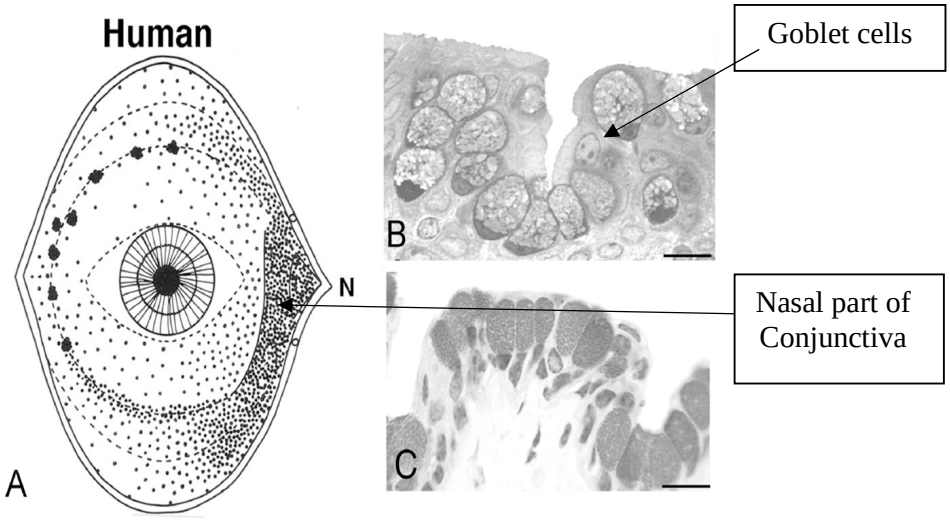


Figure 1: Distribution of conjunctival goblet cells

Structurally, goblet cells are round cells with mucin granules. The mucin granules are surrounded by membranes which have associated anchored mucin known as MUC16. This anchored mucin is said to have a role in the barrier function. The primary function of goblet cells is the secretion of mucin at the ocular surface. Therefore, specific roles attributed to goblet cells as a result of this are lubrication, maintenance of tear film across the ocular surface and its barrier effect.[14]

Reduction of goblet cells has been demonstrated in severe drying, cicatricial conditions of the ocular surface.

- **Cornea:**

Cornea is composed of 6 layers namely, epithelium, Bowmans membrane, stroma, pre- Descemet's membrane (Dua's layer), Descemet's membrane and endothelium. The corneal epithelium represents 10% of the entire thickness of the cornea.[12] It is a stratified squamous non keratinized epithelium, in which the adjacent cells are held together by desmosomes and connected to the basement membrane with

the help of hemidesmosomes.[15] The surface of the corneal epithelium consists of numerous microvilli and micro-plicae. These cells produce glycocalyx which is responsible for the adhesion and stability of the tear film.[16]

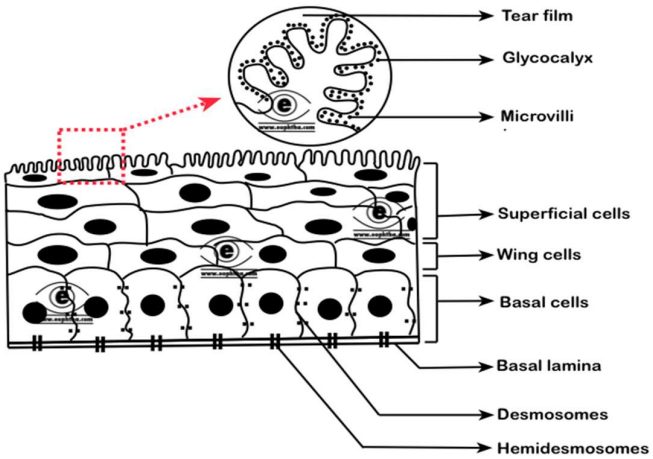


Figure 2: Micro-structure of corneal epithelium

The glycocalyx situated on the micro-plicae are large glycoproteins that cover the surface of the epithelium. They are composed of membrane associated mucins mainly MUC1 and MUC16 which are concentrated at the tip of the micro-plicae. The three main functions of these transmembrane mucins are:

1. Surface protection against friction.
2. Surface barrier against pathogens.
3. Improving epithelium wettability by converting the hydrophobic plasma membrane to a hydrophilic one through the process of glycosylation.

In eyes with ocular surface disorders, the mucin glycosylation is altered. Contact lens users, DED patients and glaucoma patients treated with beta- blockers have a decrease in sialylated mucin chains. This alteration further leads to formation of shorter and fewer mucin glycan, creating a vicious cycle.[16]

- **Lacrimal Functional Unit**

The lacrimal functional unit (LFU) is a multifaceted apparatus encompassing the lacrimal glands, ocular surface, and eyelids, including the sensory and motor nerves that link these elements. It protects layers of tear film and maintains homeostasis of ocular surface.[7] The LFU responds to environmental, endocrinologic, and cortical stimuli and makes amends in the tear film morphology. Its major function is to preserve the following:

1. Integrity of the tear film.
2. Health of the ocular surface.
3. The quality of the image cast onto the retina.

The afferent component of the LFU is facilitated through ocular surface and trigeminal nociceptors, which synapse in the brainstem with autonomic and motor (efferent) nerves. The autonomic nerves innervate the meibomian glands, conjunctival goblet cells, and lacrimal glands.[17]

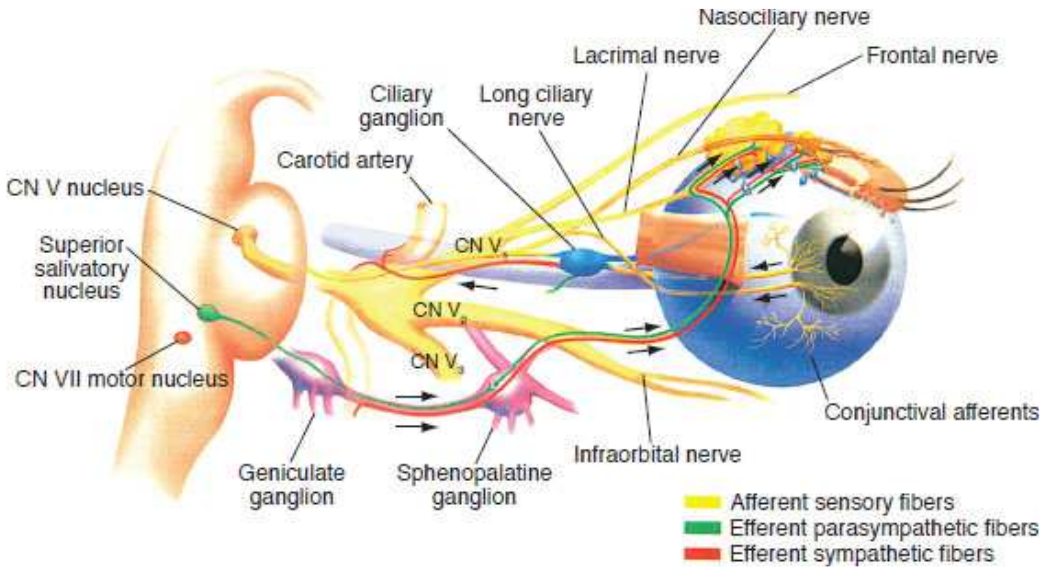


Figure 3: Lacrimal Functional Unit

- **Tear Film:**

Tear production occurs at the rate of 1.2 microlitres per minute. The total volume of tears is 6 microlitres with a turnover of 16% per minute.[18] The dynamic balance is maintained by processes like evaporation, absorption and drainage.

The precorneal tear film is not secreted as a unitary solution. Its components are secreted from various glands distributed around the eye.[19]

The tear film is composed of three layers:

1. Lipid layer: secreted by Meibomian glands
2. Aqueous layer: secreted by Lacrimal gland
3. Mucous layer: secreted by Goblet cells of conjunctiva[20]

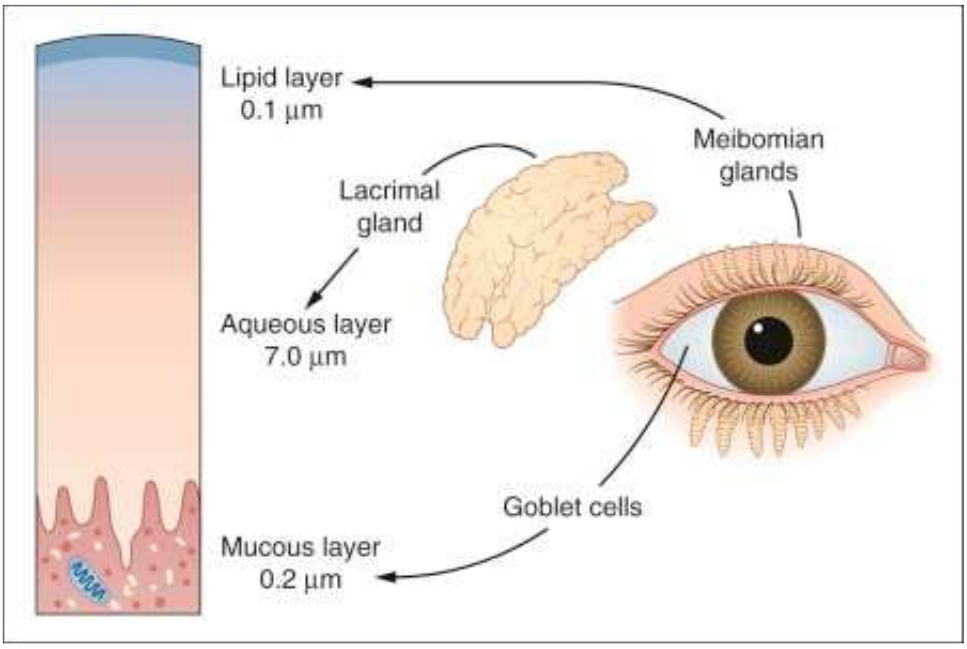


Figure 4: Layers of the tear film

The Mucin Layer:

The innermost layer of the tear film is the mucous layer which is 1.0 μm thick, made up of secreted mucins, electrolytes and water produced by the conjunctival goblet cells.[20] It is a major protective mechanism developed by the goblet cells. Mucins found on the ocular surface are produced by goblet cells, apical cells of the conjunctiva and the acinar cells of the lacrimal gland.

Mucins are glycoproteins, composed of a protein backbone added with a long chain carbohydrate compound. There are 20 different types of mucins in the human body and 7 of these are found on the ocular surface. The mucin molecules can be either membrane anchored or soluble. The membrane bound mucins include MUC1, MUC4 and MUC16 which are manufactured by the apical cells of the conjunctiva and cornea.[18] These mucins are responsible for the formation of glycocalyx that essentially protects the ocular surface. The soluble mucins are derived from 2 types of cells, the conjunctival goblet cells and the acinar cells of the lacrimal gland.[16] The goblet cells produce MUC5AC which is known as the gel producing mucin and the lacrimal acinar cells produce MUC7. The MUC5AC form bonds with the membrane anchored mucins. The other soluble mucins dissolve in the aqueous layer and function as lubricating agents.[21]

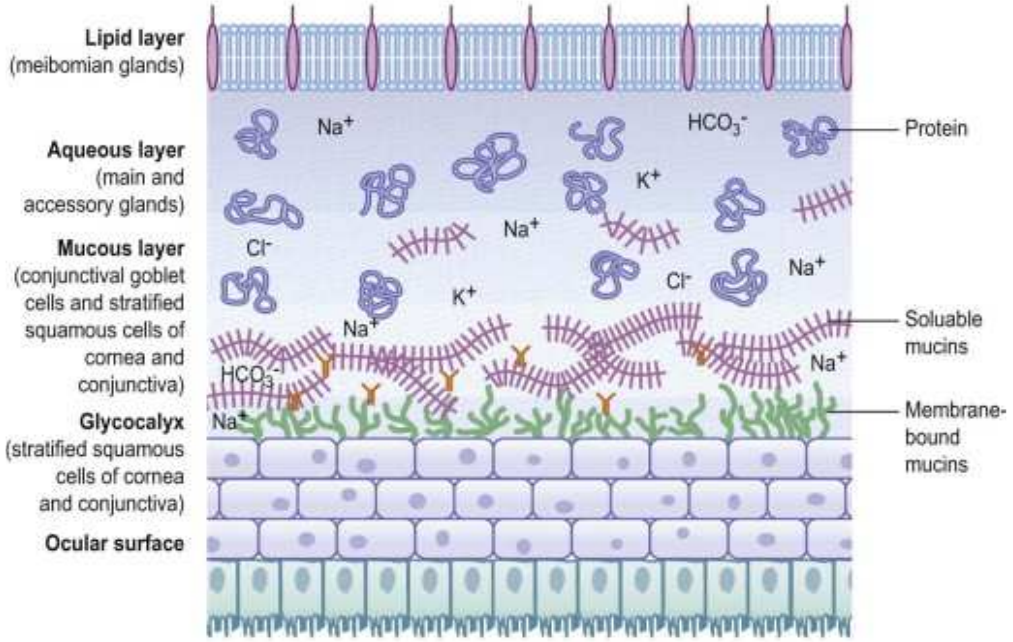


Figure 5: Tear film interface

The mucin layer also has a high carbohydrate content which makes it hydrophilic. This provides a medium for nutrients and gases to pass. Numerous studies suggest that mucins also play a role as reporters of tissue damage and serve as tear film sensors that help cells respond to a change in the tear osmolarity.[22]

The Aqueous Layer:

The aqueous layer is the most predominant layer of the tear film, about 7 μm in thickness. It is secreted by the main lacrimal gland which also secretes electrolytes and various proteins. It functions to supply oxygen to the corneal epithelium, and as protective layer. 98% of the aqueous layer is water, the other constituents being Sodium, Potassium, Chloride, Bicarbonates, Calcium, Amino acids and Vascular endothelial growth factors (VEGF).[20] Proteins in the aqueous phase include lysozymes, lactoferrin, betalysin and immunoglobulins which have a bactericidal

effect. Immunoglobulins, mainly IgA, are formed by plasma cells in the interstitial tissues of the main and accessory lacrimal gland.[23]

Aqueous layer secretion is mainly a reflex mechanism. The 5th cranial nerve – Trigeminal Nerve, forms the afferent pathway in the reflex arc, stimulation of which induces secretion. The efferent arc is more complex. The parasympathetic fibres leave the 7th cranial nerve – Facial nerve via the Greater petrosal nerve and reach the sphenopalatine ganglion.[24] The lacrimal secretory fibres then travel to the zygomaticotemporal nerve of the maxillary division of the Trigeminal nerve and combine with the lacrimal part of the ophthalmic division of the Trigeminal nerve before entering the lacrimal gland. [25]

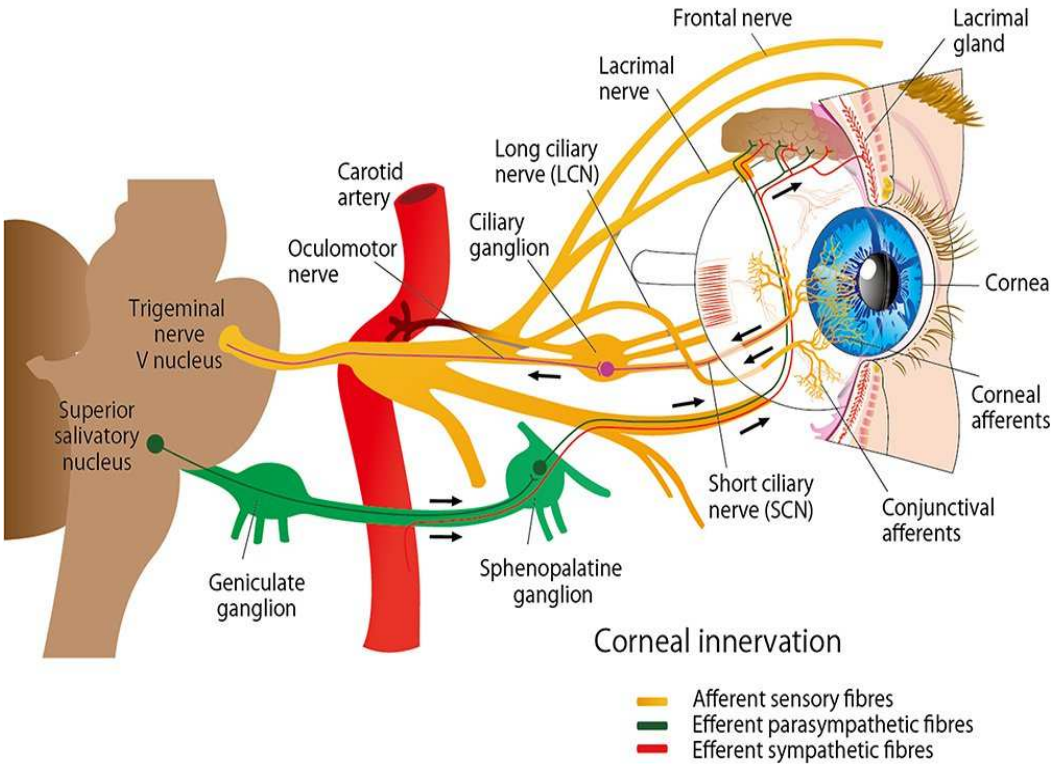


Figure 6: Corneal innervation

The lacrimal gland also receives hormonal input. Androgens play an important role in the aqueous secretion from the lacrimal gland. It not only stimulates the secretion of certain proteins but also controls the structure and morphology of the lacrimal tissue.[17]

The Lipid Layer:

The tear film lipid layer is the outermost layer of the tear film at the air-tear interface. It is composed chiefly of meibomian lipid, derived from the marginal glands of the lids. It forms a continuous sheet anteriorly, extending from upper to the lower lid. It is a relatively thin layer, 0.015 to 0.160 μm thick. [20]

Meibomian glands are tubulo-acinar glands which exude their entire contents in the process of secretion. They are embedded in the tarsal plates, about 20-30 in number. The orifices open into the skin of the lid margin. The meibomian secretions are delivered into the marginal reservoirs from where the secretions are spread onto the pre-corneal tear film with each blink.[26]

The lipid layer is composed of cholesteryl ester and waxes along with diesters, fatty acids, cholesterol and triacylglycerol. The tear film lipid layer, with its complex structure is organized in a multi-layered fashion. The interface between the lipid layer and the underlying aqueous layer is by a thin layer of polar amphiphilic lipids mainly phospholipids and glycolipids. Above these lie the bulkier non-polar hydrophobic lipids which are wax esters, cholesterol and cholesterol esters comprising 80% of the lipid layer.[27]

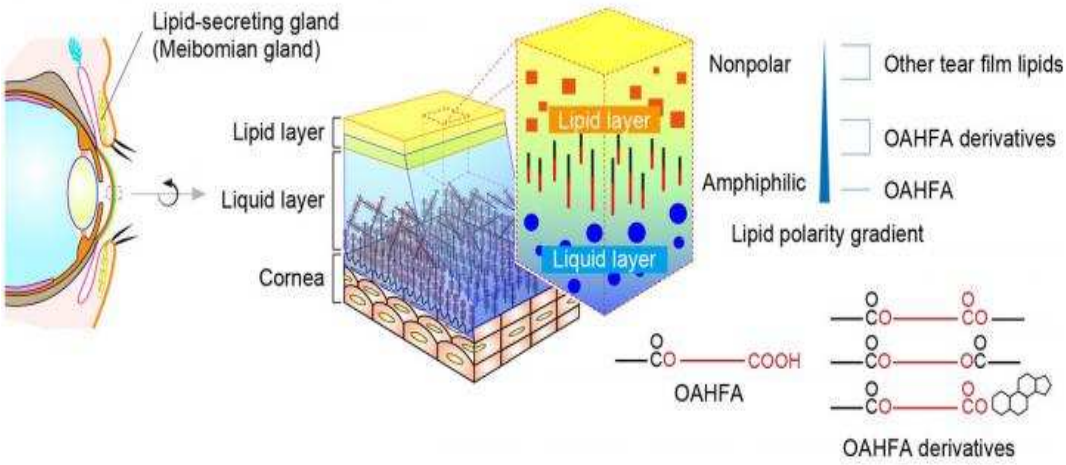


Figure 7: Lipid layer of tear film

The main function of the lipid layer is to reduce the surface tension of the tear film. The surface tension is reduced to almost 2/3rd of that of water which stabilizes the tear film. It also smoothens the surface, retards the evaporation of water and also demonstrates antimicrobial function.[28]

The lipid layer is delivered onto the tear film during the up-phase of each blink. When the lids are apposed, the upper lid draws oil from the marginal reservoirs. As the upper lid rises, the lipid film spreads over the aqueous surface.[29]

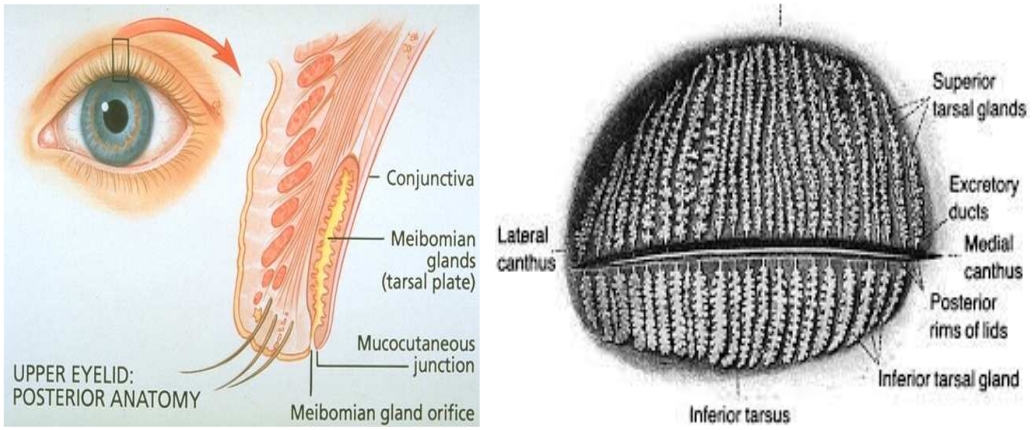


Figure 8 a) Structure of eyelid b) apposed eyelids.

Regulation of Tear Secretion:

There are 2 types of secretion, basic secretion and reflex secretion. Jones defined basic secretion as a constant, baseline secretion and reflex secretion as that caused by stimulation. The main lacrimal gland has been described as a reflex secretor and its regulation has been discussed earlier whereas the meibomian glands and goblet cells as the basic secretions.[30]

- Regulation of mucus layer:

  The goblet cells of the conjunctiva are not directly innervated unlike the other epithelial cells. However, the conjunctival stroma is innervated by sensory, sympathetic and parasympathetic nerves. The loose stroma provides a conduit for diffusion and spread of the neurotransmitters to the goblet cells. It is hypothesized that the secretion from the goblet cells is under the control of neural pathways and cAMP dependent transduction pathways.[21]

- Regulation of lipid layer:

  Meibomian glands are innervated with sympathetic and parasympathetic nerves along with the help of neuropeptides. The meibomian gland lipids are secreted by a holocrine mechanism, which means that the entire contents and the cell itself are released as a whole. Hence the regulation occurs at two levels, first the rate of synthesis of lipids can be regulated and next the maturation and disintegration of the cells storing the lipids can be regulated. These mechanisms are hormone dependent and hormonal regulation precedes the neural regulation in this case. Androgens are the main hormones in this regard.[24]

Tear Film Dynamics – Role of blinking

The blink cycle consists of 4 parts. The downstroke is the movement of the upper lid in the inferior direction, where it comes to a stop[31]. In the downstroke and following upstroke the tear film is moves rapidly causing ripples. At the turning point, the downward motion of the upper lid stops and the direction is changed to start the upstroke. After the turning point, the upstroke begins, at approximately half the speed of the downstroke. During this phase, the tear film is painted efficiently on the surface. Even when the movement of the upper lid stops, the tear film continues to move superiorly owing to the surfactant action of the lipids. As this kinesis is against gravity this phenomenon is called as "upward drift". The interblink period denotes the time frame in which the tear film flow is minimized and hence the evaporation prevails.[29]

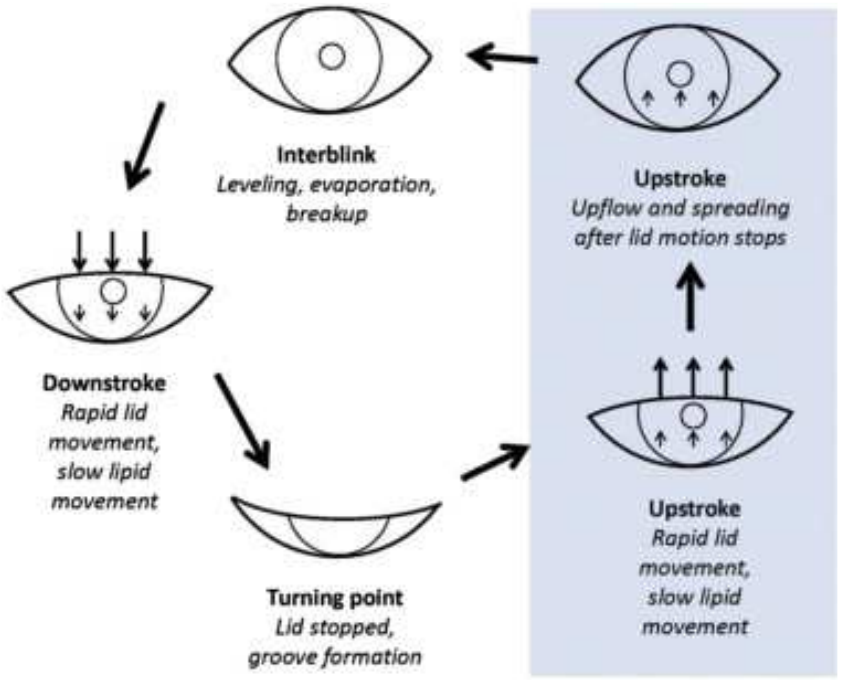


Figure 9: The blink cycle.

DRY EYE DISEASE

Dry eye disease occurs when there is flawed tear volume or function, subsequently causing an unstable tear film and ocular surface disease. It is illustrated by a vicious cycle of tear film instability, hyperosmolarity, chronic inflammation and neurosensory abnormalities which eventually lead to ocular surface damage.[9] The definition of dry eye is a constantly evolving one.

The current most widely accepted definition is given by TFOS DEWS II as : "Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles."[11]

Prevalence of Dry Eye Disease:

In various population-based studies conducted world-wide, the prevalence of dry eye disease (DED) ranges from 5% - 50%. This huge difference in the available prevalence data arises due to two main factors: (a) differences in the demographic characteristics and (b) choice of diagnostic tests and criteria used to detect dry eye.[4] The prevalence pattern of DED in India also shows a wide variation. An ideal method to estimate prevalence of a disease is to carry out hospital-based studies. An extrapolation of the data combined using hospital-based studies tends to be inaccurate with regards to the generalized population.[3]

A hospital-based study from north India reported a prevalence rate of 32%, with patients categorized into moderate and severe DED. Another study from south India reported an incidence rate of 1.46%. In contrast to these, there are other studies

from eastern regions of India, in which a lower prevalence rate has been reported. Rege et al. reported a prevalence rate of 15.4%, while Sahai et al. reported a prevalence rate of 18.4%. [32]

Risk Factors:

The most consistent risk factors associated with dry eye disease include:

1. Older age
2. Female gender, especially post-menopausal females on estrogen therapy
3. Medications – Anti-histaminic drugs
4. Connective tissue disease
5. Refractive laser surgeries
6. Vitamin A deficiency
7. Inflammatory disorders and autoimmune disorders
8. Contact lens use
9. Environmental conditions.

Classification of Dry Eye Disease:

The classification of dry eye usually applied is that of the 2007 International Dry Eye Workshop (DEWS) which divided dry eye into aqueous deficient and evaporative.[11]

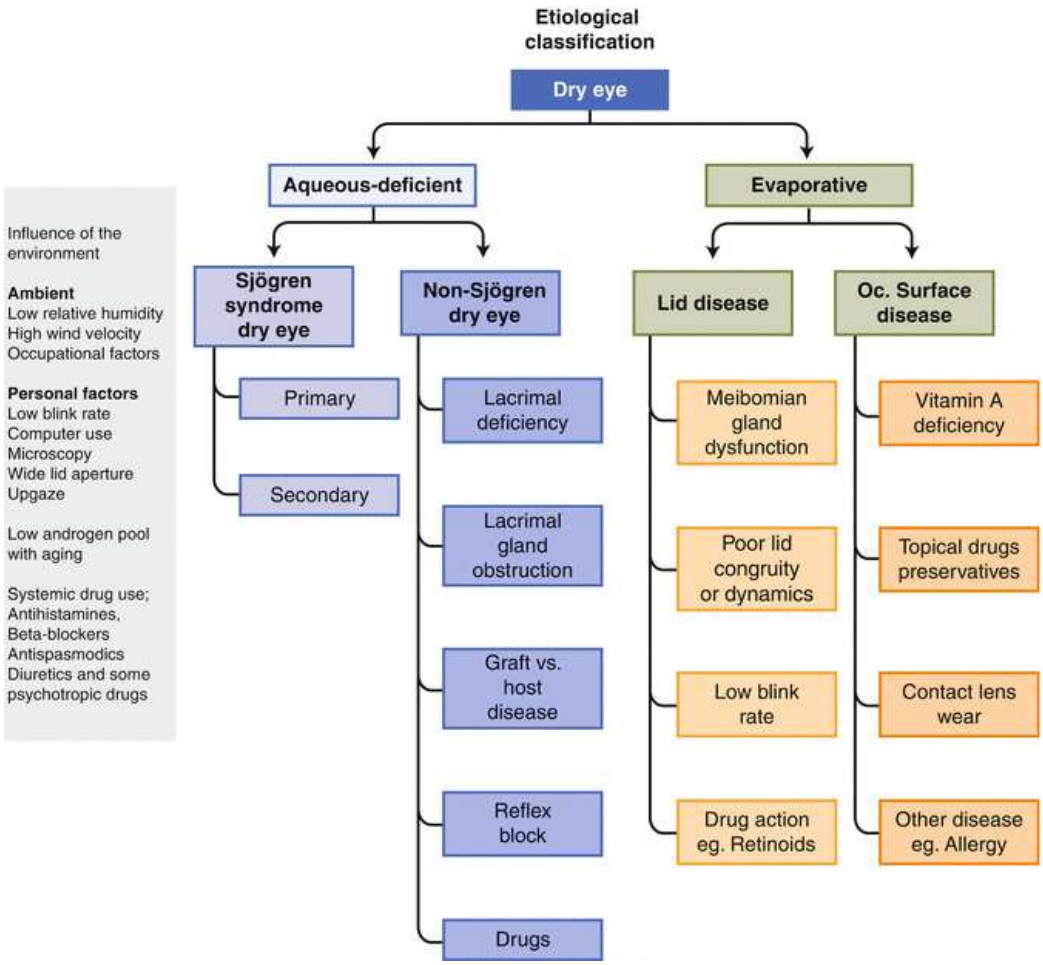


Figure 10: Classification of Dry Eye Disease

Pathophysiology of Dry Eye Disease:

There are four crucial inter-related mechanisms responsible for the manifestations of dry eye disease include, tear instability, tear hyperosmolarity, inflammation and ocular surface damage. It is characterized by a loss of homeostasis leading to a vicious cycle of tear film instability and hyperosmolarity which in turn cause damage to the ocular surface, inflammation and neurosensory abnormalities.[11,1]

Environmental conditions, contact lens wear, decreased blink frequency, and other factors predispose to evaporative type of dry eye (EDE) causing specific spots of tear film instability, in contrast, meibomian gland dysfunction–related EDE involves widespread deficiency of the tear film lipid layer.[33]

Aqueous-deficient dry eye is produced due to reduced lacrimal secretion. This could be attributable to advanced age, systemic drug use, or inflammatory infiltration or to lacrimal duct obstruction observed in cicatricial conjunctival disease and other pathology.[34]

Tear instability along with hyperosmolarity, initiated by multiple intrinsic as well as extrinsic stimuli, activates signalling pathways in the ocular surface which triggers the release of innate immunity and inflammatory molecules starting a vicious cycle. Hyperosmolarity of either eye ($\geq$ 308 mOsm/L) or an intereye difference $\geq$ 8 mOsm/L is suggestive of tear film instability.[16]

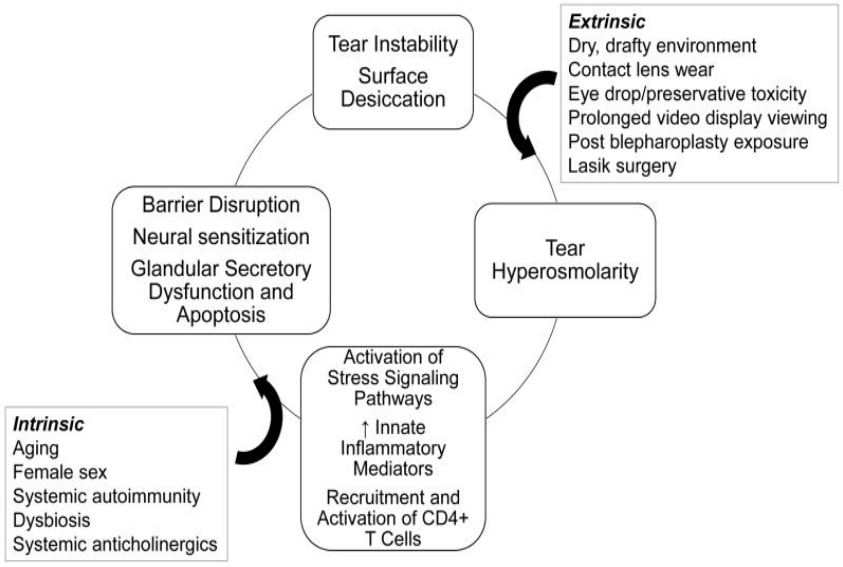


Figure 11: Pathophysiology of Dry Eye Disease

Numerous mechanisms involved in maintaining homeostasis within the ocular surface are disrupted in DED. Hyperosmolar stress has a direct pro-inflammatory effect on the ocular surface which causes activation of mitogen activated protein kinases (MAPK's) ,cytokines like IL-1β , TNF-α and IL-6, and matrix metalloproteinases (MMP-3 and MMP-9). [10]

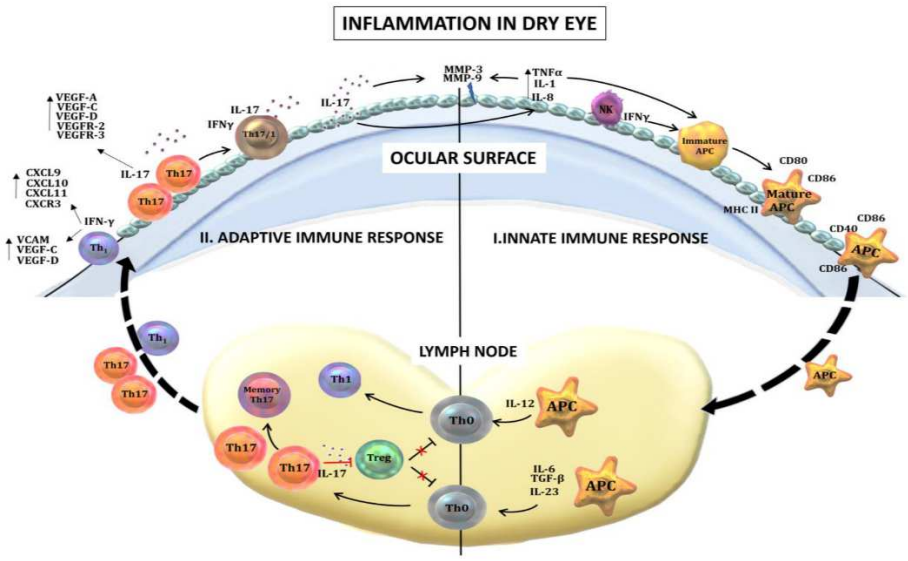


Figure 12: Immune response in Dry Eye Disease

Clinical Features of Dry Eye Disease:

Symptoms: The most common ocular symptoms associated with DED include:

- Dryness, Grittiness
- Burning sensation
- Stringy discharge
- Blurring of vision
- Redness

Signs:

- Posterior (seborrheic) blepharitis with meibomian gland dysfunction is often present
- Conjunctival congestion
- Staining of conjunctiva with rose Bengal and lissamine green
- Keratinization
- Punctate epithelial erosions on the cornea that stain well with fluorescein
- Filaments comprise of strands of mucus and debris such as shed epithelial cells and are typically attached at one end to the corneal surface. The filaments stain well with rose Bengal
- Mucous plaques with similar constituents may occur in severe dry eye. They consist of semi-transparent, white to grey, often slightly elevated lesions of varying size.

Complications:

1. **Sterile stromal ulcer:** Corneal melting can occur, which forms a typically oval, non- infiltrated ulcer. These ulcers progress quickly and often perforate.
2. **Blepahritis and Conjunctivitis:** There is an increased risk of infection due to the loss of tear film antibacterial substances.
3. **Band keratopathy**
4. **Corneal vascularization**[20]

Diagnostic Methodology:

Although various signs and symptoms have been enumerated in literature over the decades, there is a poor correlation between the symptoms appreciated by the patients and the signs present in DED. There are numerous dry eye societies across the globe, for example, Ocular Dryness and Disease Severity (ODISSEY) European Consensus Group, Japan Dry Eye Society (JDES), Asia Dry Eye Society (ADES) and all have a different diagnostic algorithm. But the one common frame work followed is correlation of symptoms and diagnostic tests to come to a conclusive diagnosis of DED.[35,36]

Symptoms are assessed in the form of questionnaires. A variety of questionnaires developed over decades are used for this purpose. The most commonly used ones include:

1. **Ocular Surface Disease Index questionnaire (OSDI):** OSDI is a 12-item questionnaire that assesses dry eye symptoms and the effects it has on visual function in the past one week of the patient's life. This questionnaire has 3 subscales: ocular symptoms, vision-related function, and environmental triggers.

Patients valuate their responses on a 0 to 4 scale with 0 corresponding to "none of the time" and 4 being equivalent to "all of the time." The final score is then estimated which spans from 0 to 100 with scores 0 to 12 indicating normal, 13 to 22 signifying mild dry eye disease, 23 to 32 representing moderate dry eye disease, and greater than 33 stands for severe dry eye disease.[37]

2. **Standard Patient Evaluation of Eye Dryness Questionnaire (SPEED):** This questionnaire gives a score from 0 to 28 which is the result of 8 items that gauge frequency and severity of symptoms. The symptoms evaluated include dryness, grittiness, scratchiness, irritation, burning, watering, soreness, and eye fatigue. The questionnaire further calculates whether these symptoms were not problematic, tolerable, uncomfortable, bothersome, or intolerable. The sensitivity and specificity of this questionnaire are 0.90 and 0.80 respectively.[37]
3. **McMonnies Questionnaire:** This instrument has 14 questions that concentrates on risk factors of dry eye. These risk factors consist of gender, age, contact lens history, dry eye symptoms, secondary symptoms, medical conditions associated with dry eye syndrome, dryness of mucous membranes, medication use, and previous dry eye treatments.[37]
4. **Other questionnaires:** Dry Eye Questionnaire, Contact Lens Dry eye questionnaire, Impact of Dry Eye in Everyday Life (IDEEL) questionnaire, Dry Eye Epidemiology Projects (DEEP) questionnaire etc.[37]

Diagnostic testing: The diagnostic tests are divided into different categories depending on the specific part being assessed.

- **Tests to assess Tear Film Stability:**

1. **Fluorescein Break Up Time**: The instillation of fluorescein dye makes this a quick and simple diagnostic procedure. The lower fornix is injected with 2% fluorescein or a strip impregnated with fluorescein and saline. The patient must maintain a fixed, unblinking gaze. With a slit lamp and a broad beam through a cobalt blue filter, the time it takes for a break in the tear film to appear in the cornea is measured. TBUT 10 seconds or more is typically accepted as typical.[38]
2. **Non- Invasive TBUT**: It has been hypothesised from studies of fluorescein dye's effects on the corneal epithelium that the dye itself may interfere with the tear film; as a result, non-invasive measurement of tear TBUT using technological advancements has gained popularity in recent years. Grid patterns and Placido disc images are used to evaluate how the image reflected from the ocular surface breaks up.[38,39]

Fluorescein break up patterns:

- ❖ Area Break: It occurs instantly, and it covers a large percentage of the cornea. According to this pattern, a severe lack of water may be present.
- ❖ Spot Break: There is an appearance of a spot-like shape immediately after eye opening with instant BUT typically in the upper part of the cornea. This pattern suggests locally impaired wettability that is correlated with deficiency of membrane-associated mucin, MUC16.
- ❖ Dimple Break: Irregular but vertical line–like shape in the central part of the cornea.

- ❖ Line Break: Patients with mild to moderate aqueous deficiency dry eye have a linear depression in the lower cornea. The average time, however, is only two or three seconds.
- ❖ Random Break: irregular, indeterminate shapes that shift form with each blink. Nonetheless, the average delay exceeds 5 seconds.[38]

- **Tests for Tear Volume Assessment:**

1. **Schirmer's Test**: A special (no.41 Whatman) filter paper measuring 5 mm in width and 35 mm in length is wetted and the amount of wetting is measured. The Schirmer 2 test, which requires an anaesthetic, measures baseline secretion, whereas the Schirmer 1 test, which does not, measures the maximal amount of secretion (basal plus reflex). The filter paper is inserted at the seam between the middle and outer thirds of the lower lid, after being folded 5 mm from one end. The patient's eyes should be gently closed. After waiting 5 minutes, the filter paper is taken out of the fold and the amount of wetness is recorded. Tear production of less than 15 mm in 5 minutes is commonly indicative of dry eye syndrome, though interpretations may vary.[20,40]
2. **Tear Meniscus Assessment**: With a Cobalt blue filter and a Slit lamp, you can see the tear meniscus floating above the lower lid margin where fluorescein dye was injected. The slit lamp beam can be used as a rough guide to estimate its height. Meniscus tear height can be measured more objectively with anterior segment OCT.[41]

- **Tear Osmolarity:** Tear osmolarity can be quickly measured with the help of the nanolitre tear lab osmometer. To get a reading, you place the device over the lower tear meniscus and the card that contains the test there over the eye. It can be used with as little as 100 nanoliters of tears, so even people with extremely dry eyes can benefit. Both eyes with an osmolarity of less than 300

mOsm/L and an inter-eye difference of less than 8 mOsm/L were considered to be within the normal range. The presence of dry eye is indicated by readings over 300 mOsm/kg. In the range of 300–320 mOsm/L, the condition is considered mild; between 320 and 340 mOsm/L, it's considered moderate; and above 340 mOsm/L, it's considered severe..[38]

- **Matrix Metalloproteinase – 9 Test:**

  Loss of ocular surface barrier function is represented by the presence of MMPs, which are inflammatory products in the tear film. Prior to any other procedures, including the injection of anaesthetic or dyes, a test for MMPs is performed. The palpebral conjunctiva is wiped clean, and a tear is taken to mix with the test buffer. After waiting for 10 minutes, the results are read in colour. For this qualitative assay, results above 40 g/ml indicate a positive result.[38]

- **Lactoferrin Test**: Lactoferrin is produced by the acinar cells of the lacrimal gland and can be detected in tears. It has antimicrobial and anti-inflammatory properties. The concentration of lactoferrin reduces in dry eye.[38]

- **Interferometry**: Reflection patterns on the tear film's surface can be used as a proxy for the film's thickness. A quantitative evaluation of lipid layer thickness is provided in colour units by the interferometer, which measures the time elapsed between blinks.. [38]

- **Meibography**: Black-and-white photography, an infrared camera, or a near-infrared charge-coupled device (CCD) video camera can be used to image illuminated everted eyelids, providing a silhouette of the meibomian gland structure. Meiboscore is one of several scoring systems that can be used to quantitatively assess meibomian gland structure.[38]

- **The Sjo Test:** The purpose of this screening is to identify Sjögren's syndrome cases at an early stage. It's a blood test used for diagnosis that can be performed right there in the doctor's office. A drop of blood is taken from the patient's fingertip, and the soaked filter paper is then sent off to the lab. Four classical SS antibodies (anti-SS-A/Ro, -B/La, rheumatoid factor [RF], and antinuclear antibody [ANA] and three tissue-specific autoantibodies make up the diagnostic testing panel (anti-SP-1, anti-PSP, and anti-CA-6). The American College of Rheumatology updated their classification criteria for SS in 2016, and anti-SSA antibody was included as the sole serological marker..[38,35]
- **Conjunctival Impression Cytology:** When it comes to researching the cornea, this is a top choice. The epithelium that lines the surface of the eye is stripped and then covered with cellulose acetate strips. Then, immunohistological and molecular tests are performed on the cells. [38]

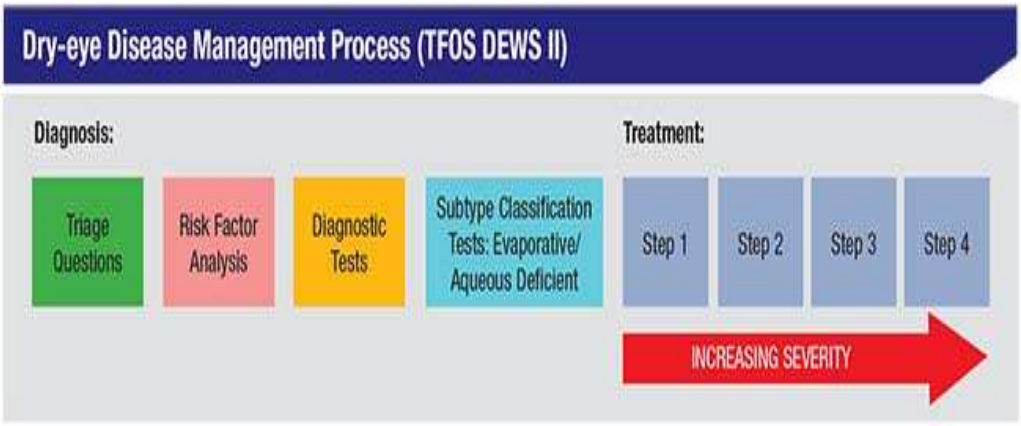


Figure 13: TFOS DEWS II Management Protocol

Treatment of Dry Eye Disease:

The ultimate goal of treatment of DED is to restore the homeostasis of the ocular surface by breaking the cycle that stimulates the disease.

TFOS DEWS II gave guidelines based on earlier International Taskforce Guidelines for Dry Eye, in which they suggested treatment options depend on the level of severity of DED graded from 1 to 4.[10]

TREATMENT OPTIONS:

- LEVEL 1:
    - Education and environmental/dietary modifications:

    This includes lifestyle review which includes the importance of blinking whilst reading, watching television or using a computer screen.

    Environmental review, e.g. increasing humidity may be possible for some environments.

    - Systemic medication review:

    This is done to evaluate the toxic side effects of various systemic medications and if possible discontinuation of the offending agent is advised.

    - Eyelid Therapy:

    When it comes to blepharitis, the bare minimum is to use warm compresses and clean your eyelids. Taping the eyelids shut before bed, sleeping with swimming goggles on, or, in severe cases, lateral tarsorrhaphy are all viable treatments for nocturnal lagophthalmos.[10]

- LEVEL 2:
    - Non-preserved tear substitutes are branded as level 2

  treatment by some authorities.
    - Anti-inflammatory agents such as topical steroids, oral omega fatty acids and other agents such as topical ciclosporin.
    - Tetracyclines (for meibomitis, rosacea).
    - Punctal plugs.
    - Secretagogues, e.g. pilocarpine, cevimeline, rebamipide.
    - Moisture chamber spectacles and spectacle side shields.
- LEVEL 3:
    - Serum eye drops. Autologous or umbilical cord serum.
    - Permanent punctal occlusion.
- LEVEL 4:
    - Systemic anti-inflammatory agents.
    - Eyelid surgery, such as tarsorrhaphy.
    - Salivary gland auto-transplantation.
    - Mucous membrane or amniotic membrane transplantation for corneal complications.[10]

Tear Substitutes:

To treat DED, patients typically use artificial tears, which can be found in the form of eyedrops, eye ointments, gels, etc.

Electrolyte solutions with various buffers and their properties are what make up tear replacements. The efficacy and tolerability of a compound are affected by its composition, type of preservative, duration of action, viscosity, osmolarity, and pH..[20]

Generations of tear substitutes:

- First-generation tear substitutes are typically saline-based hypotonic or isotonic solutions preserved with benzalkonium chloride (BAK). These have a short duration and need to be applied frequently.
- Tear replacements of the second generation feature milder preservatives or are preservative-free. These tear replacements are made from natural polymers like methylcellulose derivatives or synthetic polymers like polyvinyl alcohol or polysorbate-80. These substances are thicker in consistency and can form gels. Thus, the afford better retention, but may temporarily blur vision.
- Tear replacements of the third generation contain hyaluronic acid (HA). Natural sources of HA include the vitreous humour and other connective tissues of the eye and body. The HA salt sodium hyaluronate is a potent humectant. It does a good job of retaining moisture and halting its loss from the cornea.
- Incorporating lipid oil in water nano-emulsions, fourth-generation tear substitutes are an evolutionary leap from previous generations. They aid in restoring the tear film's lipid layer, which in turn slows tear film evaporation and strengthens the tear film as a whole.[35,42]

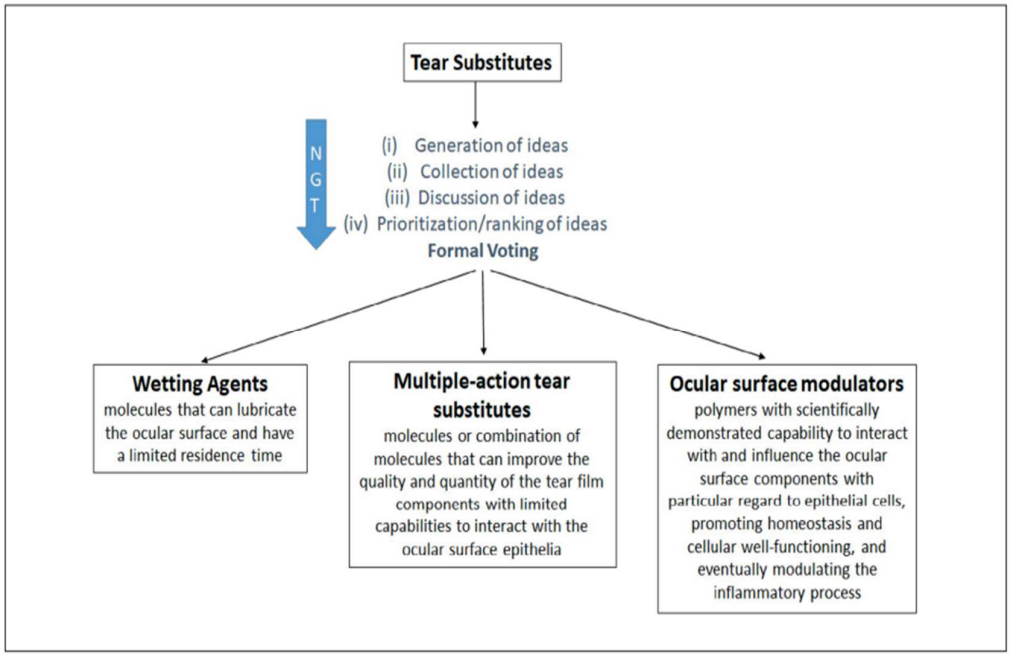


Figure 14: Classification of tear substitutes.

Punctal Occlusion:

When artificial tears don't help relieve dry eye symptoms, lacrimal occlusion is a non-pharmacological option. Punctal occlusion devices and canalicular occlusion devices have different anatomical targets.[20]

After being inserted through the punctum's conical tip, the plug's narrow neck and wide cap rest on the punctum's surface. In addition to facilitating insertion and preventing extrusion, the conical tip shape and wide cap both serve to halt any potential for distal migration.

On a slit lamp, punctal plugs are clearly visible and simple to remove. Their risk of extrusion is higher due to their superficial placement, but they are less likely to migrate through the lacrimal drainage system.

Plugs for the canaliculus are typically made of silicone and are inserted into the canaliculus in one of two orientations (vertical or horizontal). Since they are set further back, the risk of extrusion is lower, but the risk of migration is higher. You can choose from two distinct canalicular plugs.

- Thermosensitive plugs: These are acrylic canalicular plugs, which measure 6 mm in length and 0.4 mm in diameter prior to insertion. Once inside the canaliculus, as there is a rise in the temperature, the plug shortens in length to 1.5 mm and widens in diameter to 1 mm.
- Absorptive Plugs: These plugs have the advantage of hydrogel's water absorbing properties. Once implanted, the canalicular plug comes in contact with the tear film and the hydrogel expands into a soft gelatinous material within the canaliculus.[43,44]

The lacrimal occluders can also be classified based on the duration for which the occlusion is required.

- Inserting collagen plugs into the canaliculi provides temporary occlusion. They are gone in a couple of weeks. The main goal of these procedures is to prevent epiphora from happening after a successful occlusion procedure.
- Reversible occlusion can be accomplished with silicone or long-acting collagen plugs.
- Only in patients with severe dry eyes who show improvement with temporary plugs without epiphora is permanent occlusion attempted. After a punctum has been dilated, the proximal canaliculus can be occluded permanently by coagulating the tissue with cautery. [35,43]

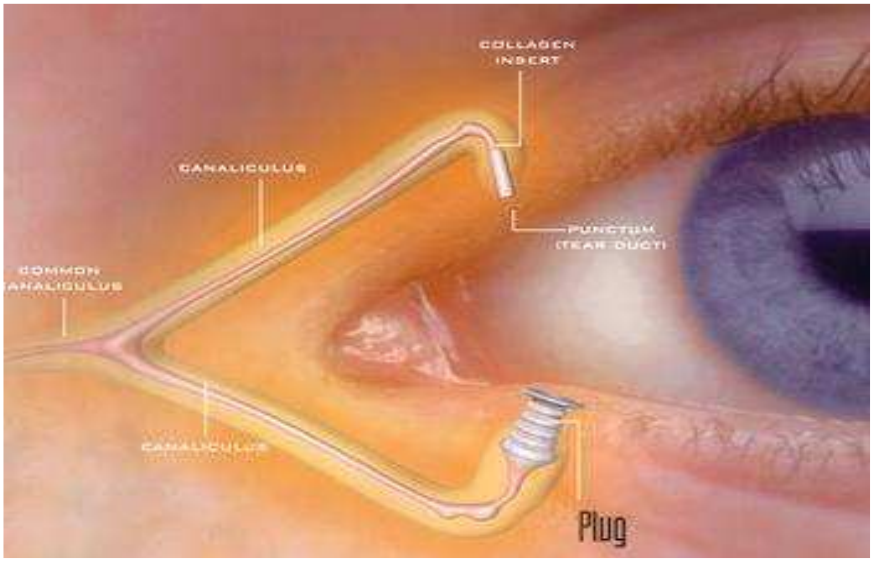


Figure 15: Punctal plugs

Anti- Inflammatory agents:

The inflammation of the ocular surface is the end result of a cascade of events that leads to dry eye disease. Chronic irritative stress or a systemic inflammatory autoimmune condition can set off this inflammatory reaction. Corticosteroids, immunomodulatory drugs, and essential fatty acids are just a few of the anti-inflammatory treatments available for dry eye. [45]

- ❖ Corticosteroids: They cause inhibitory mechanism on a range of immune responses mediated by T cells, B cells and Cytokines.
  - DED was initially treated with methylprednisolone, a corticosteroid. Methylprednisolone 1% is typically administered three to four times daily for one to two weeks, with dosage reduction beginning two weeks later. Methylprednisolone is effective at alleviating symptoms and boosting TBUT and Schirmer's.
  - Prednisolone: To put it simply, this is a man-made substitute for hydrocortisone. It's possible to get prednisolone acetate in either 1% or 0.125% strength. When taken as a

1% solution of prednisolone three times daily for a month, patients with DED report fewer symptoms.

- Lotepredenol Etabonate: To date, the majority of research involving this drug has focused on its use to treat DED. Lipophilic lotepredenol etabonate is absorbed by the eye after topical application. For four weeks, 0.5% lotepredenol etabonate is effective in treating moderate to severe dry eye.
- Fluoromethalone (FML): It's a corticosteroid, and it comes in both a liquid suspension and a topical ointment, both of moderate potency. Despite being more lipophilic, its concentration in aqueous was found to be lower than that of Lotepredenol's. The standard treatment protocol for DED involves the administration of 0.1% FML for a duration of 3 weeks.[20,46]

❖ Immunomodulators: This refers to substances that can alter the body's immune system chemically. In order to achieve long-term control of inflammation without the negative effects of corticosteroids, these are used as steroid sparing agents. The following are the different types of topical immunomodulators used to treat DED:

- Inhibitors of T-cell signaling: Cyclosporin A and Tacrolimus are two examples of drugs that fit this description. A calcinurin inhibitor, cyclosporin A is taken twice daily at a dosage of 0.05%. Also having an immunosuppressive effect, the macrolide antibiotic tacrolimus. Inhibition of T cell activation is achieved through binding to the FK-506 protein on the T cell. Tacrolimus is taken at a dosage of 0.005% once daily.
- Biological response modifiers: Such substances are derived from living organisms and are used to alter the progression of disease. Lifetigrast, a topical antigen-1 antagonist for lymphocytes. On the surface of T and B cells is an integrin called Lymphocyte Function-Associated antigen (LFA-1). This medication blocks lymphocyte-ICAM interaction by binding to LFA-1. The subsequent growth and discharge of

inflammatory cells and cytokines are thus halted. Only in cases where DED has not responded to artificial tears do these medications need to be considered.[47]

- Essential Fatty Acids (EFA) : Since recent years, EFA have been studied for their potential to alleviate DED symptoms. Both cyclooxygenase and 5-lipoxygenase require a substrate, and the omega-3 fatty acid eicosapentaenoic acid (EPA) and the omega-6 fatty acid arachidonic acid (AA) are direct competitors. Inhibition of Arachidonic Acid's conversion to potentially harmful inflammatory mediators like Prostaglandin E2 (PGE2) and Leukotriene B4 is thought to be the mechanism underlying these compounds' anti-inflammatory effects.[48]

Contact Lens:

Contact lenses are known to exacerbate dry eyes by contributing to their inflammatory, sensory, and evaporative effects. However, there is the possibility of a reservoir effect from the fluid that is caught behind the lens. Additionally, contact lenses are helpful in alleviating symptoms brought on by secondary corneal changes.

Several brands of contact lenses have been created to alleviate dry eye symptoms while wearing them. Silicone hydrogel, one of the materials, can keep its moisture for up to 16 hours. In rare cases, people with extremely dry eyes may benefit from wearing occlusive gas-permeable scleral contact lenses, which create a reservoir of saline over the cornea.[49]

Autologous Blood Serum (ABS) Eyedrops:

On the basis of the hypothesis that ABS, not only functions as a lubricating agent but also mimics the tear film, ABS promises advantages over the commonly used artificial tears.

Aside from having more vitamin A, lysozyme, transforming growth factor (TGF), and fibronectin and fewer immunoglobulin A (IgA), epithelial growth factor (EGF), and vitamin C, serum has a composition similar to that of tears. The concentration of biological factors in actual tears informs the standard 20% concentration for autologous serum administration. An excess of TGF-, a component of ABS, is known to have anti-proliferative effects that can supress wound healing of the ocular surface, which is one of the negative effects of ABS.

Indications include severe ocular surface disorders such as dry eye after LASIK surgery, superior limbic keratoconjunctivitis, and postoperative dry eye from Sjögren's syndrome or non-tear Sjögren's deficiency due to graft-versus-host disease.[50]

DIABETES MELLITUS

The World Health Organization (WHO) defines diabetes as a collection of metabolic disorders whose hallmark is uncontrolled high blood sugar (hyperglycemia) despite medical intervention.[51]

One of the 10 leading causes of death worldwide in this century, diabetes is also one of the fastest growing health problems in the world. According to the World Health Organization, in 2019, 1.6 million people died as a direct result of diabetes. Noncommunicable diseases accounted for 74% of all deaths worldwide in 2019. Although previously limited to the developed world, type 2 diabetes now affects people of all ages, including children and young adults. [5]

In recent years, India has seen a dramatic increase in the number of people diagnosed with diabetes or at risk for developing the condition. IDF estimates that 8.9% of the Indian population, or 77 million people, have diabetes as of 2019. One in six Indian adults now has diabetes, making the country the world's second-largest sufferer population. The fact that over half of all cases of diabetes go undiagnosed is also concerning. In 2015, it was estimated that 25.2% of adults had impaired glucose tolerance (IGT); this number is expected to rise to 35.4% by 2045. Additionally, the incidence of diabetes is on the rise, with a parallel progression from euglycemia to pre-diabetes to diabetes. [32]

WHO Classification of Diabetes 2019:

In an ideal situation, a single classification system would serve the three primary purposes – clinical care, etiopathogenesis and epidemiology. However, this is not possible with the current state of knowledge and the variation in health care facilities across the globe. Hence, with the aim to prioritize health care, the following classification was put forth by WHO is 2019.[52]

Class	Subclass
Type 1	
Type 2	
Hybrid forms	• Slowly evolving immune-mediated diabetes of adults • Ketosis-prone T2DM
Other specific types	• Monogenic diabetes » Monogenic defects of beta cell function » Monogenic defects in insulin action • Diseases of the exocrine pancreas • Endocrine disorders • Drug- or chemical-induced • Infection-related diabetes • Uncommon specific forms of immune-mediated diabetes • Other genetic syndromes associated with diabetes
Unclassified diabetes	
Hyperglycemia first detected in pregnancy	• DM in pregnancy • Gestational DM

Source: World Health Organization (WHO). 2019. Classification of Diabetes Mellitus. Geneva: April. https://www.who.int/publications-detail/classification-of-diabetes-mellitus.

Figure 16: WHO Classification of DM

Etiopathogenesis of Diabetes Mellitus:

The aetiology and pathogenesis of diabetes can be described simply as problems revolving around insulin sensitivity and insulin secretion, but the underlying specific defects are complex". It is now generalized that the underlying mechanism common to all the forms of DM are dysfunction or destruction of β cells of pancreas.[53]

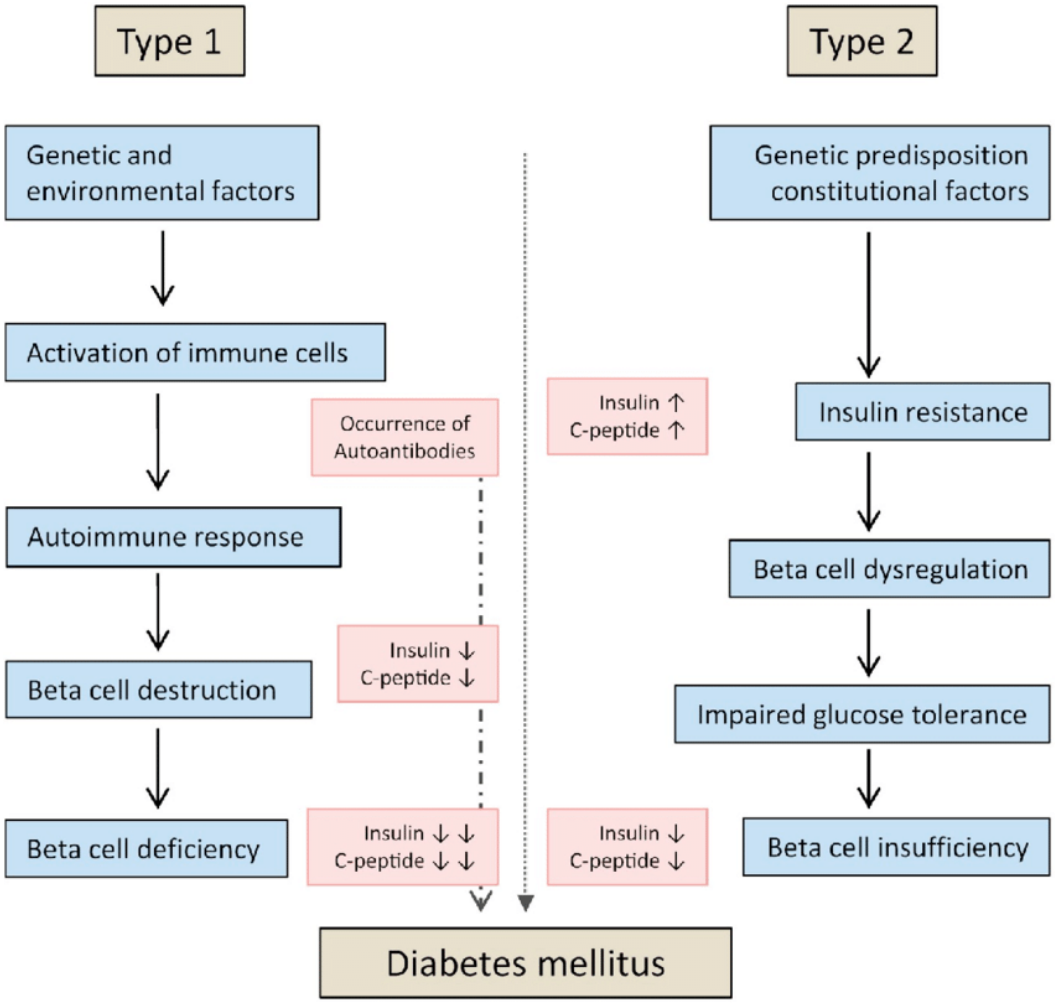


Figure 17: Etiopathogenesis of Diabetes Mellitus

Complications of Diabetes Mellitus:

Microvascular and macrovascular complications are the two main categories for diabetes complications. Because of their distinct architecture and cellular components, micro vessels serve as the cardiovascular system's fundamental functional unit. Micro vessels, as opposed to macro vessels, play a crucial role in regulating blood pressure and ensuring that organs receive adequate nutrients. Sometimes, changes in microvascular function occur before the more obvious hyperglycemia and vascular pathologic changes become evident in the body. Diabetic microangiopathy is characterised by pathognomonic changes in the microvasculature, specifically an increase in the thickness of the capillary basement membrane in the arterioles of the glomeruli, retina, myocardium, skin, and muscle. Diabetic retinopathy, diabetic neuropathy, and diabetic nephropathy are the three most common microvascular complications of diabetes mellitus..[54]

- **Diabetic Retinopathy (DR):**

Diabetic retinopathy exemplifies the broad range of microvascular end-organ damage brought on by DM. By 2030, it is predicted that 191.0 million people worldwide will have diabetic retinopathy, and that 56.3 million will have vision-threatening diabetic retinopathy.[55]

Risk Factors for DR:

1. Diabetes duration
2. Uncontrolled glucose or blood pressure levels are associated with increased risk.
3. Hypertension
4. Dyslipidemia
5. Ethnicity
6. Pregnancy
7. Smoking

Pathology of DR:

It's important to distinguish between proliferative DR (PDR) and non-proliferative DR (NPDR), the former of which does not involve the formation of new blood vessels and the latter of which does. Retinal ischaemia, capillary leakage, and capillary occlusion are the three main pathological processes involved.

- Pathophysiology of NPDR: Retinal capillaries are damaged by advanced glycation end-products (AGEs) formed during hyperglycemia. The endothelium is damaged, and the capillary walls become vulnerable, leading to the development of micro-aneurysms. Dot haemorrhages are caused by microaneurysms that burst in the retina's deeper layers. In some cases, clots form in blood vessels due to adherent activated leukocytes. This causes blotches that look like cotton wool..[56]

- Pathophysiology of Diabetic Macular Edema (DME): Increased vascular permeability results from the up-regulation of inflammatory cytokines, which is caused by chronic inflammation and endothelial damage. As a result, fluid seeps into the retina, right under the macula, causing macular edema and eventually DME. The sediment deposits left behind as edema recedes give rise to "hard exudates," which are waxy and yellow in colour.[56]
- Pathophysiology of PDR: Ischemia develops as a result of blood vessel damage. VEGF and other pro-angiogenic factors are released by retinal cells in response to prolonged ischemia. These factors promote neovascularization, which helps the retina's blood vessels heal more quickly after being damaged. New blood vessels are formed from pre-existing capillaries during a process called angiogenesis. Retinal neovascularization is the development of new blood vessels at the retinal blood-brain barrier. Extremely immature, fragile, permeabilise, and easily bleed, these new vessels are the source of potentially blinding complications like vitreous haemorrhage and tractional retinal detachment.[56]

ETDRS Classification of Diabetic Retinopathy:

1. **Mild NPDR:** Microaneurysms only
2. **Moderate NPDR:** At least 1 hemorrhage/microaneurysm and/or at least one of the following:
    - Retinal Hemorrhages
    - Hard exudates
    - Cotton wool spots
    - Venous beading
3. **Severe NPDR:** Any of the following, but no signs of PDR (4-2-1 rule)
    - > 20 intra retinal hemorrhages in each of the 4 quadrants
    - Definite venous beading in 2 or more quadrants
    - Prominent Intra Retina Microvascular Anomaly (IRMA) in 1 or more quadrants.
4. **PDR :** One of either:
    - Neovascularization
    - Vitreous/Preretinal Hemorrhage

Early PDR : Presence of new blood vessels but not meeting the criteria for high risk PDR.

High risk PDR:

- Neovascularization of the disc (NVD) one fourth to one third of the disc area with or without vitreous hemorrhage.
- NVD less than one fourth disc with vitreous hemorrhage or pre retinal hemorrhage
- Neovascularization elsewhere (NVE) greater than or equal to one half of the disc area with pre retinal or vitreous hemorrhage.

Clinically Significant Macular Edema:

- Retinal thickening at or within 500µ at the centre of the macula
- Hard exudates at or within 500µ at the centre of the macula
- An area of retinal thickening greater than one disc area in size, at least a part of which is within one disc diameter of the centre of the macula[20,57]

- **Diabetic Neuropathy:**

When other potential causes of neuropathy in a patient with DM have been ruled out, the patient is said to have diabetic neuropathy (DN). Diabetes neuropathy (DN) is a common form of peripheral neuropathy. It's the leading reason for amputations that aren't caused by trauma and the leading cause of hospitalizations among diabetes complications. There are various subtypes of DN, including peripheral neuropathy, autonomic neuropathy, focal neuropathy, proximal neuropathy, and cranial neuropathy.[6]

- **Peripheral neuropathy:** It is a type of neuropathy that generally affects the feet/legs and hands/arms. It is the most common type accounting for almost 75% of DN.
- **Autonomic neuropathy:** Autonomic neuropathy causes damage to the internal organs resulting in cardiovascular, gastrointestinal, urinary, sweating, pupils, and metabolic disturbances. Autonomic DN often goes unnoticed by both the patient and the physician. Autonomic nerve involvement can occur as early as one year after the diagnosis of DM. Orthostatic hypotension, resting tachycardia, etc are pathognomonic of diabetic autonomic neuropathy.

- **Focal neuropathy :** These conditions entail damage to singular nerves. The most common example being entrapment syndromes such as carpal tunnel syndrome.
- **Proximal neuropathy:** This is a rare and disabling type of neuropathy typically affecting one side of the body.
- **Cranial neuropathy:** The oculomotor nerve, followed by the trochlear and facial nerves, are the most commonly affected by cranial neuropathy in diabetic patients. Diabetic oculomotor palsy is characterised by third nerve palsy with sparing of the pupil, which is caused by nerve infarction.[58]

Effect of Diabetes on Corneal Nerves:

The cornea has the finest and most numerous nerve endings of any human tissue. About 7000 nociceptors/mm2 can be found in its central nervous system. The cornea is sensory innervated by 70–80 long ciliary nerves from the nasociliary branch of the ophthalmic branch of the trigeminal nerve. Sympathetic innervation is provided by the superior cervical ganglion. Damage to the corneal nerve and other ocular surface abnormalities are most strongly correlated with the duration of diabetes and with poor glycemic control.

Advanced glycation end products (AGEs), reactive oxygen species (ROS), and the protein kinase pathway are all produced and activated in response to hyperglycemia.

Axonal degeneration is caused by the accumulation of glycation products in the nerve sheath, perineural collagen, Schwann cells, and axoplasm of nerves.

An additional pathway implicated in DN pathogenesis is the sorbitol-aldose reductase pathway. Degenerative changes in nerve cells result from the accumulation of sorbitol fructose, a byproduct of excess glucose. Superoxide ions are produced when an excess of glucose is metabolised via oxidative phosphorylation in the mitochondria. As a consequence, there is mitochondrial damage, which impedes nerve conduction and leads to demyelination of the axons.

processes reduce the number of corneal nerve fibres and shorten their length. Keratopathy, characterised by hypoaesthesia, dry eye, and keratitis, is a long-term effect of uncontrolled hyperglycemia.[59]

DRY EYE DISEASE IN DIABETES MELLITUS

Two of the most well-known complications of uncontrolled DM are diabetic retinopathy and diabetic cataracts. However, dry eye syndrome (DES) was only recently recognised as a result of diabetes.

It is now understood that diabetes mellitus is a significant systemic risk factor in the development of DED. A number of factors are thought to exacerbate the symptoms of DED, including poor glycemic control, a long duration of DM, diabetic peripheral neuropathy, and the presence of diabetic retinopathy..[60]

The Beaver Dam Eye Study reported that approximately 20% of dry eyes occurred in individuals with Type 2 diabetes in the age group of 43 and 86 years. [61]

Hom and De Land reported that 53% of patients with either diabetes or borderline diabetes had self-reported, clinically relevant dry eyes.[62]

Five-and-a-half percent of diabetics in a hospital-based study had DES, and there was a strong correlation between DES use and the length of time someone had diabetes.

Another study found that 17.1% of DES among DM patients had mild NPDR, 17.1% had moderate NPDR, 11.1% had severe NPDR, and 25.1% had proliferative DR (PDR).[62,63]

Individuals with DR and/or clinically significant macular edoema have a significantly higher prevalence of DES compared to the non-DR group (P = 0.006). Both DES and retinopathy were associated with HbA1c, and the odds of DR in DES were 2.29 (95% CI = 1.16-4.52, P = 0.016)..[62]

In the case of India, The prevalence rate of DED among diabetics using the OSDI is 36%, according to a cross-sectional study conducted by Manvi Aggarwal et al. in a tertiary care hospital in western Maharashtra. Forty-four percent of patients had mild DED, 16 percent had moderate DED, and 4 percent had severe DED. The Tear-film Breakup Time (TBUT) test demonstrated excellent agreement with the highest diagnostic accuracy, while Schirmer's test and the Fluorescein test demonstrated good and moderate agreement, respectively.. The limitation in this study was that the sample size was small.[2]

A hospital based, cross-sectional, observational study done by Geeta Khurana et al., at Lady Hardinge Medical College and associated hospitals, found a higher incidence of all symptoms and signs of DED except itching in patients with DR in comparison to patients without DR.[64]

Divya Kesarwani et al. found that abnormalities in the tear film are a prominent sign of diabetic ocular surface disease. Schirmer's test and TBUT scores went down dramatically. The study's limitations stemmed from its small sample size and the absence of a standardised questionnaire to evaluate the patients' self-reported symptoms..[34]

Pathogenesis:

It is unknown what role the length of time someone has had DM plays in the pathogenesis of DED, but it is one of the major risk factors. DED has a complex set of causes, but diabetes mellitus (DM) is likely to alter the morphology and pathways associated with the lacrimal functional unit.[7]

Different mechanisms may contribute to the dysregulation of LFU in people with diabetes, which is a metabolic disorder. There has been increased focus on the ocular surface in the research literature over the past two decades, particularly in relation to diabetes.

Initial events that have been linked to changes in the tear film and ocular surface in diabetic patients include: impairment on insulin action, a) chronic hyperglycemia, and b) corneal nerve damage.[65]

Tissue injury is facilitated by hyperglycemia because it fosters an inflammatory milieu, which in turn promotes further tissue damage. Damage to the ocular surface is caused by hyperglycemia, which in turn triggers the expression of inflammatory cytokines such as tumour necrosis factor alpha and interleukin 1 beta and the production of reactive oxygen species.[63]

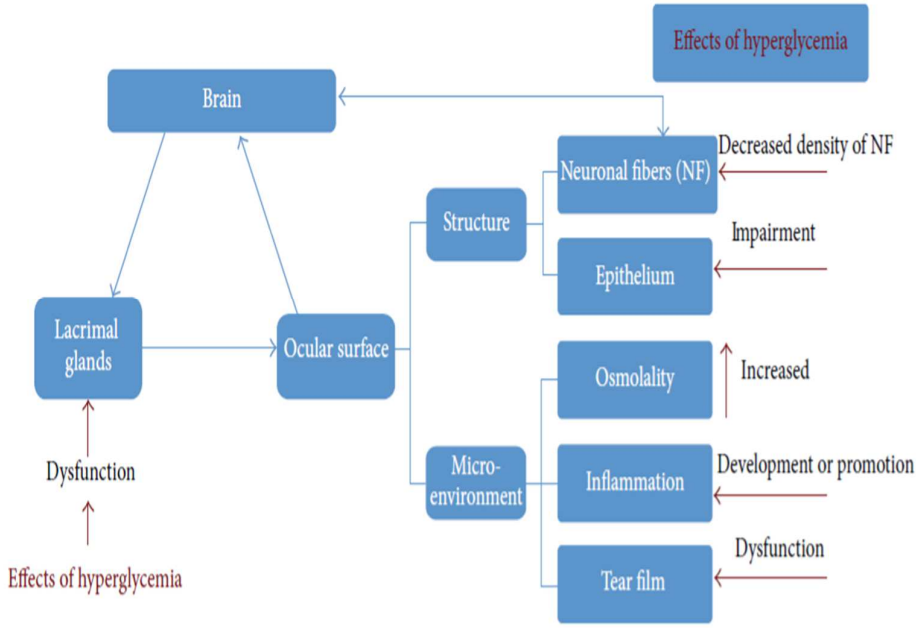


Figure 18: Effects of Hyperglycemia on Lacrimal Functional Unit

Studies have shown that people with Type 2 Diabetes have higher levels of Insulin-like Growth Factor Binding Protein in their tear secretions (IGFBP 3). In addition to its many other functions, IGFBP3 is a negative regulator of IGF signalling. Put simply, IGFBP 3 controls insulin resistance, apoptosis, and oxidative damage. Damage to the ocular surface may result from hyperglycemia, which may be caused by an increase in IGFBP3 production in response to elevated glucose levels in the tears.[66]

The basement membrane of the corneal epithelium is also affected by hyperglycemia. Inflammation and edoema of the normally dehydrated corneal stroma have been observed as a result of an increase in corneal thickness and a reduction in the barrier function of the basement membrane.[8]

Hyperglycemia causes molecular changes, such as the production of advance glycation end products (AGE). This is a crucial pathological outcome that has been linked to retinopathy, neuropathy, and nephropathy, all of which are common in people with diabetes. When advanced glycation end products (AGEs) build up in the basement membrane of the corneal epithelium, it alters cell signalling pathways, causing them to become apoptotic and pro-inflammatory and heightening oxidative stress.[66]

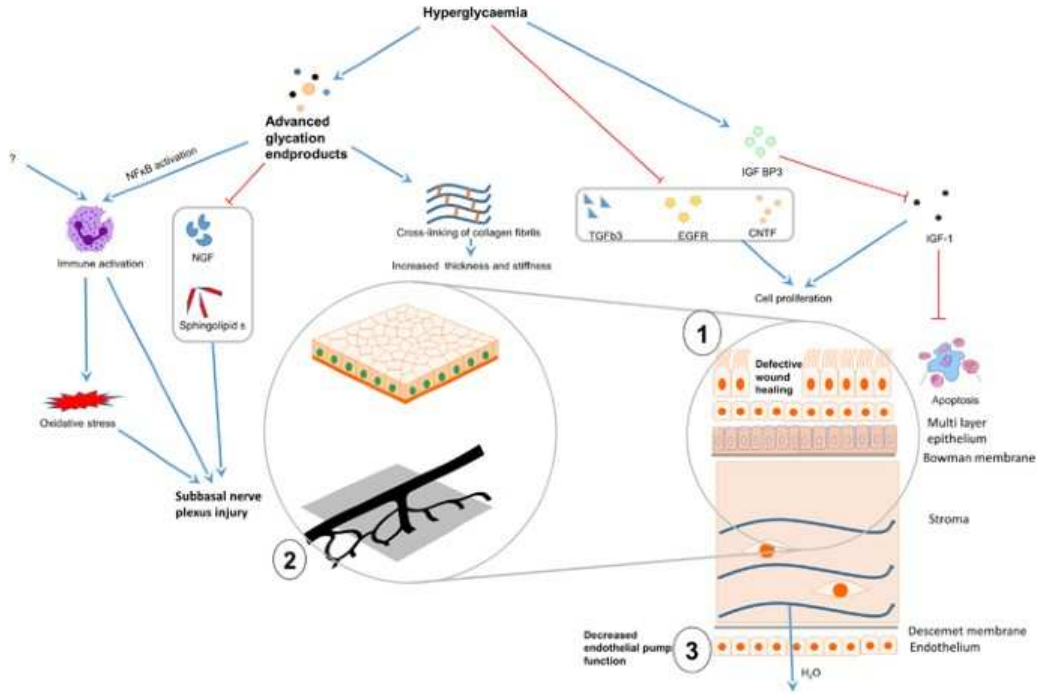


Figure 19: Effect of hyperglycemia on corneal epithelium

Autonomic neuropathy and damage to the microvasculature of the lacrimal and meibomian glands appear to contribute to impaired gland function. The precise mechanisms depicting the same are, however, still unknown.[67,7]

To be exact, the total protein concentration in a typical adult's tear is between 6 and 10 mg/ml. Both lactoferrin and lysozyme, as well as two tear-specific proteins, are among the four major proteins that are consistently detected in tears. Diabetes mellitus (DM) and dry eye disease have been linked to abnormal tear protein regulation. The tears of DM patients were found to contain six novel proteins, according to a study by Ghosh et al. Heat shock protein 60 (HSP 60) was identified as one of the proteins, and it has been shown to play a complex regulatory role in apoptosis.[28]

Corneal nerves are affected by diabetic neuropathy due to the build-up of advanced glycation end products (AGEs) and free radicals, which disrupt the myelin sheath and axonal transport.[59]

Psychological Effects of Dry Eye Disease:

Although dry eye disease does not usually result in severe vision loss, it is still a serious problem for those who have it. Because of modern living habits, the symptoms have worsened significantly and become a strain on patients as the disease advances. It can be frustrating that treating DED often necessitates using topical medications for an extended period of time. “It has been shown in a number of studies that such patients lose a substantial amount of annual productivity as a result of their condition. Read, use a computer, watch TV, etc., and patients are more likely to complain of difficulties.

MATERIALS AND METHODS

The present study was conducted at the Department of Ophthalmology, KLE'S Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the period of January 2021 to December 2021.

Primary Objective:

To compare the prevalence of dry eye disease in diabetic patients and non-diabetic patients.

Secondary Objective:

To assess the corneal nerve sensitivity in diabetic patients and non-diabetic patients.

To compare the severity of dry eye disease among diabetic patients, in those suffering from different grades of diabetic retinopathy.

Method of data collection

Study population:

All the patients attending the ophthalmology OPD of KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Study Design:

One-year prospective study

Study Duration:

One year, from 1st January 2021 to 31st December 2021

Sample Size:

400

Sampling Procedure:

The minimum sample size formula based on prevalence is

$$n = \frac{p(100 - p)Z^2}{E^2}$$

n is the sample size required, **p** is the percentage occurrence of a state or condition (proportion or prevalence), **E** is the percentage maximum error required, **Z** is the value corresponding to level of confidence required.

Prevalence of dry eyes is observed as 54.3%[1]. With percentage of maximum error as 5% at 95% confidence level sample size is given by,

$$n = \frac{54.3 \times (100 - 54.3) \times (1.96)^2}{5^2}$$

$$n \approx \mathbf{381}$$

Minimum sample size required was 381.

For the ease of calculation, a sample size of 400 was taken for this study.

Selection Criteria:

Inclusion Criteria:

Patients above the age of 18 years, attending the Ophthalmology OPD.

Exclusion Criteria:

- Patients with history of current or recent use of topical/systemic ocular medication like beta blockers, diuretics, anticholinergics, anti-histaminics, anti-depressants, lubricants.
- Patients with history or clinical evidence of any ocular disorders like blepharitis, lid deformities, recent ocular surgery or trauma, systemic autoimmune diseases known to affect ocular surface.

- Contact lens wearers, patients who have undergone LASIK surgery, cigarette smokers.
- Patients with clinical evidence of Vitamin A deficiency.
- Eyes in which fundus examination is not possible.

Ethical clearance was obtained from the local institutional ethical committee. Informed/written consent of patients was taken after explaining in detail about the methods and procedures involved in the study in their own vernacular language.

Data regarding demographic parameters such as age, sex, occupation and address were noted on a predesigned proforma by the investigator at the time of first visit.

A detailed clinical history including the history of diabetes, drug history for systemic illnesses and use of contact lenses was taken from the patients.

The patients eligible for the study were divided into two groups:

Group 1: Non-Diabetic individuals

Group 2: Diabetic individuals

To assess the symptoms of dry eye disease, Standard Patient Evaluation of Eye Dryness (SPEED) Questionnaire was used.

The SPEED Questionnaire assessed the following symptoms:

- Dryness/Grittiness/Scratchiness
- Soreness/Irritation
- Burning/Watering
- Eye fatigue

The patients were asked to recall the occurrence of one or more than one of these symptoms over the period of three months.

The frequency of the symptoms was gauged by a scale of 0-3

- 0 = Never
- 1 = Sometimes
- 2 = Often
- 3 = Constant

Similarly, the severity of these symptoms was evaluated on the scale of 0-4,

- 0 = No problem
- 1 = Tolerable – not perfect but not uncomfortable
- 2 = Uncomfortable – irritating, but does not interfere with the day
- 3 = Bothersome – irritating and interferes with the day
- 4 = Intolerable – unable to perform daily activities

The maximum score that can be achieved on SPEED Questionnaire is 28 points.

Scoring was done as follows:

0-4 points: Mild dry eye symptoms

5-7 points: Moderate dry eye symptoms

8 + points: Severe dry eye symptoms[2]

Ocular examination:

- To record best-corrected vision, we used the Snellen's visual acuity chart.
- The eyelids and adnexa, conjunctiva, cornea, anterior chamber, iris, pupils, and lens were all examined with a slit lamp biomicroscope as part of an anterior segment examination.

- Indirect ophthalmoscopy was used to examine the posterior segment. If diabetic retinopathy was noted, its severity was determined using the ETDRS Classification system.
- Four quadrants of the cornea were tested for nerve sensitivity with a wisp of cotton.

Syringing the lacrimal sac was used as a diagnostic tool to determine if the nasolacrimal duct was patent.

Testing for dry eye disease was then performed, using Schirmer's test and Tear film break-up time (TBUT)..

Schirmer's Test:

Sterile strips of Whatman No. 41 paper measuring 5 mm x 35 mm were used for the Schirmer's 2 test. The patient was confined to a room with no ventilation and asked to wait there. Proparacaine hydrochloride 0.5% ophthalmic solution, a topical anaesthetic, was administered to the patient's eyes. This was carried out to eradicate the reflex tearing and to gauge the amount of basal secretions. To avoid cross-contamination from skin oils, the end of the Schirmer's strip was folded at the 90 degree at the marked area. The patient's lower eyelids were retracted 5 minutes after the application of the topical anaesthetic. The folded paper was then inserted along the junction of medial two thirds and lateral one third of the lower eyelid. Since blinking can trigger reflex tear secretion, the patient was instructed to keep their eyes gently closed. From the curved end to the wetted strip area, millimetres of wetting were recorded (mm).

Interpretation:

Measurement of more than 15 mm of wetting was considered to be normal.

10 mm – 15 mm : Mild dry eye

5 mm – 10 mm: Moderate dry eye

0 mm – 5 mm: Severe dry eye

Tear Film Break-up Time (TBUT)

Evaporative dry eye disease can be tested for with the tear film stability test (TBUT) because it is a quantitative method for measuring the rate of evaporation. The time in seconds that pass between the last blink and emergence of the first dry spot on the cornea is used to determine this.

The patient was positioned in front of a slit lamp. One drop of antibiotic solution was applied to a fluoresceine strip that had been kept in sterile conditions. The lower palpebral conjunctiva was stained by placing the wet fluoresceine end of the strip in the cul-de-sac. After removing the strip, the patient was instructed to blink several times to ensure that the dye had spread throughout the tear film. The patient was then instructed to stare straight ahead without blinking, and tear film was examined using a cobalt blue filter to look for the first sign of a tear, which would appear as a black dot on the tear film's otherwise green background. The break up time of the tear film was calculated as the number of seconds that elapsed after the last blink before the first black spot appeared..

TBUT > 10 seconds = Normal tear film stability.

5 – 10 seconds = Marginal tear film stability.

< 5 seconds = Low tear film stability.

Grading of dry eye:

- Normal eyes was defined as patients who had Schirmer's II test value > 15mm and TBUT > 10 seconds
- Mild dry eye was defined as patients who had Schirmer II test value between 10 – 15 mm of wetting and TBUT of 10 seconds.
- Moderate dry eye was defined as patients who had Schirmer II test value of 5 – 10 mm of wetting and TBUT of 5 – 10 seconds.
- Severe dry eye was defined as patients who had Schirmer II test value of < 5 mm of wetting and TBUT of < 5 seconds.

STATISTICAL ANALYSIS

Data was analysed using statistical software R version 4.2.1. and Microsoft Excel. Categorical variables are represented by frequency and percentage. Continuous variables given in Mean ± SD / Median (Min, Max) form. Normality of variable was checked by Shapiro Wilk test and QQ plot. Mann Whitney U test was used to compare the distributions of variables over groups. Chi-square test was used to check the dependency between categorical variables. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

The present study was conducted at KLE's Dr Prabhakar Kore Hospital and Medical Research Centre, in the duration of one year from 1st January 2021 to 31st December 2021.

400 patients were enrolled in this study and divided into 2 groups of 200 each, Group 1 – Non-diabetic individuals and Group 2 – Diabetic individuals, based on their diabetic status. Data was analysed using statistical software R version 4.2.1. and Microsoft Excel. The results acquired are tabulated as follows.

The collected sample size was seen in the age group of 30 – 87 years, the mean age being 55.02 ± 12.21 years.

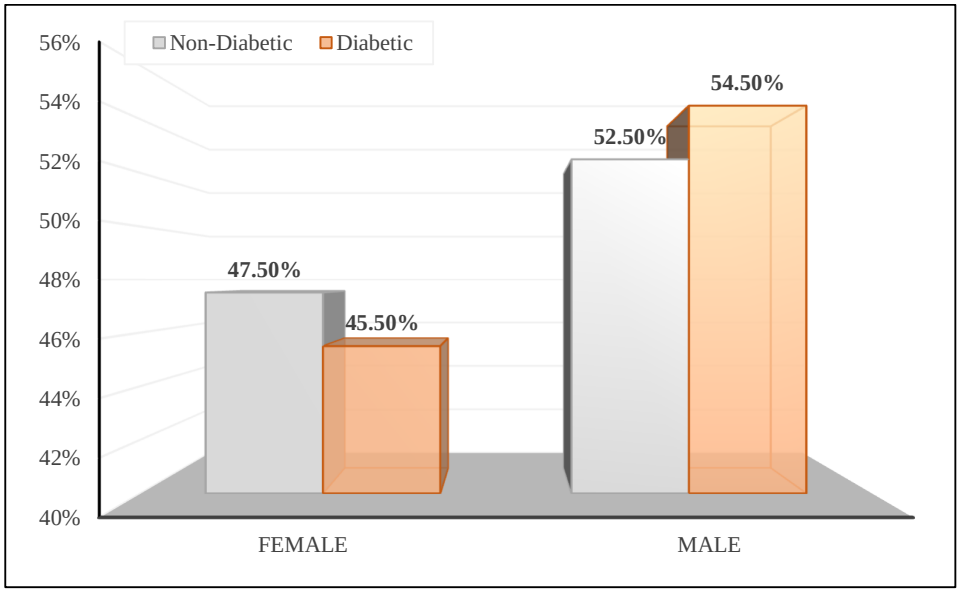
Variables	Sub Category	Group		Total
		Non-Diabetic	Diabetic	
Age (years)	Mean ± SD	55.94 ± 10.96	54.09 ± 13.31	55.02 ± 12.21
	Median (Min, Max)	56 (33, 87)	55.5 (30, 85)	56 (30, 87)

Table 1: Age wise distribution of Diabetics and Non Diabetics

In the present study, the number of males were marginally more than females, males being 53.5% and females being 46.5%

Variables	Sub Category	Group		Total
		Non-Diabetic	Diabetic	
Gender	Female	95 (47.5%)	91 (45.5%)	186 (46.5%)
	Male	105 (52.5%)	109 (54.5%)	214 (53.5%)

Table 2: Gender wise distribution of Diabetics and Non Diabetics



Graph 1: Distribution of gender over groups.

Among the 200 diabetic patients enrolled in this study, 84.5% patients were taking treatment for DM either in the form of oral hypoglycemic agents, or insulin or both. 15.5% patients were not taking any treatment measures for the same.

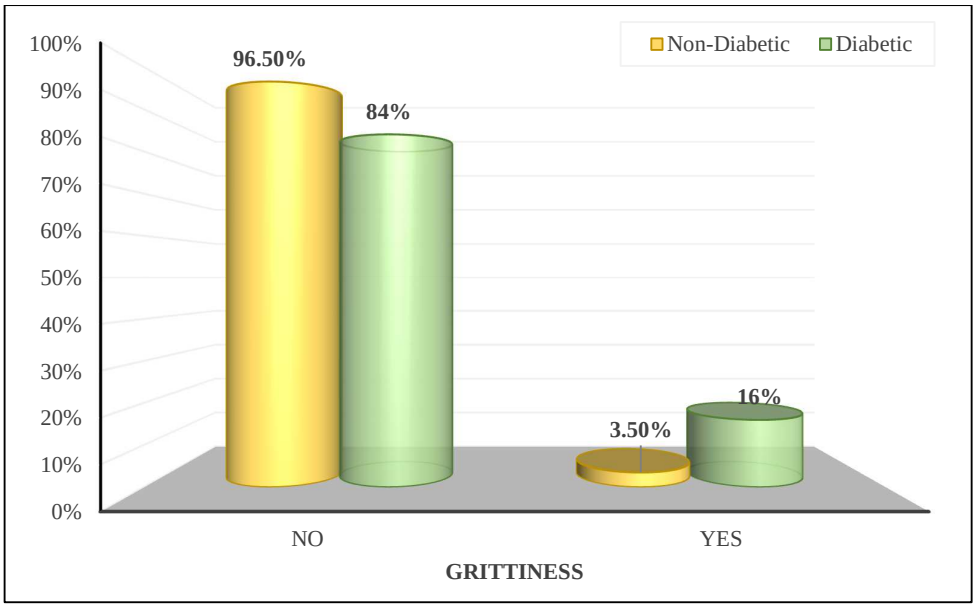
Variables	Sub Category	Group		Total
		Non-Diabetic	Diabetic	
History of DM	No	200 (100%)	0	200 (50%)
	Yes	0	200 (100%)	200 (50%)
Treatment	No	200 (100%)	31 (15.5%)	231 (57.75%)
	Yes	0	169 (84.5%)	169 (42.25%)

Table 3: Distribution of Diabetics and Non Diabetics based on Treatment of Diabetes Response to the proforma provided including the SPEED Questionnaire

Variables	Sub Category	Group		Total	p-value
		Non-Diabetic	Diabetic		
Grittiness	No	193 (96.5%)	168 (84%)	361 (90.25%)	**< 0.001[C]***
	Yes	7 (3.5%)	32 (16%)	39 (9.75%)	
Burning	No	187 (93.5%)	152 (76%)	339 (84.75%)	**< 0.001[C]***
	Yes	13 (6.5%)	48 (24%)	61 (15.25%)	
Irritation	No	168 (84%)	138 (69%)	306 (76.5%)	**< 0.001[C]***
	Yes	32 (16%)	62 (31%)	94 (23.5%)	
Eye Fatigue	No	156 (78%)	120 (60%)	276 (69%)	**< 0.001[C]***
	Yes	44 (22%)	80 (40%)	124 (31%)	
Frequency	0	136 (68%)	67 (33.5%)	203 (50.75%)	**< 0.001[MC]***
	1	48 (24%)	79 (39.5%)	127 (31.75%)	
	2	16 (8%)	53 (26.5%)	69 (17.25%)	
	3	0	1 (0.5%)	1 (0.25%)	
Severity	0	136 (68%)	67 (33.5%)	203 (50.75%)	**< 0.001[MC]***
	1	40 (20%)	66 (33%)	106 (26.5%)	
	2	21 (10.5%)	63 (31.5%)	84 (21%)	
	3	3 (1.5%)	4 (2%)	7 (1.75%)	
Speed Score	Normal	136 (68%)	67 (33.5%)	203 (50.75%)	**< 0.001[C]***
	Mild	49 (24.5%)	87 (43.5%)	136 (34%)	
	Moderate	5 (2.5%)	9 (4.5%)	14 (3.5%)	
	Severe	10 (5%)	37 (18.5%)	47 (11.75%)	
	Mean ± SD Median (Min, Max)	1.42 ± 4.23 2 (0, 24)	1.44 ± 3.21 0 (0, 20)	2.42 ± 3.87 0 (0, 24)	**< 0.001[MW]***

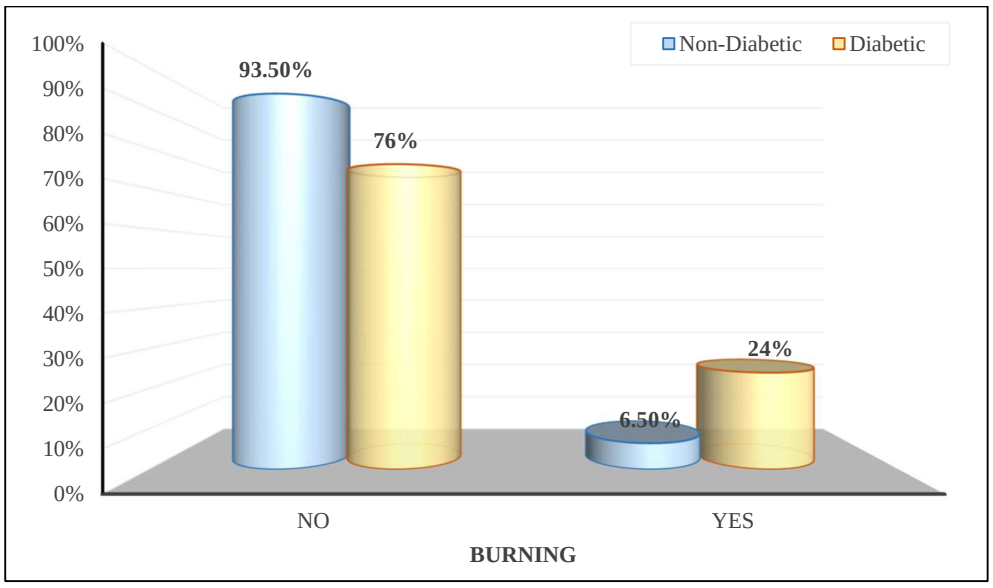
Table 4: Distribution of patients based on SPEED questionnaire and score

In response to the SPEED questionnaire, the following results were obtained. Although grittiness was not a major symptom in the overall sample size, it was seen in 3.5% of Non-diabetic patients and 16% of Diabetic patients.



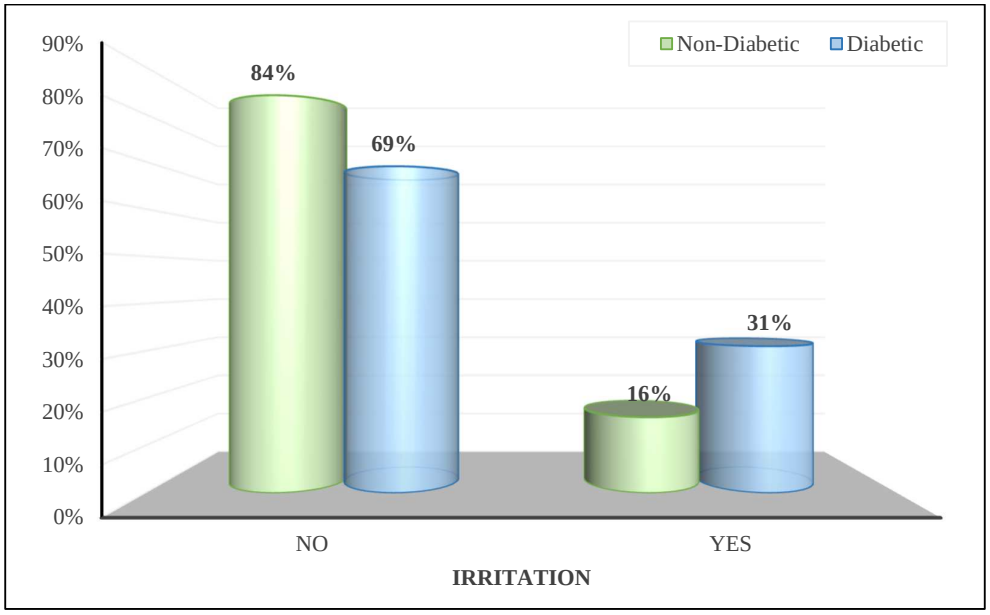
Graph 2: Distribution of Grittiness over groups.

Burning was seen in 6.5% of Non-diabetic population and 24% Diabetic population.



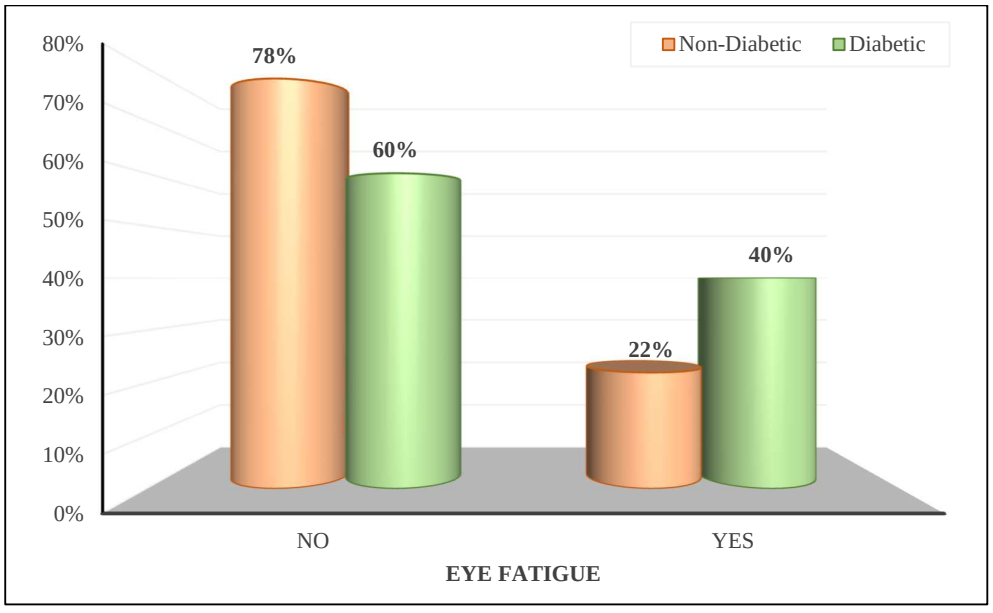
Graph 3: Distribution of Burning over groups.

Irritation was another common complaint, experienced by 16% of Non-Diabetic patients and 31% of Diabetic patients.



Graph 4: Distribution of Irritation over groups.

The most common symptom noticed was eye fatigue, which was seen in 22% of Non-Diabetic population and 40% of Diabetic population.



Graph 5: Distribution of Eye fatigue over groups

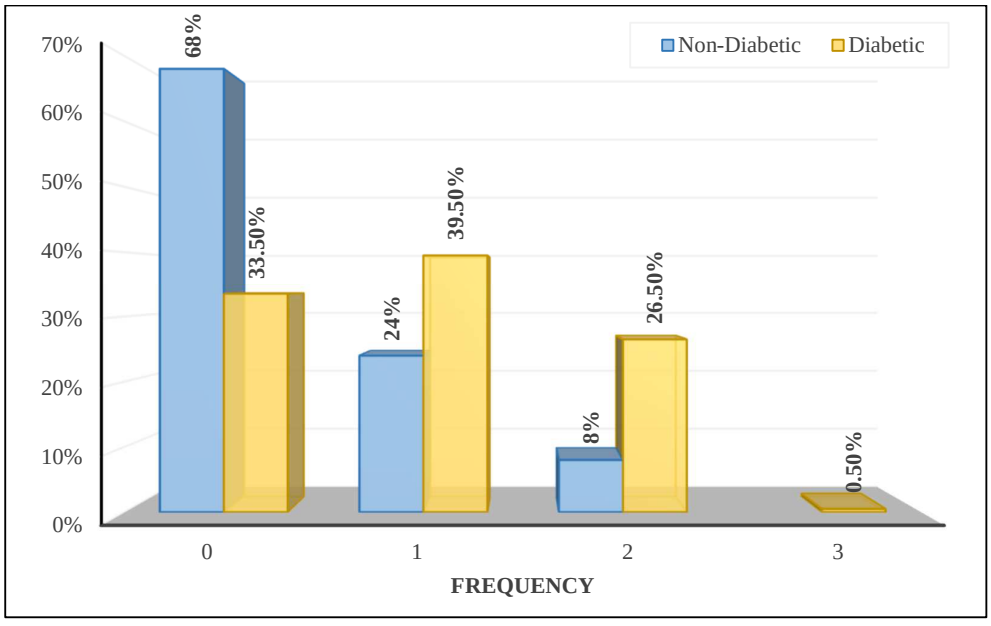
The frequency of the above-mentioned symptoms is as follows.

Variables	Sub Category	Group		Total
		Non-Diabetic	Diabetic	
Frequency	0	136 (68%)	67 (33.5%)	203 (50.75%)
	1	48 (24%)	79 (39.5%)	127 (31.75%)
	2	16 (8%)	53 (26.5%)	69 (17.25%)
	3	0	1 (0.5%)	1 (0.25%)

0 = Never, 1 = Sometimes, 2 = Often, 3 = Constant

Table 5: Distribution of patients based on frequency of symptoms

The frequency with which these symptoms were encountered was higher in Diabetic patients than the Non-Diabetic patients.



Graph 6: Distribution of frequency over groups.

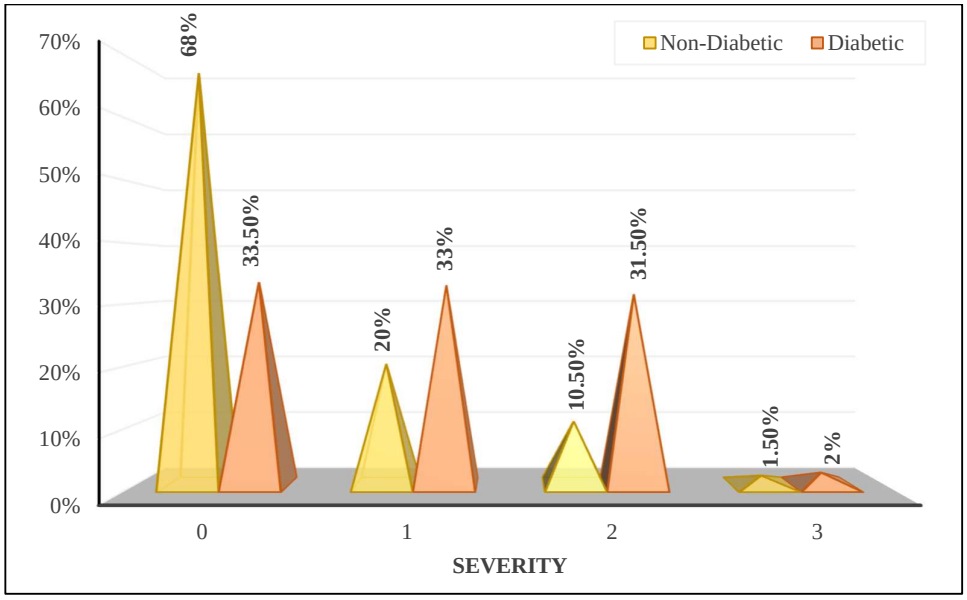
The severity with which these symptoms were experienced is as follows.

Variables	Sub Category	Group		Total
		Non-Diabetic	Diabetic	
Severity	0	136 (68%)	67 (33.5%)	203 (50.75%)
	1	40 (20%)	66 (33%)	106 (26.5%)
	2	21 (10.5%)	63 (31.5%)	84 (21%)
	3	3 (1.5%)	4 (2%)	7 (1.75%)

0 = No Problem, 1 = Tolerable - not perfect, but not uncomfortable; **2** = Uncomfortable - irritating, but does not interfere with my day; **3** = Bothersome - irritating and interferes with my day; **4** = Intolerable - unable to perform my daily tasks

Table 6: Distribution of patients based on severity of symptoms

The severity charting also indicates that the Diabetic population was more affected than the Non-diabetic population.



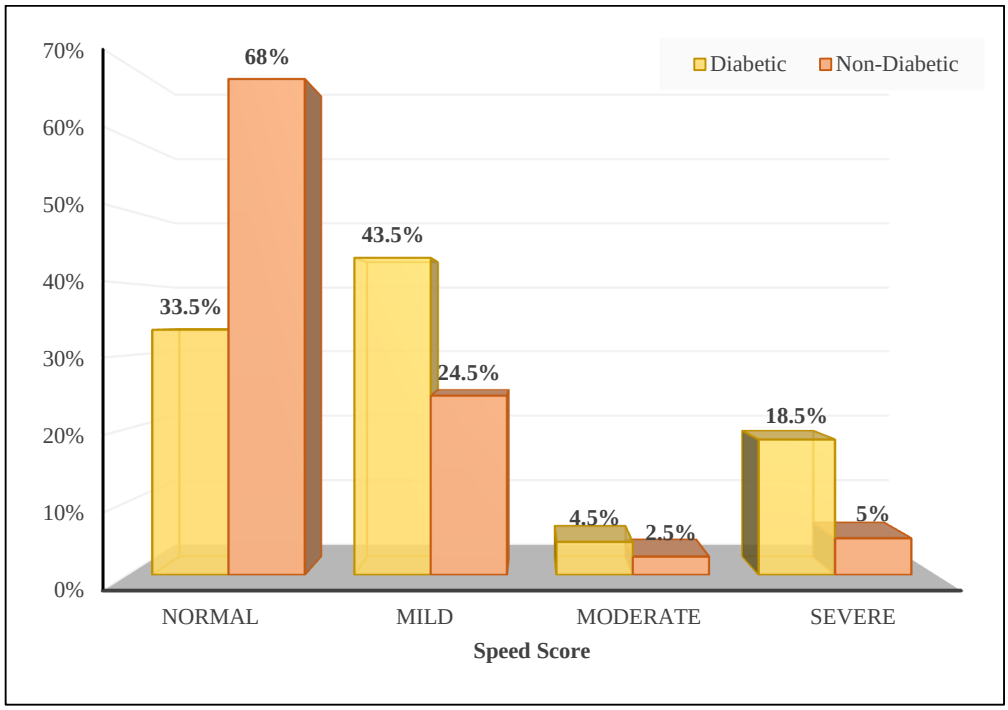
Graph 7: Distribution of severity over groups.

Based on the SPEED Questionnaire, and the SPEED scoring system, The two groups were classified as Normal, Mild DED, Moderate DED and Severe DED.

Variables	Sub Category	Group		Total
		Non-Diabetic	Diabetic	
SPEED Score	Normal	136 (68%)	67 (33.5%)	203 (50.75%)
	Mild	49 (24.5%)	87 (43.5%)	136 (34%)
	Moderate	5 (2.5%)	9 (4.5%)	14 (3.5%)
	Severe	10 (5%)	37 (18.5%)	47 (11.75%)

Table 7: Distribution of patients based on SPEED Score

The SPEED score was in the normal range in case of 68% non-diabetics and 33.5% diabetics. Mild DED was indicated in 43.5% Diabetics and 24.5% Non-diabetics". As the score proceeded towards moderate and severe DED the diabetic population showed a larger number of cases. Moderate DED was seen in 4.5% of Diabetics whereas, severe DED was seen in 18.5% Diabetic cases, which was significantly higher than the non-diabetic counterparts.



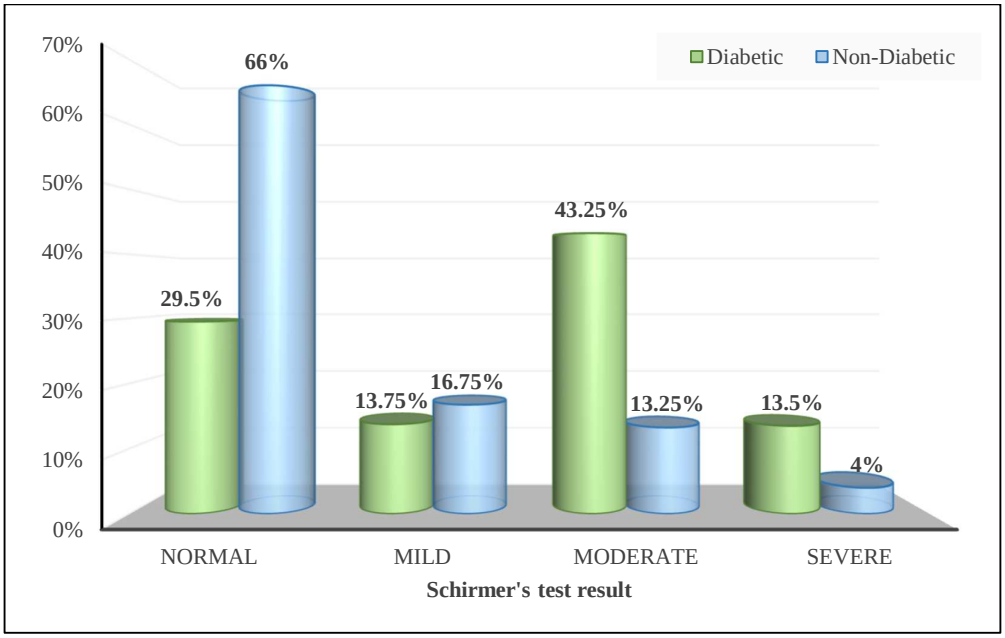
Graph 8: Distribution of Dry eye based on speed score over groups.

Schirmer's II test was done and the results were as follows.

Variables	Sub Category	Groups		Total
		Diabetic	Non-Diabetic	
Schirmer's II Test	Normal	118 (29.5%)	264 (66%)	382 (47.75%)
	Mild	55 (13.75%)	67 (16.75%)	122 (15.25%)
	Moderate	173 (43.25%)	53 (13.25%)	226 (28.25%)
	Severe	54 (13.5%)	16 (4%)	70 (8.75%)
	Mean ± SD	11.61 ± 5.69	17.89 ± 7.07	14.75 ± 7.14
	Median (Min, Max)	10 (2, 32)	18 (2, 35)	15 (2, 35)

Table 8: Distribution of patients based on Schimer's II Test

According to Schirmer's II test, 29.5% diabetics and 66% non-diabetics had normal Schirmer's values and no DED. Mild DED was seen in 13.75% diabetics and 16.75% non-diabetics. Moderate DED was indicated in 43.25% diabetics and 13.25% non-diabetics. 13.5% diabetics and 4% non-diabetics showed severe DED.



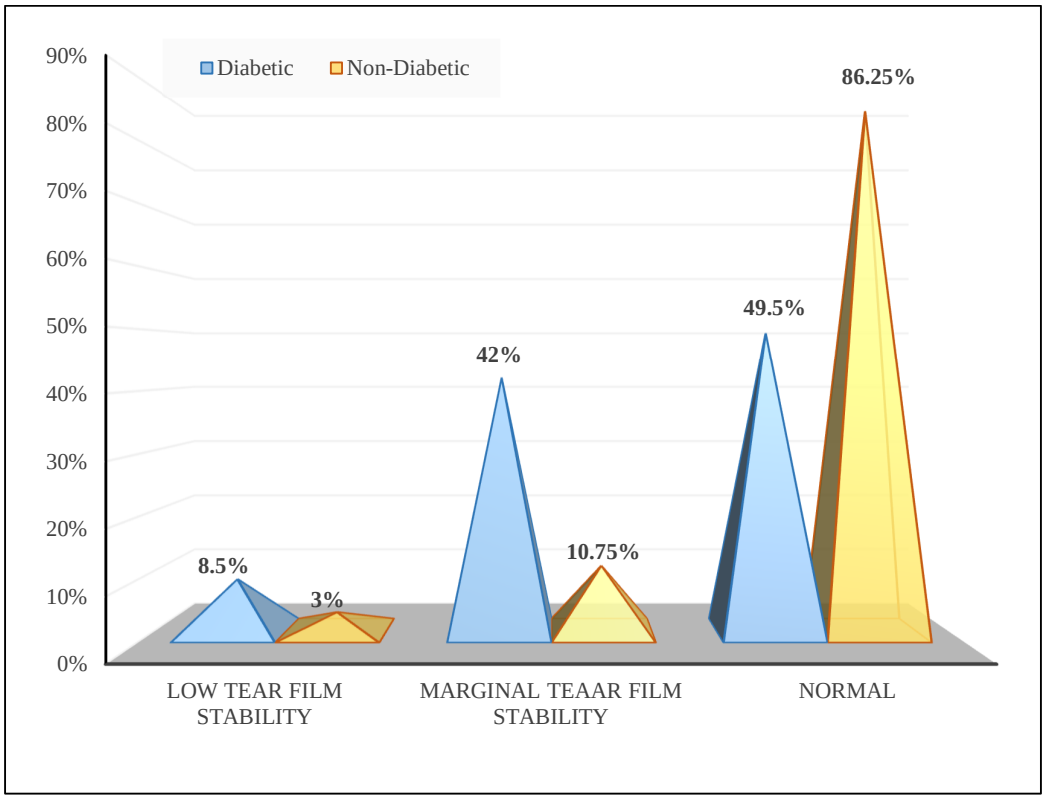
Graph 9: Distribution of Dry eye based on Schirmer's test result over groups.

TBUT results were as follows.

Variables	Sub Category	Groups		Total
		Diabetic	Non-Diabetic	
TBUT	Low tear film stability	34 (8.5%)	12 (3%)	46 (5.75%)
	Marginal tear film stability	168 (42%)	43 (10.75%)	211 (26.38%)
	Normal	198 (49.5%)	345 (86.25%)	543 (67.88%)
	Mean ± SD	12.43 ± 5.32	16.46 ± 4.55	14.44 ± 5.34
	Median (Min, Max)	10 (3, 28)	17 (2, 27)	16 (2, 28)

Table 9: Distribution of patients based on TBUT

On the basis of TBUT, low tear film stability was noted in 8.5% diabetics and 3% non-diabetics. Marginal tear film stability was seen in 42% diabetics and 10.5% non-diabetics. However 49.5% diabetics and 86.25% non-diabetics showed normal tear film stability.



Graph 10: Distribution of Dry eye based on TBUT result over groups

The final grading of DED was done taking into consideration both the Schirmer's II test results and the TBUT results.

- Normal eyes/ No DED – Schirmer's test result >15mm and TBUT >10s.
- Mild DED – Schirmer's test result between 10-15mm and TBUT >10s.
- Moderate DED – Schirmer's test result between 5-10mm and TBUT 5-10s.
- Severe DED – Schirmer's test result between <5mm and TBUT <5s.

Dry eye grade	Number of eyes (%)
Normal	382 (47.75%)
Mild	181 (22.63%)
Moderate	222 (27.75%)
Severe	15 (1.88%)

Table 10: Distribution of patients based on grade of DED

The above table shows that, out of the total 800 eyes assessed for DED, 382 eyes (47.75%) were normal, i.e., with no evidence of DED. Mild DED was seen in 181 eyes (22.63%), Moderate DED was seen in 222 eyes (27.75%) and Severe DED was seen in 15 eyes (1.88%)

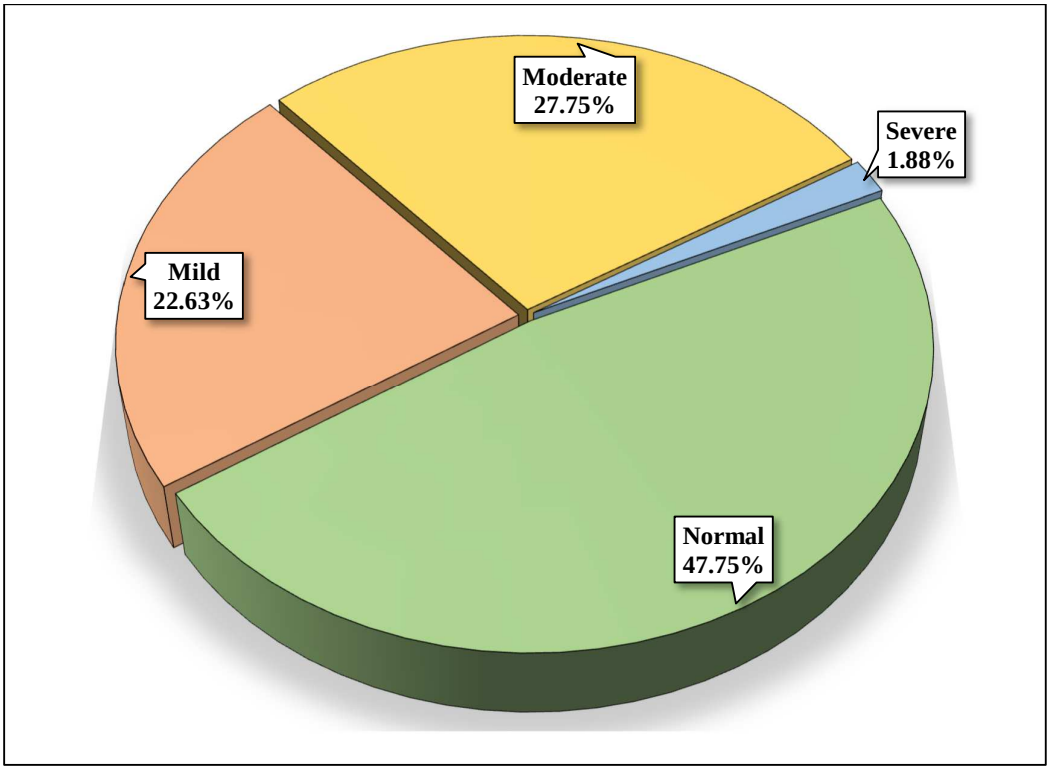


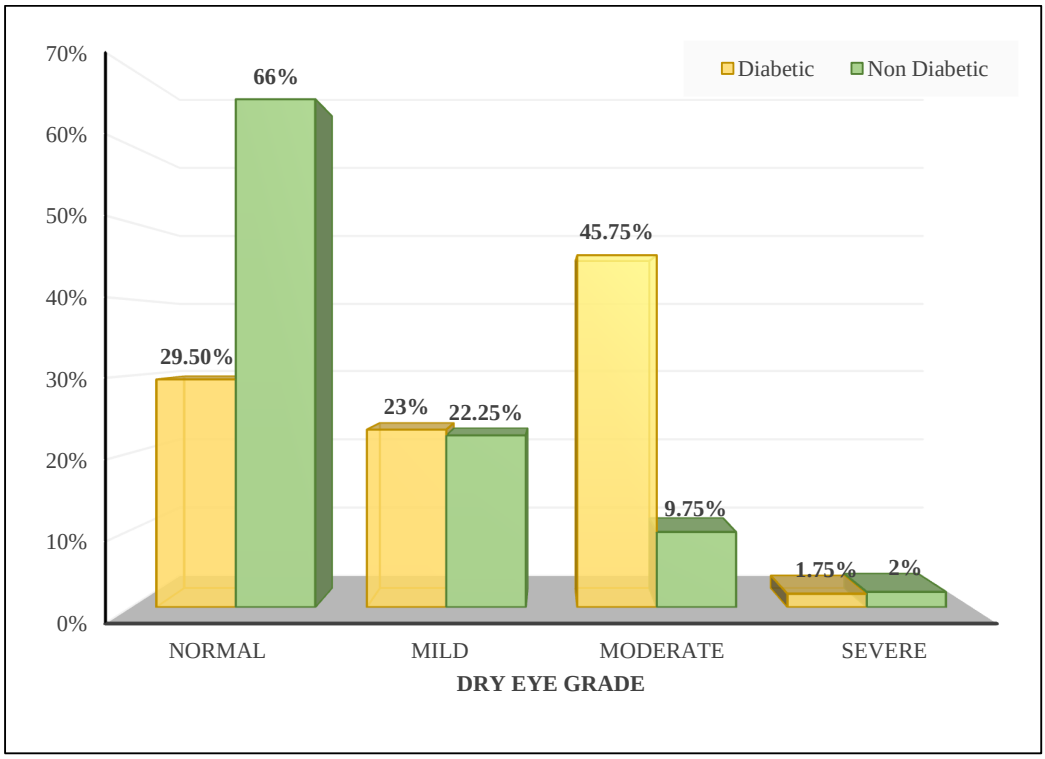
Figure 20: Distribution of eyes according to dry eye grade.

The following table gives the details of distribution of DED across the 2 groups, Diabetics and Non-Diabetics.

Dry eye grade	Groups		Total
	Diabetic	Non-Diabetic	
Normal	118 (29.5%)	264 (66%)	382 (47.75%)
Mild	92 (23%)	89 (22.25%)	181 (22.63%)
Moderate	183 (45.75%)	39 (9.75%)	222 (27.75%)
Severe	8 (2%)	7 (1.75%)	15 (1.88%)

Table 11: Distribution of Dibetics and Non Diabetics with respect to DED

Out of the total 382 normal eyes, 118 (29.5%) were of diabetic patients and 264 (66%) were of non-diabetic patients. Of the 418 patients who were diagnosed with DED, mild DED was seen in 92 eyes (23%) of diabetic patients and 89 eyes (22.25%) of non-diabetic patients. Moderate DED was seen in 183 eyes (45.75%) of diabetic patients and 39 eyes (9.75%) of non-diabetic patients. Severe DED was seen in 8 eyes (2%) of diabetic patients and 7 eyes (1.75%) of non-diabetic patients.



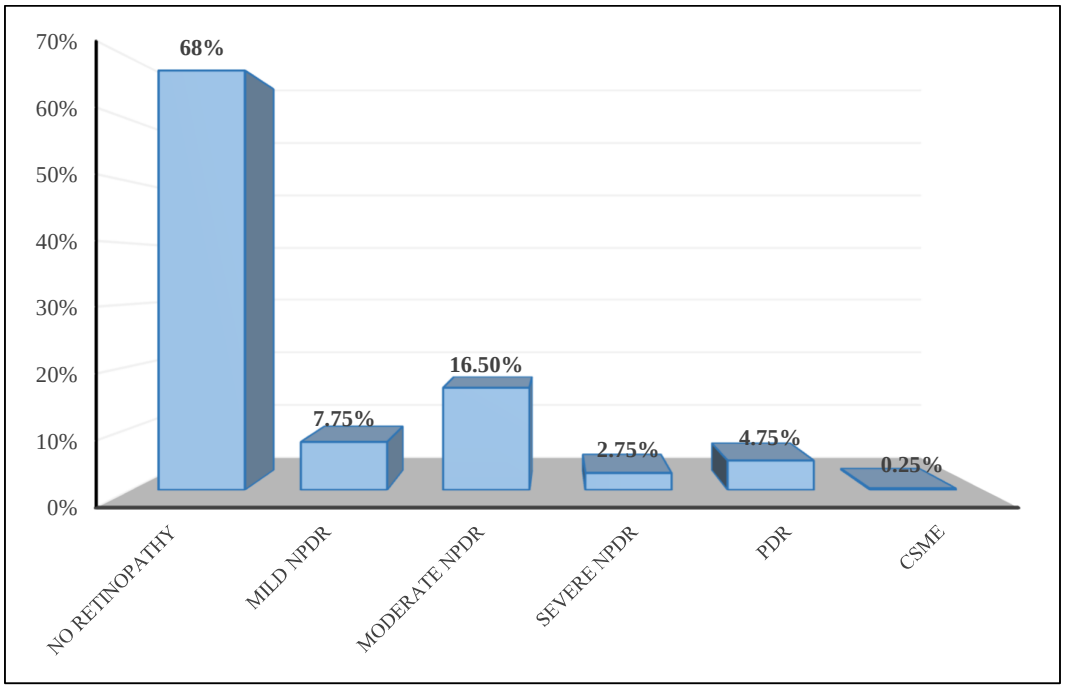
Graph 11: Distribution of Dry eye grade over groups.

Fundus examination was done and 400 eyes of the 200 patients included in the diabetic group were classified using the ETDRS Classification into the following groups.

ETDRS Grade	Number of Eyes (%)
No Retinopathy	272 (68%)
Mild NPDR	31 (7.75%)
Moderate NPDR	66 (16.5%)
Severe NPDR	11 (2.75%)
PDR	19 (4.75%)
CSME	1 (0.25%)

Table 12: Distribution of patients based on grade of DR

According to the ETDRS Classification, 272 eyes (68%) showed no evidence of diabetic retinopathy. 31 eyes (7.75%) had changes of Mild NPDR, 66 eyes (16.5%) showed changes of Moderate NPDR and 11 eyes (2.75%) showed changes of Severe NPDR. PDR was seen in 19 eyes (4.75%) and CSME or diabetic maculopathy was seen in 1 eye (0.25%).



Graph 12: Distribution of subjects in diabetic group with respect to ETDRS Grade.

In diabetic patients, the grades of Diabetic Retinopathy and the severity of DED were taken into consideration to assess if any association existed.

ETDRS Grade	Severity of Dry eye			
	Normal	**Mild**	**Moderate**	**Severe**
No Retinopathy	118 (100%)	67 (72.83%)	85 (46.45%)	2 (28.57%)
Mild NPDR	0	6 (6.52%)	24 (13.11%)	1 (14.29%)
Moderate NPDR	0	16 (17.39%)	49 (26.78%)	1 (14.29%)
Severe NPDR	0	0	11 (6.01%)	0
PDR	0	3 (3.26%)	13 (7.1%)	3 (42.86%)
CSME	0	0	1 (0.55%)	0

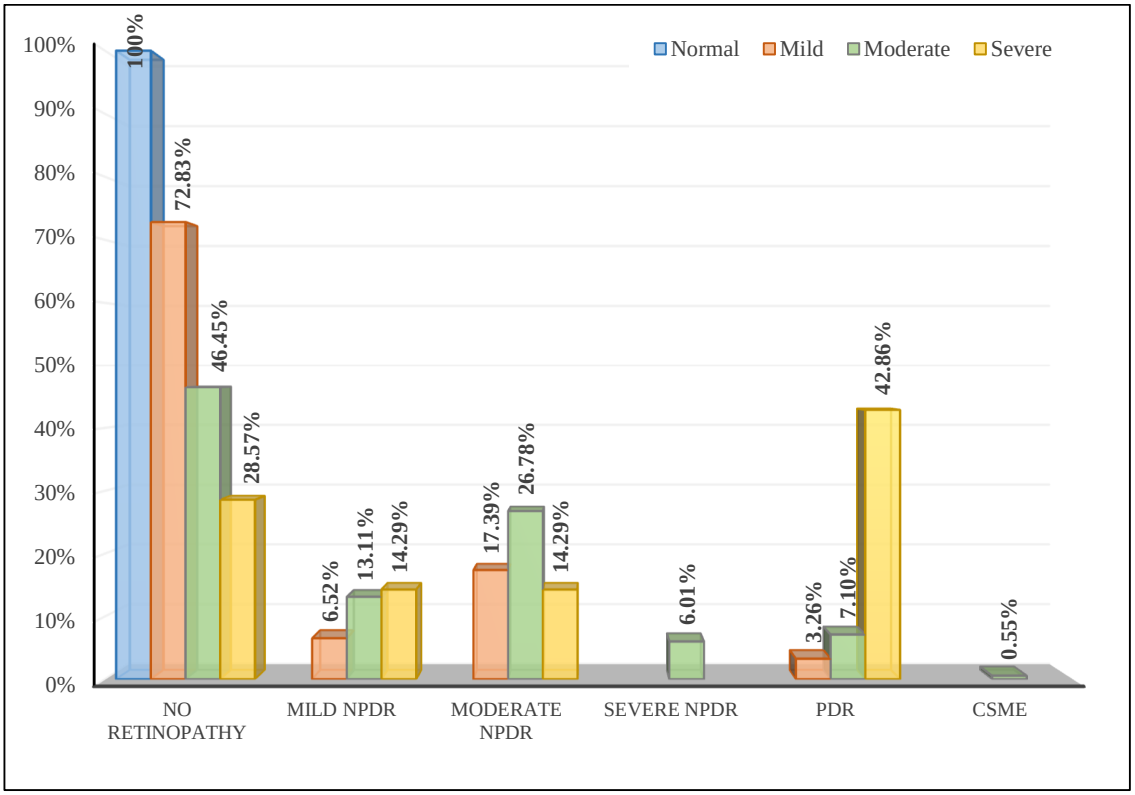
Table 13: Distribution of patients with different grades of DR over severity of DED

In the 400 eyes examined and assessed, 118 were eyes which had no DED. In these eyes, when the fundus examination was done, no evidence Diabetic Retinopathy was noted. In eyes having DED, no retinopathy was seen in 72.83% of patients with mild DED, 46.45% of moderate DED and 28.57% of severe DED.

Mild DED was seen in 6.52% cases of mild NPDR, 17.39% cases of moderate NPDR, 0% cases of severe NPDR and 3.26% cases of PDR.

Moderate DED was seen in 13.11% cases of mild NPDR, 26.78% cases of moderate NPDR, 6.01% cases of severe NPDR and 7.1% cases of PDR. Moderate DED was also seen in 1 eye, 0.55% of a case of diabetic maculopathy.

Severe DED was seen in 14.29% of cases of both mild and moderate NPDR each and in 42.86% of cases of PDR.



Graph 13: Distribution of grade of diabetic retinopathy according to severity of dry eye in diabetic patients who had dry eye.

Corneal Nerve Sensitivity (CNS) assessment was done in both the groups – diabetics as well as non-diabetics and the following results were noted.

Variables	Sub Category	Groups		Total
		Diabetic	Non-Diabetic	
Corneal Nerve Sensitivity	NORMAL	212 (53%)	388 (97%)	600 (75%)
	REDUCED	188 (47%)	12 (3%)	200 (25%)

Table 14: Distribution of CNS with respect to Diabetics and Non Diabetics

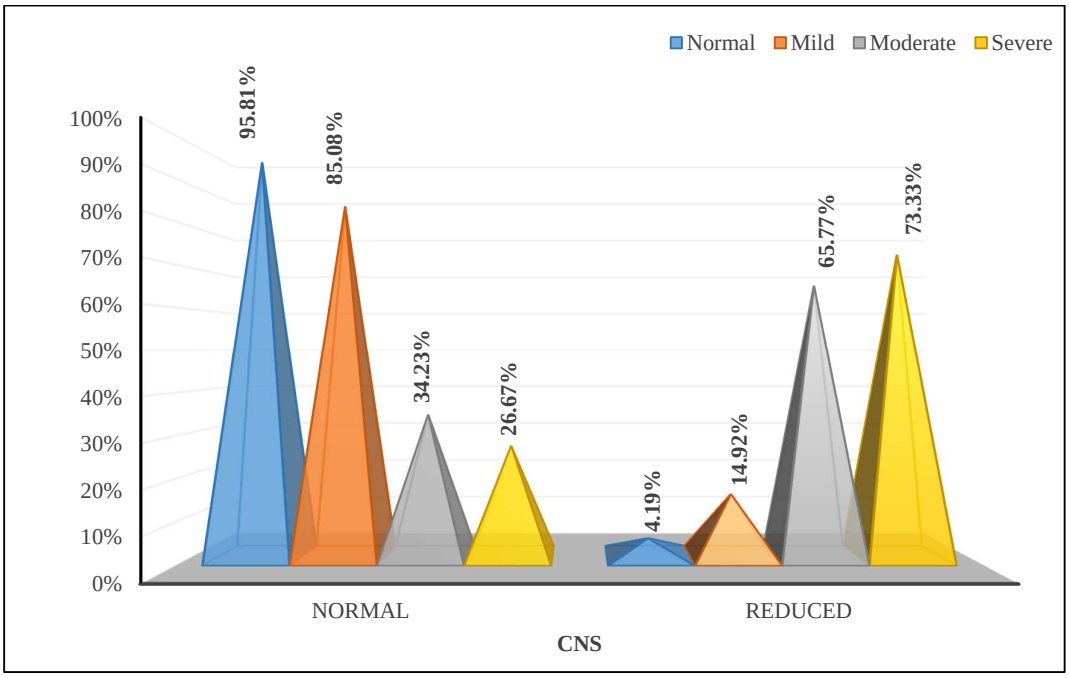
Out of 800 eyes, 200 eyes (25%) had reduced CNS. CNS was reduced in 188 eyes (47%) of diabetic patients and 12 eyes (3%) of non-diabetic patients.

Association of CNS was done with the severity of DED and the following was noted.

Corneal Nerve Sensitivity	Severity of Dry eye			
	Normal	Mild	Moderate	Severe
Normal	366 (95.81%)	154 (85.08%)	76 (34.23%)	4 (26.67%)
Reduced	16 (4.19%)	27 (14.92%)	146 (65.77%)	11 (73.33%)

Table 15: Distribution of Corneal Nerve Sensitivity with respect to severity of DED

Out of the 800 eyes assessed, Corneal nerve sensitivity was reduced in 4.19% of normal eyes, 14.92% of eyes with Mild DED, 65.77% of eyes with Moderate DED and 73.33% of eyes with Severe DED.



Graph 14: Distribution of Corneal Nerve Sensitivity with dry eye.

DISCUSSION

In recent years, there has been extensive dialogue about ocular surface disorders. The molecular basis of ocular surface diseases is the subject of extensive research into the ocular surface's molecular anatomy.

Over the past several decades, researchers have been trying to learn more about the pathological processes that link diabetes and dry eye syndrome (DMDES)..[2] Patients with diabetes who experience DED report similar symptoms to those who do not have diabetes, with the only difference being an increase in severity. Patients who have trouble keeping their blood sugar levels under control may experience a worsening of DED symptoms..[62] The diagnostic modalities available for the evaluation of DMDES are same as that of DED. No single targeted tool exists for this entity.[2]

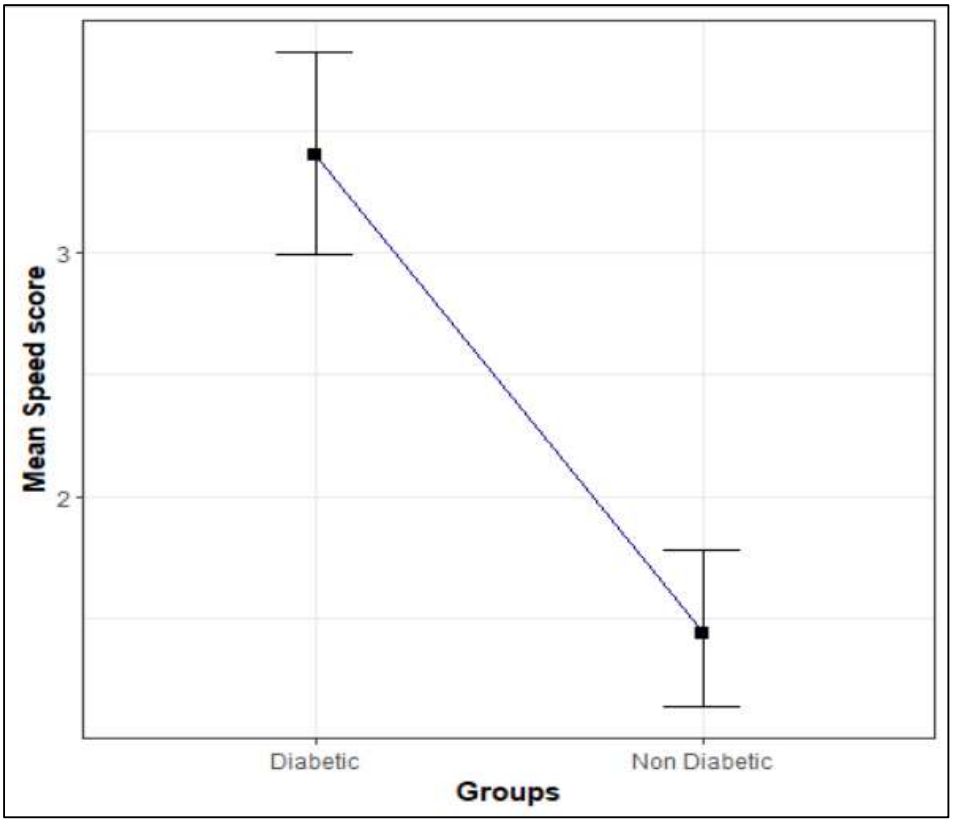
The ocular surface and its disorders are profoundly impacted by sociodemographic variables.[65].

A total of 400 patients were enrolled in the study, spanning an age range from 30 to 87 years old (the mean age was 55.02). Patients were split into two categories: those with diabetes and those without; the two groups had similar median ages (55.5 and 56 years, respectively).

In the non-diabetic group, 47.5% were women and 52.5% were men; in the diabetic group, 45.5% were women and 54.5% were men. With a p value of 0.6884, there was no statistically significant difference in the male to female ratio between the two groups.

Employees (those with sedentary jobs), farmers, and educators made up the bulk of the workforce, and they shared many of the same environmental hazards.

The SPEED Questionnaire was used for subjective testing of DED symptoms. The frequency and severity of grittiness, burning, irritation, and eye fatigue were assessed through a questionnaire. As can be seen in the graph below, the results of this subjective evaluation show that the SPEED score was normal in 68.2% of the non-diabetic population and 31.5% of the diabetic population. In the diabetic population, 43.5% of patients experienced mild symptoms, 4.5% experienced moderate symptoms, and 18.5% experienced severe symptoms of dry eye, compared to 24.5%, 2.5%, and 5%, respectively, in the control population. Mild DED symptoms were more common in both groups, but the Diabetic group had a higher prevalence of symptoms overall.

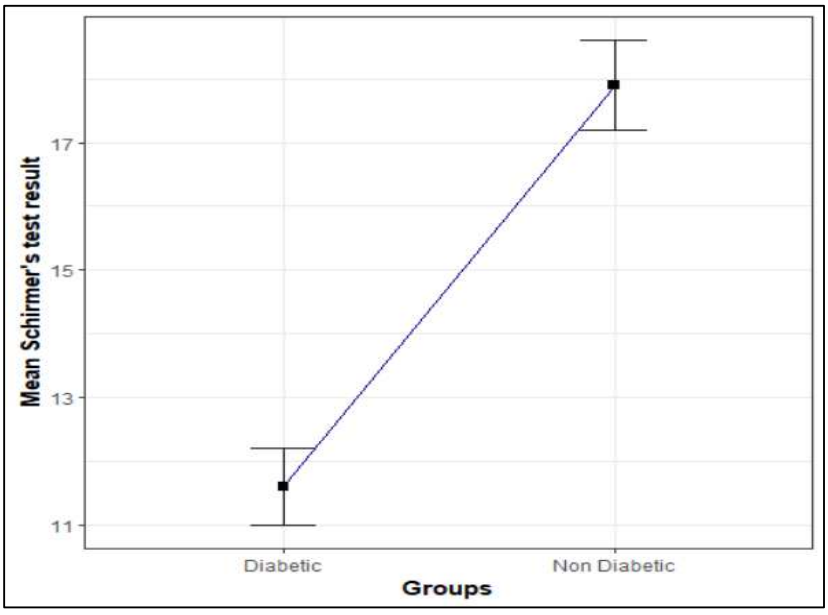


Graph 15: Mean plot of speed score over groups.

This is consistent with another study done by Y Lyu et al, where the median SPEED Score was 5 and 6 in the non-diabetic and diabetic group respectively.[65] Dry

eye symptoms were evaluated with the Ocular Surface Disease Index (OSDI) questionnaire in a study by Divya K et al, which found that the OSDI score was also higher in the diabetic group compared to the non-diabetic group.[34]

Schirmer's II test results showed that 29.5% of diabetics had normal results, while 66% of non-diabetics did. The prevalence of mild DED was similar between the diabetic and non-diabetic groups, with 21.75 percent in the diabetic group and 21 percent in the non-diabetic group, according to the results of Schirmer's II. There were significantly more cases of moderate and severe DED in the diabetic group (39.5% and 9.25%, respectively) compared to the non-diabetic group (9.5%). Schirmer's II values were 11.61 mm (SD 5.69) and 17.89 mm (SD 7.07) in the diabetic and non-diabetic groups, respectively. Our results showed that mild DED was also present in the non-diabetic population, but that the prevalence of moderate and severe DED was higher in the diabetic population.

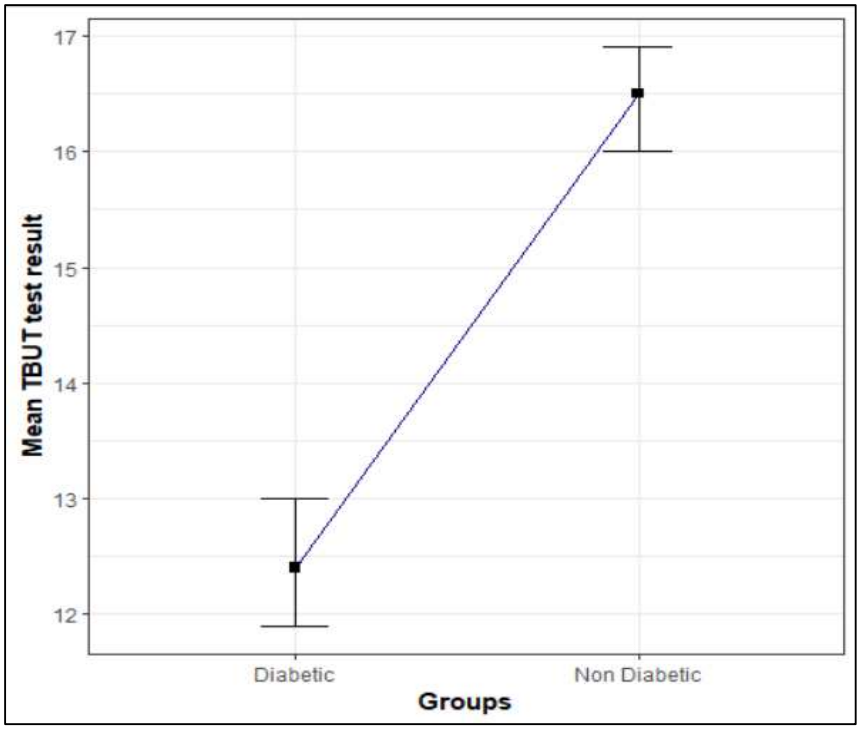


Graph 16: Mean plot of Schirmer's test result over groups.

This is consistent with the results of other studies, such as that conducted by Dr. Rehman and colleagues, who discovered that more than 60.86 percent of diabetic

patients had a positive Schirmer's I test. Sixteen percent of diabetic patients had a Schirmer's test score of less than 10 according to data from Hasan et al..[68] Schirmer's values were about 7.4mm in diabetic patients compared to 13.53mm in non-diabetic patients in a different study by K Divya et al.[34]

The results of the TBUT showed that only 49.5% of diabetic patients and 86.25%; of non-diabetic patients had normal tear film stability. Mean TBUT values were 12.43 seconds with a SD of 5.32 seconds in the diabetic group and 16.46 seconds with a SD of 4.55 seconds in the non-diabetic group. Results showed that tear film stability in diabetic patients was significantly lower than in the non-diabetic group.



Graph 17: Mean plot of TBUT test result over groups.

Research by Dr. Rajender S. Chauhan et al. corroborates these values, showing that the TBUT in the diabetic group is less than 10 seconds. Hasan et al. found that 67% of diabetic patients had TBUTvalue of less than 10 seconds. Another study by Seifart et al. found that 94.2% of diabetic patients had TBUT value of less than 10 seconds..[68] There were notable differences between diabetic and non-diabetic patients, as demonstrated by the fact that 30% of diabetics had TBUTs of less than 10 seconds, according to a study by K Divya et al.[34]

We evaluated corneal nerve sensations (CNS) in relation to DED in this study. 4.19 percent of healthy people, 14.04 percent of those with mild DED, 58.67 percent of those with moderate DED, and 88.1 percent of those with severe DED were found to have diminished CNS. This suggests that eyes with advanced DED are more likely to have a diminished central nervous system.

When Y. Lyu et al. evaluated the corneal sensitivity and corneal nerve fibre length, they also found that DED had a major impact, especially in the diabetic group.[65]

We also established a connection between DED and DR severity in diabetic patients. When looking across all ETDRS categories, we discovered that moderate DED was by far the most common form of the DR. However, severe DED was the norm among PDR patients.

These results are consistent with those found by Geetika Khurrana et al., who found a positive correlation between the severity of dry eye syndrome and DR.[64] O Revanna Reddy et al. found a similar correlation between DED severity and DR stages.

DED is becoming more widely recognised as a complication of diabetes as the incidence of the disease rises. Throughout the years, multiple subjective and objective methods of gauging DED have emerged. There is no one "gold standard" test, though. According to our findings, DED is significantly more common in DM patients and its severity is correlated with DM severity.

CONCLUSION

The aim of this study was to find a correlation between Diabetes Mellitus and Dry Eye Disease, the associated impact on Corneal Nerve Sensitivity and correlation between the severity of both these diseases. In our study we found that patients with Diabetes Mellitus are at an increased risk for developing Dry Eye Disease compared to those without this comorbidity. The assessment of corneal nerve sensitivity indicated that there is a significantly reduced sensitivity in patients diagnosed with Diabetes Mellitus. Furthermore, the occurrence of Dry Eye Disease exaggerated this problem. It was noted that patients with a higher severity of Dry Eye Disease were more likely to have a reduced corneal nerve sensitivity. Also, diabetic patients who have progressed to suffer from Diabetic Retinopathy are likely to experience more serious forms of ocular surface diseases. Diabetes Mellitus associated Dry Eye Disease is an upcoming entity and more research is needed in this area to determine the best strategies for preventing this condition and ultimately preventing further visual impairment.

BIBLIOGRAPHY

1. S A, J D, S M, A G, L.K S. Dry Eye- Study of Prevalence, Associated Risk Factors and Frequency of Symptoms in Meerut District. jemds. 2019 Nov 11;8(45):3382–3386.

2. Aggarwal M, Goud R, Radhakrishnan OK, Mantri P, Shah A. Prevalence of dry eyes in patients with Type-2 diabetes mellitus. Indian Journal of Clinical and Experimental Ophthalmology. 2020 Jun 15;6(2):286–290.

3. Dry eye disease in India : Indian Journal of Ophthalmology [Internet]. LWW. [cited 2022 Sep 20]. Available from: https://journals.lww.com/ijo/Fulltext/2020/68070/Dry_eye_disease_in_India.83.aspx

4. Pr D, P K, Ss S, V S, Av D, S B. Incidence, demographics, types and risk factors of dry eye disease in India: Electronic medical records driven big data analytics report I. The ocular surface [Internet]. Ocul Surf; 2019 Apr [cited 2022 Sep 20];17(2). Available from: https://pubmed.ncbi.nlm.nih.gov/30802671/ PMID: 30802671

5. Pradeepa R, Mohan V. Epidemiology of type 2 diabetes in India. Indian Journal of Ophthalmology. Wolters Kluwer -- Medknow Publications; 2021 Nov;69(11):2932. PMID: 34708726

6. Bansal V, Kalita J, Misra UK. Diabetic neuropathy. Postgraduate Medical Journal. BMJ Publishing Group; 2006 Feb;82(964):95. PMID: 16461471

7. X Z, Y L, Z G, Z J, C Z, X L, C C, Y G, Y N, Y W, Y Z, S Z. The Effects of Diabetic Duration on Lacrimal Functional Unit in Patients with Type II Diabetes.

Journal of ophthalmology [Internet]. J Ophthalmol; 2019 Jan 10 [cited 2022 Sep 20];2019. Available from: https://pubmed.ncbi.nlm.nih.gov/30766731/ PMID: 30766731

8. S P, J H, D M, A G. Corneal Nerve Abnormalities in Ocular and Systemic Diseases. Experimental eye research [Internet]. Exp Eye Res; 2021 Jan [cited 2022 Sep 20];202. Available from: https://pubmed.ncbi.nlm.nih.gov/33045221/ PMID: 33045221

9. J S. Definition and Diagnostic Criteria of Dry Eye Disease: Historical Overview and Future Directions. Investigative ophthalmology & visual science [Internet]. Invest Ophthalmol Vis Sci; 2018 Nov 1 [cited 2022 Sep 20];59(14). Available from: https://pubmed.ncbi.nlm.nih.gov/30481800/ PMID: 30481800

10. Mdp W, P A, Ga G, Jm H, Gw L, Tj M, Eb P, Jp R, Ta S, U S, T S, Ln S, Oö U, L J. TFOS DEWS II Tear Film Report. The ocular surface [Internet]. Ocul Surf; 2017 Jul [cited 2022 Sep 20];15(3). Available from: https://pubmed.ncbi.nlm.nih.gov/28736338/ PMID: 28736338

11. Jp C, Kk N, Ek A, B C, Hs D, Ck J, Z L, Jd N, Jj N, K T, F S. TFOS DEWS II Definition and Classification Report. The ocular surface [Internet]. Ocul Surf; 2017 Jul [cited 2022 Sep 20];15(3). Available from: https://pubmed.ncbi.nlm.nih.gov/28736335/ PMID: 28736335

12. Sridhar MS. Anatomy of cornea and ocular surface. Indian Journal of Ophthalmology. Wolters Kluwer -- Medknow Publications; 2018 Feb;66(2):190. PMID: 29380756

13. Shumway CL, Motlagh M, Wade M. Anatomy, Head and Neck, Eye Conjunctiva [Internet]. StatPearls [Internet]. StatPearls Publishing; 2021 [cited 2022 Oct 3]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK519502/ PMID: 30137787

14. Gipson IK. Goblet Cells of the Conjunctiva: A Review of Recent Findings. Progress in retinal and eye research. NIH Public Access; 2016 Sep;54:49. PMID: 27091323

15. eOphtha. Anatomy of Cornea [Internet]. [cited 2022 Oct 3]. Available from: https://www.eophtha.com/posts/anatomy-of-cornea

16. Y U. The Ocular Surface Glycocalyx and its Alteration in Dry Eye Disease: A Review. Investigative ophthalmology & visual science [Internet]. Invest Ophthalmol Vis Sci; 2018 Nov 1 [cited 2022 Sep 20];59(14). Available from: https://pubmed.ncbi.nlm.nih.gov/30481821/ PMID: 30481821

17. 2020–2021 BCSC Basic and Clinical Science Course™ [Internet]. [cited 2022 Oct 3]. Available from: https://www.aao.org/bcscsnippetdetail.aspx?id=892ab460-fb9a-4972-b271-3e8f06dec26e

18. Jm T. The normal tear film. Developments in ophthalmology [Internet]. Dev Ophthalmol; 2008 [cited 2022 Sep 20];41. Available from: https://pubmed.ncbi.nlm.nih.gov/18453758/ PMID: 18453758

19. X Z, Y L, Z G, Z J, C Z, X L, C C, Y G, Y N, Y W, Y Z, S Z. The Effects of Diabetic Duration on Lacrimal Functional Unit in Patients with Type II Diabetes. Journal of ophthalmology [Internet]. J Ophthalmol; 2019 Jan 10 [cited 2022 Oct

3];2019. Available from: https://pubmed.ncbi.nlm.nih.gov/30766731/ PMID: 30766731

20. Kanski's Clinical Ophthalmology E-Book [Internet]. [cited 2022 Oct 3]. Available from: https://books.google.com/books/about/Kanski_s_Clinical_Ophthalmology_E_Book.html?id=HXW7DwAAQBAJ

21. Hodges RR, Dartt DA. Tear Film Mucins: Front Line Defenders of the Ocular Surface; Comparison with Airway and Gastrointestinal Tract Mucins. Experimental eye research. NIH Public Access; 2013 Dec;117:62. PMID: 23954166

22. Review of Ophthalmology E-Book [Internet]. [cited 2022 Oct 3]. Available from: https://books.google.com/books/about/Review_of_Ophthalmology_E_Book.html?id=D4jKDQAAQBAJ

23. Shetgar AC, Viswanathan V, Patil BA, Kulkarni KR, S HD. Tear film changes in diabetic retinopathy: A hospital based case control study. Indian Journal of Clinical and Experimental Ophthalmology. 2020 Jun 15;6(2):252–255.

24. Da D. Neural regulation of lacrimal gland secretory processes: relevance in dry eye diseases. Progress in retinal and eye research [Internet]. Prog Retin Eye Res; 2009 May [cited 2022 Sep 20];28(3). Available from: https://pubmed.ncbi.nlm.nih.gov/19376264/ PMID: 19376264

25. C W, Rj B, Aj B. Central Connections of the Lacrimal Functional Unit. Cornea [Internet]. Cornea; 2017 Aug [cited 2022 Sep 20];36(8). Available from: https://pubmed.ncbi.nlm.nih.gov/28628505/ PMID: 28628505

26. Aj B, Jm T, Sm G, N Y, Lw V. Functional aspects of the tear film lipid layer. Experimental eye research [Internet]. Exp Eye Res; 2004 Mar [cited 2022 Sep 20];78(3). Available from: https://pubmed.ncbi.nlm.nih.gov/15106912/ PMID: 15106912

27. L C. Tear film lipid layer: A molecular level view. Biochimica et biophysica acta [Internet]. Biochim Biophys Acta; 2016 Oct [cited 2022 Sep 20];1858(10). Available from: https://pubmed.ncbi.nlm.nih.gov/26898663/ PMID: 26898663

28. S G, S G, M A, S B, H D, A D. Change in tear protein profile in diabetic retinopathy with duration of diabetes. Diabetes & metabolic syndrome [Internet]. Diabetes Metab Syndr; 2014 Dec [cited 2022 Sep 20];8(4). Available from: https://pubmed.ncbi.nlm.nih.gov/25311821/ PMID: 25311821

29. Rj B, Pe KS, Cg B, L L, Nr G. Dynamics and function of the tear film in relation to the blink cycle. Progress in retinal and eye research [Internet]. Prog Retin Eye Res; 2015 Mar [cited 2022 Sep 20];45. Available from: https://pubmed.ncbi.nlm.nih.gov/25479602/ PMID: 25479602

30. Da D. Regulation of tear secretion. Advances in experimental medicine and biology [Internet]. Adv Exp Med Biol; 1994 [cited 2022 Sep 20];350. Available from: https://pubmed.ncbi.nlm.nih.gov/8030459/ PMID: 8030459

31. Mtm W, L T, A H, Jm L, D K, M M, Jp C. Impact of blinking on ocular surface and tear film parameters. The ocular surface [Internet]. Ocul Surf; 2018 Oct [cited 2022 Oct 3];16(4). Available from: https://pubmed.ncbi.nlm.nih.gov/29883739/ PMID: 29883739

32. P R, R J, N G, N S, A M. Prevalence and trends of the diabetes epidemic in urban and rural India: A pooled systematic review and meta-analysis of 1.7 million adults. Annals of epidemiology [Internet]. Ann Epidemiol; 2021 Jun [cited 2022 Sep 20];58. Available from: https://pubmed.ncbi.nlm.nih.gov/33727086/ PMID: 33727086

33. Patil S, Bishnoi S, Joshi A. Study of dry eye parameters in diabetics in a tertiary care hospital. Medical Journal of Dr DY Patil Vidyapeeth. 2021 Mar 1;14(2):155.

34. Divya. Dry eye disease and tear dysfunction in patients with type 2 diabetes: A hospital-based study from South India [Internet]. [cited 2022 Sep 20]. Available from:https://www.sjopthal.net/article.asp?issn=1858-540X;year=2019;volume=11;issue=1;spage=14;epage=18;aulast=Divya

35. Messmer EM. The Pathophysiology, Diagnosis, and Treatment of Dry Eye Disease. Deutsches Ärzteblatt International. Deutscher Arzte-Verlag GmbH; 2015 Jan;112(5):71. PMID: 25686388

36. J S. Definition and Diagnostic Criteria of Dry Eye Disease: Historical Overview and Future Directions. Investigative ophthalmology & visual science [Internet]. Invest Ophthalmol Vis Sci; 2018 Nov 1 [cited 2022 Oct 3];59(14). Available from: https://pubmed.ncbi.nlm.nih.gov/30481800/ PMID: 30481800

37. Dry Eye Syndrome questionnaires - EyeWiki [Internet]. [cited 2022 Oct 3]. Available from: https://eyewiki.aao.org/Dry_Eye_Syndrome_questionnaires

38. Diagnostic Testing for Dry Eye - EyeWiki [Internet]. [cited 2022 Oct 3]. Available from: https://eyewiki.aao.org/Diagnostic_Testing_for_Dry_Eye

39. Pauk SV, Petriček I, Jukić T, Popović-Suić S, Tomić M, Kalauz M, Jandroković S, Masnec S. NONINVASIVE TEAR FILM BREAK-UP TIME ASSESSMENT USING HANDHELD LIPID LAYER EXAMINATION INSTRUMENT. Acta Clinica Croatica. Sestre Milosrdnice University Hospital Center; 2019 Mar;58(1):63. PMID: 31363327

40. Cornea E-Book [Internet]. [cited 2022 Oct 3]. Available from: https://books.google.com/books/about/Cornea_E_Book.html?id=eR0PDQAAQBAJ

41. Diagnostic Testing for Dry Eye - EyeWiki [Internet]. [cited 2022 Oct 3]. Available from: https://eyewiki.aao.org/Diagnostic_Testing_for_Dry_Eye

42. S B, Jm BDC, T F, M L, N M, M M, Tp U, M R. Dry eye disease treatment: the role of tear substitutes, their future, and an updated classification. European review for medical and pharmacological sciences [Internet]. Eur Rev Med Pharmacol Sci; 2020 Sep [cited 2022 Sep 20];24(17). Available from: https://pubmed.ncbi.nlm.nih.gov/32964952/ PMID: 32964952

43. Am E, A L, Ad P. Punctal occlusion for dry eye syndrome. The Cochrane database of systematic reviews [Internet]. Cochrane Database Syst Rev; 2017 Jun 26 [cited 2022 Oct 3];6(6). Available from: https://pubmed.ncbi.nlm.nih.gov/28649802/ PMID: 28649802

44. Ervin AM, Wojciechowski R, Schein O. Punctal occlusion for dry eye syndrome. The Cochrane database of systematic reviews. NIH Public Access; (9):CD006775. PMID: 20824852

45. Sc P. Antiinflammatory therapy for dry eye. American journal of ophthalmology [Internet]. Am J Ophthalmol; 2004 Feb [cited 2022 Oct 3];137(2). Available from: https://pubmed.ncbi.nlm.nih.gov/14962426/ PMID: 14962426

46. Aragona P, Giannaccare G, Mencucci R, Rubino P, Cantera E, Rolando M. Modern approach to the treatment of dry eye, a complex multifactorial disease: a P.I.C.A.S.S.O. board review. British Journal of Ophthalmology. BMJ Publishing Group Ltd; 2021 Apr 1;105(4):446–453. PMID: 32703782

47. Ambroziak AM, Szaflik J, Szaflik JP, Ambroziak M, Witkiewicz J, Skopiński P. Immunomodulation on the ocular surface: a review. Central-European Journal of Immunology. Termedia Publishing; 2016;41(2):195. PMID: 27536206

48. Mahmood AMA, Al-Swailem SA. Essential fatty acids in the treatment of dry eye syndrome: A myth or reality? Saudi Journal of Ophthalmology. Wolters Kluwer -- Medknow Publications; 2014 Jul;28(3):195. PMID: 25278796

49. Therapeutic Contact Lenses in the Treatment of Corneal and... : The Asia-Pacific Journal of Ophthalmology [Internet]. LWW. [cited 2022 Oct 6]. Available from: https://journals.lww.com/apjoo/Fulltext/2020/12000/Therapeutic_Contact_Lenses_in_the_Treatment_of.7.aspx

50. Pan Q, Angelina A, Marrone M, Stark WJ, Akpek EK. Autologous serum eye drops for dry eye. The Cochrane Database of Systematic Reviews [Internet]. John Wiley and Sons, Inc. and the Cochrane Library; 2017 Feb [cited 2022 Sep 20];2017(2). Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5510593/ PMID: 28245347

51. Diabetes [Internet]. [cited 2022 Oct 6]. Available from: https://www.who.int/news-room/fact-sheets/detail/diabetes

52. Classification of diabetes mellitus [Internet]. [cited 2022 Oct 6]. Available from: https://www.who.int/publications-detail-redirect/classification-of-diabetes-mellitus

53. F F, N P. [Etiopathogenesis and pathophysiology of type 2 diabetes]. Revue medicale de Liege [Internet]. Rev Med Liege; 2005 Jun [cited 2022 Oct 6];60(5–6). Available from: https://pubmed.ncbi.nlm.nih.gov/16035295/ PMID: 16035295

54. Arroba AI, Aguilar-Diosdado M. Special Issue “The Prevention, Treatment, and Complications of Diabetes Mellitus” Journal of Clinical Medicine. Multidisciplinary Digital Publishing Institute; 2022 Jan;11(18):5305.

55. Diabetic retinopathy, an overview. Vision Research. Pergamon; 2017 Oct 1;139:1–6.

56. Diabetic Retinopathy Pathophysiology - EyeWiki [Internet]. [cited 2022 Oct 6]. Available from: https://eyewiki.aao.org/Diabetic_Retinopathy_Pathophysiology

57. Ophthalmology E-Book [Internet]. [cited 2022 Oct 6]. Available from: https://books.google.com/books/about/Ophthalmology_E_Book.html?id=34toDwAAQBAJ

58. Juster-Switlyk K, Smith AG. Updates in diabetic peripheral neuropathy. F1000Research [Internet]. Faculty of 1000 Ltd; 2016 [cited 2022 Oct 3];5. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4847561/ PMID: 27158461

59. Bikbova G, Oshitari T, Baba T, Bikbov M, Yamamoto S. Diabetic corneal neuropathy: clinical perspectives. Clinical Ophthalmology (Auckland, NZ). Dove Press; 2018;12:981. PMID: 29872257

60. Tk Y, E O. Diabetes mellitus is associated with dry eye syndrome: a meta-analysis. International ophthalmology [Internet]. Int Ophthalmol; 2019 Nov [cited 2022 Sep 20];39(11). Available from: https://pubmed.ncbi.nlm.nih.gov/31065905/ PMID: 31065905

61. Klein R, Klein BEK, Knudtson MD, Wong TY, Tsai MY. Are Inflammatory Factors Related to Retinal Vessel Caliber?: The Beaver Dam Eye Study. Arch Ophthalmol. American Medical Association; 2006 Jan 1;124(1):87–94.

62. Hindawi. Dry Eye Syndrome in Patients with Diabetes Mellitus: Prevalence, Etiology, and Clinical Characteristics [Internet]. [cited 2022 Sep 20]. Available from: https://www.hindawi.com/journals/joph/2016/8201053/

63. H N, Tg M, A M, A A. Association of Dry Eye Disease with Diabetic Retinopathy. Journal of the College of Physicians and Surgeons--Pakistan : JCPSP [Internet]. J Coll Physicians Surg Pak; 2020 May [cited 2022 Sep 20];30(5). Available from: https://pubmed.ncbi.nlm.nih.gov/32580845/ PMID: 32580845

64. Khurana G, Khurana D, Jain R. Dry Eye in Patients With Diabetic Retinopathy: A Clinical Study. The Official Scientific Journal of Delhi Ophthalmological Society. 2016 Dec 24;27(3):190–193.

65. Y L, X Z, F L, S Z. The effect of the duration of diabetes on dry eye and corneal nerves. Contact lens & anterior eye : the journal of the British Contact Lens Association [Internet]. Cont Lens Anterior Eye; 2019 Aug [cited 2022 Sep

20];42(4). Available from: https://pubmed.ncbi.nlm.nih.gov/31029535/ PMID: 31029535

66. Kc S, Ks L, L T. A systematic review on the impact of diabetes mellitus on the ocular surface. Nutrition & diabetes [Internet]. Nutr Diabetes; 2017 Mar 20 [cited 2022 Sep 20];7(3). Available from: https://pubmed.ncbi.nlm.nih.gov/28319106/ PMID: 28319106

67. Reddy OR, Asritha B, Sushma K. Study of dry eye in diabetes mellitus type II and it's association with diabetic retinopathy. Indian Journal of Clinical and Experimental Ophthalmology. 2020 Dec 15;6(4):626–628.

68. (PDF) Comparative Evaluation of Incidence of Dry Eye in Patients with and Without Diabetes Mellitus [Internet]. ResearchGate. [cited 2022 Oct 8]. Available from: https://www.researchgate.net/publication/340983808_Comparative_Evaluation_of_Incidence_of_Dry_Eye_in_Patients_with_and_Without_Diabetes_Mellitus

ANNEXURE I: ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed to-be-University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 02 **Date: 25/01/2021**

To.
REG. NO: BK0120002
PG student in Ophthalmology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A ONE YEAR PROSPECTIVE STUDY TO COMPARE PREVALENCE OF DRY EYE DISEASE AND CORNEAL SENSITIVITY BETWEEN DIABETICS AND NON-DIABETICS AT KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

151

ANNEXURE II:

INFORMED CONSENT

Title of Research Study:

A ONE YEAR PROSPECTVE STUDY TO COMPARE PREVALENCE OF DRY EYE DISEASE AND ASSESSMENT OF CORNEAL NERVE SENSITIVITY BETWEEN DIABETICS AND NON-DIABETICS AT KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI.

Principal Investigator:

DR. REG. NO: BK0120002

POST GRADUATE STUDENT,

DEPARTMENT OF OPHTHALMOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

K.L.E. ACADEMY OF HIGHER EDUCATION, BELAGAVI – 590010

Guide:

DR. _____,

PROFESSOR AND HEAD,

DEPARTMENT OF OPHTHALMOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

K.L.E. ACADEMY OF HIGHER EDUCATION, BELAGAVI - 590010

Introduction and Purpose

The purpose of the study is to compare the prevalence of dry eye disease and corneal nerve sensitivity in diabetics and non-diabetics. It also aims at comparing the severity of dry eye symptoms in those suffering from diabetic retinopathy as opposed to those without retinopathy.

Procedure

If, you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations.

Risk: NIL

Benefits:

This study will help by giving us a better understanding of the effect of diabetes on the tear film dynamics.

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study. If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality

All the information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy

Does not apply to this research

Financial incentives for participation

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KAHER, Belgaum as part of requirement towards the completion of MS degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

1. **REG. NO: BK0120002**
   Principal investigator,
   PG in Ophthalmology,
   J.N.M.C., Belagavi

2. Dr. _____,
   Professor and Head,
   Dept. of Ophthalmology
   J.N.M.C., Belagavi

3. Dr. Harsha Hegde,
   Chairperson,
   J.N.M.C., IEC & Scientist D,
   ICMR, National Institute of Traditional Medicine,
   Belagavi – 10

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, and I have had all the questions answered. The study and the consent form has been explained to me in my language.

Participant's Name:

…….

Name of the Legally Authorized Representative / Guardian:

…….

Signature / Left Thumb print of the Participant

or Legally Authorized Representative

Witness' Name: ……

Investigator's Name and Signature: ……

Signature / Left thumb Impression

Signature / Left thumb Impression

DATE:

PLACE:

ANNEXURE III:

PROFORMA

Hospital ID: Study ID:

NAME: _____

AGE:_____Years

GENDER: MALE ☐ FEMALE ☐

ADDRESS: _____

_____

OCCUPATION: _____

CONTACT NUMBER: ☐☐☐☐☐☐☐☐☐☐

DATE OF EXAMINATION: / /

IS THE PATIENT ELIGIBLE FOR STUDY? YES ☐ NO ☐

HAS INFORMED CONSENT BEEN GIVEN? YES ☐ NO ☐

CHIEF COMPLAINTS:

1. DIMINUTION OF VISION: YES ☐ NO ☐
   RE: DURATION _____ Days/Months/Years
   LE: DURATION _____ Days/Months/Years

GRADUAL ☐ PROGRESSIVE ☐ SUDDEN ☐ NON-PROGRESSIVE ☐

2. SPEED QUESTIONNAIRE:

	At this visit		Within past 72 hours		Within past 3 months	
Symptoms	**Yes**	**No**	**Yes**	**No**	**Yes**	**No**
Dryness, Grittiness or Scratchiness						
Soreness or Irritation						
Burning or Watering						
Eye Fatigue						

FREQUENCY of your symptoms:

	0	1	2	3
Dryness, Grittiness or Scratchiness				
Soreness or Irritation				
Burning or Watering				
Eye Fatigue				

Response Options: 0 = Never; **1** = Sometimes; **2** = Often; **3** = Constant

SEVERITY of your symptoms:

	0	1	2	3	4
Dryness, Grittiness or Scratchiness					
Soreness or Irritation					
Burning or Watering					
Eye Fatigue					

Response Options: 0 = No Problems; **1** = Tolerable - not perfect, but not uncomfortable; **2** = Uncomfortable - irritating, but does not interfere with my day; **3** = Bothersome - irritating and interferes with my day; **4** = Intolerable - unable to perform my daily tasks.

3. H/O DRY MOUTH: YES ☐ NO ☐

PAST HISTORY:

1. DIABETES: PRESENT ☐ ABSENT ☐

   DURATION OF DIABETES: _____ Months/Years

   ANY MEDICATION: YES ☐ NO ☐ IF YES, DRUGS TAKEN
   _____

2. HYPERTENSION: PRESENT ☐ ABSENT ☐
   ANY MEDICATION:
   _____

3. OCULAR TRAUMA: PRESENT ☐ ABSENT ☐

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

5. H/O CONTACT LENS USE: YES ☐ NO ☐
   IF YES, DURATION: _____

6. H/O JOINT PAIN: YES ☐ NO ☐
   IF YES, DURATION: _____

7. DRUG HISTORY:
   _____

OCULAR EXAMINATION:

a. **ANTERIOR SEGMENT:** RIGHT EYE LEFT EYE

VISUAL ACUITY

PINHOLE

WITH GLASSES

EXTRA OCULAR MOVEMENTS

EYELIDS

EYELASHES

CONJUNCTIVA

SCLERA

CORNEA

ANTERIOR CHAMBER

IRIS

PUPIL

LENS

b. **POSTERIOR SEGMENT:**

GLOW

MEDIA

DISC

C:D

B/V

B/G

MACULA

c. SCHIRMER'S II TEST:

d. TBUT (sec):

e. CORNEAL NERVE SENSITIVITY:

f. NASOLACRIMAL DUCT PATENCY:

IMPRESSION:

DIABETIC / NON-DIABETIC:

	RIGHT EYE	**LEFT EYE**
DIABETIC RETINOPATHY CHANGES		
DRY EYE SEVERITY		
CORNEAL NERVE SENSITIVITY		

NAME OF THE INVESTIGATOR : **REG. NO: BK0120002**

SIGNATURE : _____

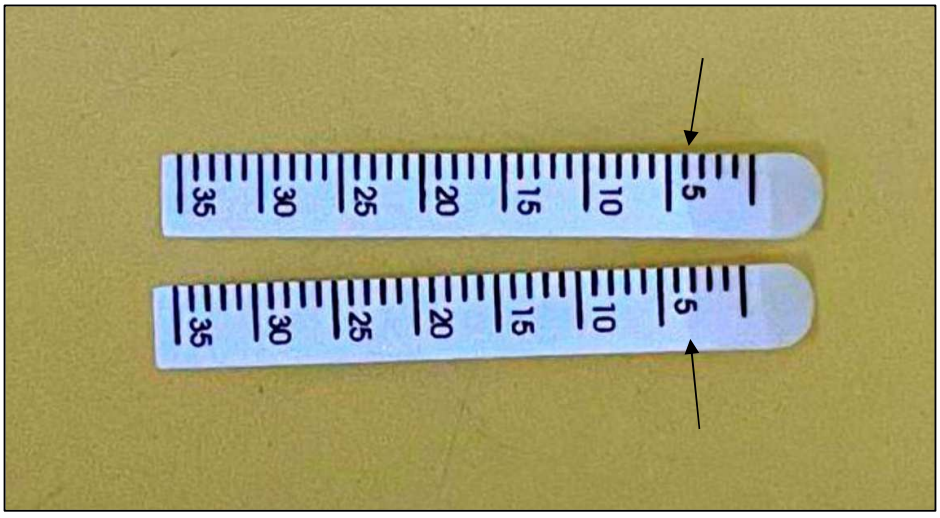
NAME OF THE GUIDE : **DR. _____**

SIGNATURE : _____

ANNEXURE III: PHOTOGRAPHS

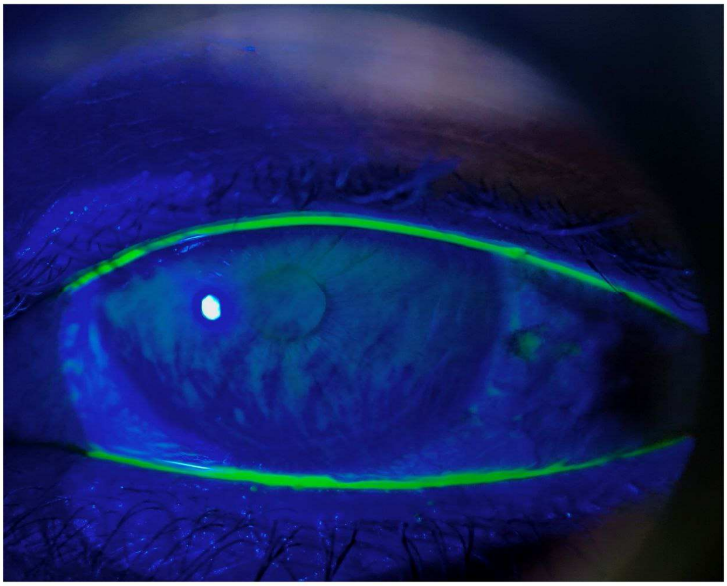
Patient undergoing Schirmer's II test

Schirmer's II test



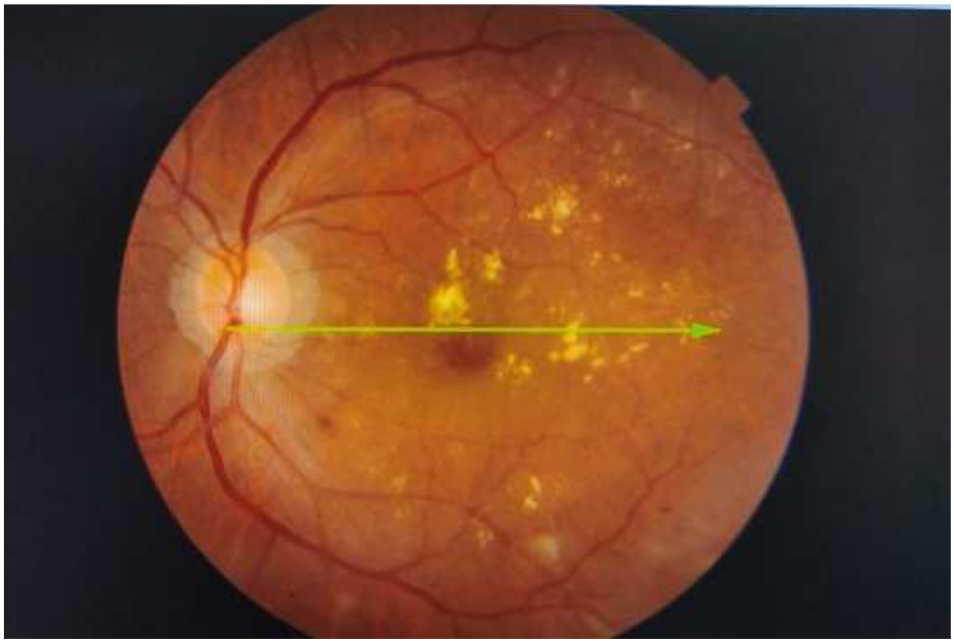
Schirmer's II Test showing Severe DED in Both eyes.

TBUT test under cobalt blue filter

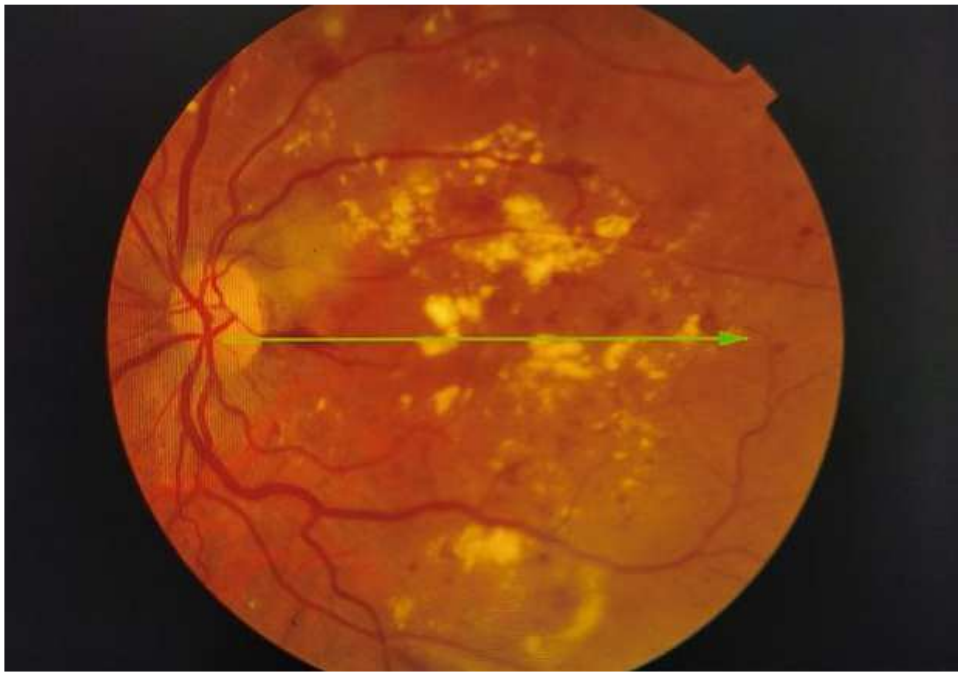


TBUT seen under cobalt blue filter.

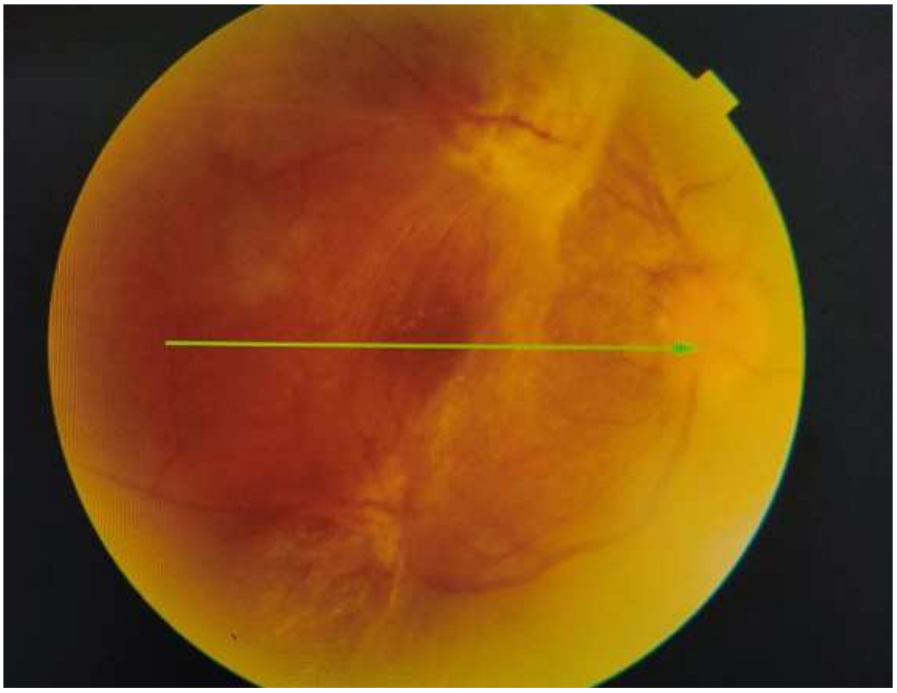
Assessment of corneal nerve sensitivity using cotton wisp



Moderate NPDR



Severe NPDR



PDR

CSME

ANNEXURE IV: KEY TO MASTER CHART

Male	=	M
Female	=	F
Yes	=	Y
No	=	N
Nuclear Sclerosis	=	NS
Posterior Subcapsular Cataract	=	PSC
Within Normal Limits	=	WNL
H'ages	=	Hemorrhages
Non Proliferative Diabetic Retinopathy	=	NPDR
Proliferative Diabetic Retinopathy	=	PDR

SPEED score –

Frequency of symptoms: 0 = Never,

1 = Sometimes,

2 = Often,

3 = Constant

Severity of symptoms: 0 = No Problem,

1 = Tolerable - not perfect, but not uncomfortable;

2 = Uncomfortable - irritating, but does not interfere with my day;

3 = Bothersome - irritating and interferes with my day;

4 = Intolerable - unable to perform my daily tasks

STUDY ID	AGE	GENDER	OCCUPATION	DIMINUTION OF VISION	DURATION	H/O DIABETES MELLITUS	DURATION		BCVA		ANTERIOR SEGMENT		FUNDUS EXAMINATION		ETDRS GRADE OF DIABETIC RETINOPATHY	SYMPTOMS				FREQUENCY	SEVERITY	SPEED SCORE	SCHIRMERS II TEST (mm)		TBUT (sec)		CORNEAL NERVE SENSITIVITY	
								TREATMENT Y/N	OD	OS	OD	OS	OD	OS		GRITTINESS	BURNING	IRRITATION	EYE FATIGUE				OD	OS	OD	OS	OD	OS
ND1	51	F	Housewife	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\36	6\36(p)	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	22	27	18	20	NORMAL	NORMAL
ND2	58	M	Employee	NO	-	N	-	N	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	Y	Y	1	2	6	22	25	18	19	NORMAL	NORMAL
ND3	60	M	Retired	NO	-	N	-	N	6\9	6\9(p)	PSEUDOPHAKIA	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	16	16	12	14	NORMAL	NORMAL
ND4	38	M	Computer engineer	NO	-	N	-	N	6\6	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	Y	1	1	2	26	22	18	20	NORMAL	NORMAL
ND5	35	M	Employee	NO	-	N	-	N	6\6	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	27	30	19	20	NORMAL	NORMAL
ND6	32	M	Worker	NO	-	N	-	N	6\6	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	Y	1	1	2	16	22	16	20	NORMAL	NORMAL
ND7	37	M	Police	NO	-	N	-	N	6\6	6\6	PSC	PSC	WNL	WNL	-	N	Y	N	N	1	1	2	30	26	19	19	NORMAL	NORMAL
ND8	58	M	Employee	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\9	6\6(p)	GRADE 2 NS	GRADE 2 NS	GRADE 1 HTN RETINOPATHY	GRADE 1 HTN RETINOPATHY	-	N	N	N	N	0	0	0	12	16	15	15	NORMAL	NORMAL
ND9	60	M	Retired	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\18	6\18	GRADE 2 NS + PSC	GRADE 3 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	Y	Y	N	1	1	4	7	8	9	9	NORMAL	NORMAL
ND10	60	F	Housewife	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	CF 1M	CF 3M	GRADE 3 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	Y	1	1	2	21	19	15	16	NORMAL	NORMAL
ND11	42	F	Employee	NO	-	N	-	N	6\6	6\6	GRADE 1 NS	GRADE 1 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	Y	N	1	1	2	19	22	20	21	NORMAL	NORMAL
ND12	33	F	Nurse	NO	-	N	-	N	6\9	6\9	MILD CC	MILD CC	WNL	WNL	-	N	N	Y	Y	1	1	4	7	7	8	8	NORMAL	NORMAL
ND13	30	M	Employee	GRADUAL, PROGRESSIVE	15 DAYS	N	-	N	6\9(p)	6\9(p)	PSC	PSC	Old Toxoplamsa scar	WNL	-	N	N	N	N	0	0	0	16	16	15	15	NORMAL	NORMAL
ND14	46	M	Bank Employee	NO	-	N	-	N	6\6	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	Y	1	1	2	17	17	21	20	NORMAL	NORMAL
ND15	72	M	Retired	GRADUAL, PROGRESSIVE	8 MONTHS	N	-	N	6\9	6\9	PSEUDOPHAKIA	PSEUDOPHAKIA	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	Y	1	1	2	18	16	11	12	NORMAL	NORMAL
ND16	49	M	Police	NO	-	N	-	N	6\6	6\6	WNL	WNL	WNL	WNL	-	N	N	Y	Y	1	1	4	12	10	16	14	NORMAL	NORMAL
ND17	56	F	Housewife	GRADUAL, PROGRESSIVE	1.5 YEARS	N	-	N	6\6	6\24	PSEUDOPHAKIA	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	15	18	20	18	NORMAL	NORMAL
ND18	60	F	Housewife	GRADUAL, PROGRESSIVE	3 YEARS	N	-	N	6\36	CF 3M	GRADE 3 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	19	24	18	20	NORMAL	NORMAL
ND19	83	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	6\36	GRADE 3 NS + PSC	GRADE 2 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	25	22	16	17	NORMAL	NORMAL
ND20	60	M	Farmer	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\18(p)	6\12(p)	GRADE 2 NS	GRADE 1 NS	WNL	WNL	-	N	N	Y	N	1	1	2	27	25	19	19	NORMAL	NORMAL
ND21	67	F	Housewife	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\18	6\18	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	Y	Y	1	2	6	9	10	12	14	NORMAL	NORMAL
ND22	78	M	Farmer	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\18	CF 2M	GRADE 1 NS + CC	GRADE 3 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	Y	1	2	3	15	15	10	15	NORMAL	NORMAL
ND23	41	M	Employee	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	6\6	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	20	21	14	14	NORMAL	NORMAL
ND24	35	F	Employee	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\6	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	17	19	16	16	NORMAL	NORMAL
ND25	38	F	Employee	NO	-	N	-	N	6\6(p)	6\6(p)	CLEAR	CLEAR	WNL	WNL	-	N	N	N	Y	1	1	2	20	21	18	19	NORMAL	NORMAL
ND26	32	F	Employee	NO	-	N	-	N	6\6(p)	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	Y	1	1	2	27	35	18	20	NORMAL	NORMAL
ND27	59	M	Farmer	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\12(p)	6\9	GRADE 1 NS	GRADE 1 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	22	25	18	16	NORMAL	NORMAL
ND28	66	F	Housewife	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	CFCF	6\18	GRADE 2 NS + PSC	GRADE 1 NS	WNL	WNL	-	N	Y	N	Y	1	1	4	12	15	15	14	NORMAL	NORMAL
ND29	72	M	Retired	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	Y	2	2	4	3	3	9	9	NORMAL	NORMAL
ND30	65	M	Farmer	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\60	6\9	GRADE 2 NS + PSC	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	16	18	18	17	NORMAL	NORMAL

ND31	58	F	Housewife	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\9	CF 3M	PSEUDOPHAKIA	GRADE 3 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	16	22	20	20	NORMAL	NORMAL
ND32	75	M	Farmer	GRADUAL, PROGRESSIVE	3 MONTHS	N	-	N	6\24(p)	6\36(p)	GRADE 2 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	18	19	15	15	NORMAL	NORMAL
ND33	37	M	Employee	NO	-	N	-	N	6\6(p)	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	Y	N	1	2	3	6	6	8	10	NORMAL	NORMAL
ND34	45	F	Bank Employee	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\36	6\36	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	5	6	9	9	NORMAL	NORMAL
ND35	50	F	Housewife	NO	-	N	-	N	6\60	6\12(p)	GRADE 2 NS + PSC	GRADE 1 NS	WNL	WNL	-	N	N	N	Y	2	2	4	8	8	10	10	NORMAL	NORMAL
ND36	85	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\60	6\24	GRADE 2 NS + PSC	PSEUDOPHAKIA	GLAUCOMATOUS	GLAUCOMATOUS	-	N	N	Y	N	1	1	2	12	16	14	14	NORMAL	NORMAL
ND37	67	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 3M	CF 3M	GRADE 3 NS	GRADE 3 NS	WNL	WNL	-	N	Y	N	N	1	1	2	19	18	16	16	NORMAL	NORMAL
ND38	55	F	Housewife	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	6\12	CF 3M	GRADE 1 NS	GRADE 3 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	14	16	15	15	NORMAL	NORMAL
ND39	67	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1.5 M	CF 1.5 M	GRADE 2 NS + PSC	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	30	32	20	20	NORMAL	NORMAL
ND40	73	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	6\6	GRADE 2 NS + PSC	PSEUDOPHAKIA	WNL	HTN RETINOPATHY	-	N	N	N	N	0	0	0	29	32	16	16	NORMAL	NORMAL
ND41	46	F	Tailor	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\6	6\12	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	Y	1	1	2	13	15	15	15	NORMAL	NORMAL
ND42	70	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 3M	CF 3M	GRAE 3 NS	GRADE 3 NS + PSC	WNL	WNL	-	N	N	Y	N	1	1	2	12	12	15	14	NORMAL	NORMAL
ND43	60	F	Housewife	GRADUAL, PROGRESSIVE	3 MONTHS	N	-	N	CF 1M	6\9	GRADE 2 NS + CC	PSEUDOPHAKIA	SYNCHISIS SCINTILLANS	SYNCHYSIS SCINTILLANS	-	N	N	N	Y	1	1	2	8	8	10	10	NORMAL	NORMAL
ND44	35	F	Employee	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	Y	Y	Y	Y	2	2	16	3	3	4	4	NORMAL	NORMAL
ND45	64	M	Retired	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\18	6\12	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	23	25	16	16	NORMAL	NORMAL
ND46	56	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	CFCF	GRADE 2 NS + PSC	GRADE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	10	10	12	12	NORMAL	NORMAL
ND47	73	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\9	CF 3M	PSEUDOPHAKIA	GRADE 3 NS + PSC	GLAUCOMATOUS	GLAUCOMATOUS	-	N	N	N	N	0	0	0	23	25	19	19	NORMAL	NORMAL
ND48	60	M	Farmer	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\36	6\36	GRADE 1 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	15	20	14	17	NORMAL	NORMAL
ND49	65	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	6\6	GRADE 3 NS + PSC	PSEUDOPHAKIA	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	33	32	18	18	NORMAL	NORMAL
ND50	63	M	Farmer	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	CF 2M	GRADE 2 NS	GRADE 3 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	Y	1	1	2	7	9	10	10	NORMAL	NORMAL
ND51	80	M	Retired	GRADUAL, PROGRESSIVE	2 YEARS	N	-	N	CF 3M	CF 3M	GRADE 3 NS + PSC	GRADE 3 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	18	22	20	20	NORMAL	NORMAL
ND52	65	F	Housewife	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\18	6\24	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	Y	N	1	1	2	9	9	10	10	NORMAL	NORMAL
ND53	43	F	Housewife	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	CF 3M	CF 2M	GRADE 3 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	Y	N	1	1	2	12	15	13	13	NORMAL	NORMAL
ND54	67	F	Housewife	GRADUAL, PROGRESSIVE	4 MONTHS	N	-	N	6\6	CF 3M	PSEUDOPHAKIA	GRADE 3 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	16	20	25	25	NORMAL	NORMAL
ND55	34	M	Employee	NO	-	N	-	N	6\6(p)	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	N	Y	1	2	3	6	8	10	11	NORMAL	NORMAL
ND56	57	M	Employee	NO	-	N	-	N	6\6(p)	6\6(p)	MILD PSC	PSC	WNL	WNL	-	N	N	Y	Y	1	2	6	6	7	10	10	NORMAL	NORMAL
ND57	53	F	Housewife	NO	-	N	-	N	6\6(p)	6\9	PSC	PSC	WNL	WNL	-	Y	Y	Y	Y	2	3	20	3	3	3	4	REDUCED	REDUCED
ND58	38	F	Housewife	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	Y	Y	2	2	8	8	11	7	7	NORMAL	NORMAL
ND59	32	M	Bank Employee	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	N	Y	Y	N	2	3	10	10	16	8	12	NORMAL	NORMAL
ND60	55	F	Teacher	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	6\12	6\36	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	Y	2	2	4	16	15	15	15	NORMAL	NORMAL
ND61	46	M	Technician	GRADUAL, PROGRESSIVE	3 MONTHS	N	-	N	6\9	CF 1.5 M	PSEUDOPHAKIA	GRADE 2 NS + CC	WNL	WNL	-	N	N	N	N	0	0	0	22	21	18	16	NORMAL	NORMAL
ND62	74	M	Farmer	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	6\60	CF 1M	GRADE 2 NS + CC	GRADE 2 NS + PSC	WNL	WNL	-	N	Y	Y	N	2	2	8	22	27	18	18	NORMAL	NORMAL
ND63	40	F	Teacher	NO	-	N	-	N	6\6(p)	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	Y	1	1	2	6	6	9	10	NORMAL	NORMAL
ND64	64	M	Farmer	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\36	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	25	20	14	15	NORMAL	NORMAL
ND65	40	F	Housewife	GRADUAL, PROGRESSIVE	5 MONTHS	N	-	N	6\24	6\24	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	Y	1	1	2	26	26	20	21	NORMAL	NORMAL
ND66	68	F	Housewife	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	CF 1M	6\9	GRADE 3 NS	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	Y	1	1	2	15	17	18	21	NORMAL	NORMAL

ND67	53	F	Housewife	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	CF 2M	6\18	GRADE 2 NS + PSC	GRADE 1 NS + CC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	10	13	22	25	NORMAL	NORMAL
ND68	36	F	Employee	NO	-	N	-	N	6\6	6\6(p)	CLEAR	CLEAR	WNL	WNL	-	Y	Y	Y	N	2	2	12	4	4	4	6	REDUCED	REDUCED
ND69	56	F	Housewife	GRADUAL, PROGRESSIVE	3 MONTHS	N	-	N	CF 3M	6\9	GRADE 2 NS + CC	PSEUDOPHAKIA	HTN RETINOPATHY	HTN RETINOPATHY	-	Y	Y	Y	Y	2	2	16	2	4	5	7	REDUCED	REDUCED
ND70	60	F	Housewife	NO	-	N	-	N	6/6(p)	6\9	Pseudophakia +Epithelial defect	Pseudophakia +Epithelial defect	WNL	WNL	-	Y	Y	Y	Y	2	2	16	3	3	2	4	REDUCED	REDUCED
ND71	57	M	Employee	NO	-	N	-	N	6\6(P)	6\6(P)	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	Y	N	2	2	4	8	8	13	11	NORMAL	NORMAL
ND72	34	M	Teacher	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	16	19	16	14	NORMAL	NORMAL
ND73	67	F	Housewife	GRADUAL, PROGRESSIVE	4 MONTHS	N	-	N	6\9	CF 3M	PSEUDOPHAKIA	GARDE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	16	20	25	25	NORMAL	NORMAL
ND74	43	F	Teacher	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	CF 3M	CF 3M	GRADE 3 NS	GRADE 3 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	12	15	13	13	NORMAL	NORMAL
ND75	65	F	Housewife	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\24	6\18	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	10	9	10	10	NORMAL	NORMAL
ND76	80	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 3M	CF 2M	GRADE 3 NS	GRADE 2 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	18	22	20	20	NORMAL	NORMAL
ND77	63	M	Farmer	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	CF 2M	GRADE 2 NS + CC	GRADE 2 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	7	9	10	10	NORMAL	NORMAL
ND78	37	F	Tailor	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	18	17	19	23	NORMAL	NORMAL
ND79	37	M	Employee	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	16	14	19	19	NORMAL	NORMAL
ND80	75	M	Farmer	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\24(p)	6\36(p)	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	16	18	16	16	NORMAL	NORMAL
ND81	59	F	Housewife	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\6	6\9	PSC	GRADE 1 NS +PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	16	22	20	20	NORMAL	NORMAL
ND82	65	M	Farmer	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\60	6\9	GRADE 2 NS + PSC	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	18	23	18	17	NORMAL	NORMAL
ND83	72	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\12	6\12	PSEUDOPHAKIA	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	13	15	16	16	NORMAL	NORMAL
ND84	66	F	Housewife	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	CFCF	6\18	GRADE 3 NS	GRADE 2 NS + CC	WNL	WNL	-	N	N	N	Y	1	1	2	12	15	15	15	NORMAL	NORMAL
ND85	59	M	Farmer	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\9(p)	6\6	GRADE 1 NS + CC	GRADE 1 NS + CC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	22	25	18	16	NORMAL	NORMAL
ND86	34	F	Software engineer	NO	-	N	-	N	6\6	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	27	34	21	22	NORMAL	NORMAL
ND87	38	F	Housewife	NO	-	N	-	N	6\6(p)	6\6(p)	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	20	21	18	19	NORMAL	NORMAL
ND88	40	F	Teacher	NO	-	N	-	N	6\6	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	17	19	16	16	NORMAL	NORMAL
ND89	41	M	Employee	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	19	18	14	14	NORMAL	NORMAL
ND90	78	M	Retired	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\9(p)	CF 2M	PSEUDOPHAKIA	GRADE 3 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	Y	1	1	2	15	15	10	13	NORMAL	NORMAL
ND91	67	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\18	6\18	GRADE 2 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	10	10	12	14	NORMAL	NORMAL
ND92	60	M	Farmer	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\18	6\12	GRADE 2 NS + PSC	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	27	25	19	19	NORMAL	NORMAL
ND93	83	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CFCF	6\9	GRADE 3 NS	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	25	22	17	17	NORMAL	NORMAL
ND94	60	F	Housewife	GRADUAL, PROGRESSIVE	3 YEARS	N	-	N	6\36	HMCF	GRADE 2 NS	GRADE 3 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	19	24	18	20	NORMAL	NORMAL
ND95	55	F	Housewife	GRADUAL, PROGRESSIVE	1.5 YEARS	N	-	N	6\6	6\24	PSEUDOPHAKIA	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	18	20	18	15	NORMAL	NORMAL
ND96	49	M	Police	NO	-	N	-	N	6\6(p)	6\6(p)	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	12	10	16	16	NORMAL	NORMAL
ND97	72	M	Retired	GRADUAL, PROGRESSIVE	8 MONTHS	N	-	N	6\9	6\9	PSEUDOPHAKIA	PSEUDOPHAKIA	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	18	16	11	12	NORMAL	NORMAL
ND98	33	F	Teacher	NO	-	N	-	N	6\9	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	Y	N	1	1	2	7	7	8	8	NORMAL	NORMAL
ND99	46	M	Bank Employee	NO	-	N	-	N	6\6	6\6	GRADE 1 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	17	17	21	20	NORMAL	NORMAL
ND100	51	F	Housewife	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\36	6\36	GRADE 2 NS + CC	GRADE 2 NS + CC	WNL	WNL	-	N	N	N	N	0	0	0	22	27	18	20	NORMAL	NORMAL
ND101	58	M	Employee	NO	-	N	-	N	6\9	6\9	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	Y	Y	1	1	2	22	25	18	19	NORMAL	NORMAL
ND102	60	M	Retired	NO	-	N	-	N	6\12	6\9	PSEUDOPHAKIA	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	16	16	12	14	NORMAL	NORMAL

ND103	38	M	Employee	NO	-	N	-	N	6\9	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	26	22	18	18	NORMAL	NORMAL
ND104	35	M	Employee	NO	-	N	-	N	6\6	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	27	30	19	20	NORMAL	NORMAL
ND105	32	M	Employee	NO	-	N	-	N	6\9	6\6	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	16	22	16	20	NORMAL	NORMAL
ND106	37	M	Employee	NO	-	N	-	N	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	30	26	19	19	NORMAL	NORMAL
ND107	58	M	Employee	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\12	6\12(p)	MILD PSC	PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	12	16	15	15	NORMAL	NORMAL
ND108	60	M	Retired	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\36	6\60	GRADE 2 NS	GRADE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	22	26	17	17	NORMAL	NORMAL
ND109	55	F	Cook	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	6\24	6\12	GRADE 2 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	17	18	22	23	NORMAL	NORMAL
ND110	45	M	Hawker	NO	-	N	-	N	6\6	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	20	22	22	17	NORMAL	NORMAL
ND111	41	M	Driver	NO	-	N	-	N	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	33	34	26	24	NORMAL	NORMAL
ND112	56	F	Housewife	GRADUAL, PROGRESSIVE	4 MONTHS	N	-	N	6\12	6\18	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	Y	1	1	2	15	17	21	21	NORMAL	NORMAL
ND113	63	F	Housewife	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\9	6\60	PSEUDOPHAKIA	GRADE 2 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	23	25	25	21	NORMAL	NORMAL
ND114	65	M	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\36	6\36	GRADE 2 NS	GRADE 1 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	26	25	22	23	NORMAL	NORMAL
ND115	60	F	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\60	CF 1M	GARDE 2 NS + PSC	GRADE 3 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	25	28	20	22	NORMAL	NORMAL
ND116	38	M	Employee	NO	-	N	-	N	6\9	6\12	CLEAR	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	34	30	18	19	NORMAL	NORMAL
ND117	48	M	Employee	NO	-	N	-	N	6\18	6\12	PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	10	12	16	16	NORMAL	NORMAL
ND118	53	M	Employee	NO	-	N	-	N	6\24	6\9	GRADE 1 NS + CC	GRADE 1 NS	WNL	WNL	-	N	N	N	Y	1	1	2	10	9	10	10	NORMAL	NORMAL
ND119	76	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 3M	CF 3M	GRADE 3 NS	GRADE 2 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	Y	1	1	2	14	17	14	15	NORMAL	NORMAL
ND120	65	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 2M	6\12	GRADE 3 NS	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	18	18	13	16	NORMAL	NORMAL
ND121	43	M	Bank Employee	NO	-	N	-	N	6\9	6\12	MILD PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	16	19	15	14	NORMAL	NORMAL
ND122	66	M	Retired	GRADUAL, PROGRESSIVE	8 MONTHS	N	-	N	CF 1M	6\60	GRADE 2 NS + PSC	GRADE 2 NS	WNL	WNL	-	N	N	Y	Y	1	2	6	8	9	8	9	NORMAL	NORMAL
ND123	67	M	Driver	GRADUAL, PROGRESSIVE	3 MONTHS	N	-	N	CF 1M	CF 1M	GARDE 3 NS	GRADE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	13	17	16	16	NORMAL	NORMAL
ND124	56	M	Hawker	NO	-	N	-	N	6\12	6\12	GARDE 2 NS + PSC	GARDE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	16	16	19	21	NORMAL	NORMAL
ND125	55	F	Teacher	NO	-	N	-	N	6\36	6\18	GRADE 1 NS + CC	GARDE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	18	23	26	27	NORMAL	NORMAL
ND126	42	F	Housewife	NO	-	N	-	N	6\12	6\9	GARDE 2 NS + PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	25	29	22	23	NORMAL	NORMAL
ND127	68	F	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	6\12	6\24	GRADE 2 NS	GRADE 2 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	29	26	21	23	NORMAL	NORMAL
ND128	34	M	Employee	NO	-	N	-	N	6\6	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	31	29	22	21	NORMAL	NORMAL
ND129	56	M	Employee	NO	-	N	-	N	6\24	6\18	GRADE 2 NS	GARDE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	24	25	17	17	NORMAL	NORMAL
ND130	64	M	Retired	GRADUAL, PROGRESSIVE	5 MONTHS	N	-	N	6\36	6\60	GRADE 1 NS + CC	GRADE 2 NS + PSC	WNL	WNL	-	N	N	Y	Y	1	1	4	11	9	9	9	NORMAL	NORMAL
ND131	44	F	Housewife	NO	-	N	-	N	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	19	18	15	15	NORMAL	NORMAL
ND132	52	F	Housewife	NO	-	N	-	N	6\12	6\18	PSC	GRADE 1 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	21	22	24	25	NORMAL	NORMAL
ND133	38	F	Farmer	NO	-	N	-	N	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	19	15	16	16	NORMAL	NORMAL
ND134	42	F	Housewife	NO	-	N	-	N	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	23	23	27	25	NORMAL	NORMAL
ND135	56	F	Housewife	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\24	6\12	GARDE 2 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	Y	1	1	2	17	18	19	16	NORMAL	NORMAL
ND136	47	F	Housewife	NO	-	N	-	N	6\24	6\36	GRADE 1 NS + PSC	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	12	15	17	17	NORMAL	NORMAL
ND137	62	M	Housewife	GRADUAL, PROGRESSIVE	3 MONTHS	N	-	N	CF 2M	CF 3M	GRADE 3 NS	GRADE 3 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	21	25	16	16	NORMAL	NORMAL
ND138	47	M	Employee	NO	-	N	-	N	6\18	6\12	GARDE 1 NS	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	24	26	22	21	NORMAL	NORMAL

ND139	39	F	Employee	NO	-	N	-	N	6\12	6\18	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	13	14	16	16	NORMAL	NORMAL
ND140	43	F	Employee	NO	-	N	-	N	6\9	6\12	MILD PSC	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	19	17	19	21	NORMAL	NORMAL
ND141	76	M	Retired	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	CF 2M	GRADE 3 NS	GRADE 3 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	24	26	17	17	NORMAL	NORMAL
ND142	47	F	Employee	NO	-	N	-	N	6\9	6\12	GARDE 1 NS + PSC	GARDE 2 NS	WNL	WNL	-	N	N	N	Y	2	2	4	10	9	10	11	NORMAL	NORMAL
ND143	55	F	Employee	NO	-	N	-	N	6\9	6\9	GARDE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	18	20	22	22	NORMAL	NORMAL
ND144	38	M	Employee	NO	-	N	-	N	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	20	21	18	18	NORMAL	NORMAL
ND145	59	M	Employee	GRADUAL, PROGRESSIVE	5 MONTHS	N	-	N	6\24	6\36	GRADE 1 NS + PSC	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	23	28	19	20	NORMAL	NORMAL
ND146	61	M	Retired	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\60	6\60	GRADE 2 NS + PSC	GRADE 2 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	16	14	19	19	NORMAL	NORMAL
ND147	44	M	Employee	NO	-	N	-	N	6\12	6\9	GRADE 1 NS + PSC	GARDE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	13	17	20	20	NORMAL	NORMAL
ND148	67	F	Housewife	GRADUAL, PROGRESSIVE	4 MONTHS	N	-	N	CF 3 M	CF 3M	GRADE 3 NS	GRADE 3 NS	WNL	WNL	-	N	N	Y	N	1	1	2	10	10	13	15	NORMAL	NORMAL
ND149	52	F	Teacher	NO	-	N	-	N	6\18	6\9	GRADE 2 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	15	17	18	17	NORMAL	NORMAL
ND150	42	F	Housewife	NO	-	N	-	N	6\9	6\12	GRADE 1 NS	GRADE 1 NS + CC	WNL	WNL	-	N	N	N	N	0	0	0	23	23	17	18	NORMAL	NORMAL
ND151	56	F	Housewife	NO	-	N	-	N	6\12	6\12	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	12	16	13	14	NORMAL	NORMAL
ND152	43	M	Employee	NO	-	N	-	N	6\12	6\9	GARDE 2 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	18	16	19	19	NORMAL	NORMAL
ND153	39	M	Employee	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\24	6\24	GARDE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	20	22	18	18	NORMAL	NORMAL
ND154	45	F	Employee	NO	-	N	-	N	6\12	6\9	GRADE 2 NS	GRADE 1 NS + CC	WNL	WNL	-	N	N	Y	Y	2	1	6	12	15	16	14	NORMAL	NORMAL
ND155	70	F	Housewife	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	CF 3M	CF 2M	GARDE 3 NS	GRADE 2 NS + PSC	GLAUCOMATOUS	GLAUCOMATOUS	-	Y	Y	N	N	2	3	12	6	5	5	5	REDUCED	REDUCED
ND156	60	F	Housewife	GRADUAL, PROGRESSIVE	9 MONTHS	N	-	N	CF 1M	CF 2M	GRADE 2 NS + PSC + CC	GRADE 2 NS + PSC	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	Y	1	1	2	15	15	17	18	NORMAL	NORMAL
ND157	53	F	Employee	NO	-	N	-	N	6\18	6\12	GRADE 1 NS + PSC	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	18	18	15	19	NORMAL	NORMAL
ND158	68	M	Farmer	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 1M	CF 2M	GRADE 3 NS + PSC	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	24	23	19	15	NORMAL	NORMAL
ND159	54	M	Employee	NO	-	N	-	N	6\9	6\12	GRADE 2 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	18	14	17	16	NORMAL	NORMAL
ND160	40	M	Employee	NO	-	N	-	N	6\9	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	26	29	22	22	NORMAL	NORMAL
ND161	36	M	Employee	NO	-	N	-	N	6\6	6\12	MILD PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	34	31	25	22	NORMAL	NORMAL
ND162	45	M	Employee	NO	-	N	-	N	6\12	6\12	PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	26	24	18	19	NORMAL	NORMAL
ND163	35	M	Employee	NO	-	N	-	N	6\9	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	33	32	17	21	NORMAL	NORMAL
ND164	50	F	Tailor	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\18	6\24	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	22	26	21	17	NORMAL	NORMAL
ND165	47	M	Watchmaker	GRADUAL, PROGRESSIVE	1 MONTH	N	-	N	6\12	6\9	PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	27	28	22	25	NORMAL	NORMAL
ND166	53	M	Employee	NO	-	N	-	N	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	Y	1	1	2	10	11	17	16	NORMAL	NORMAL
ND167	54	M	Employee	NO	-	N	-	N	6\36	6\24	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	18	15	19	19	NORMAL	NORMAL
ND168	78	M	Retired	GRADUAL, PROGRESSIVE	3 MONTHS	N	-	N	CF 3M	6\24	GRADE 3 NS	PSEUDOPHAKIA	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	27	29	21	20	NORMAL	NORMAL
ND169	80	M	Retired	GRADUAL, PROGRESSIVE	8 MONTHS	N	-	N	CF 1M	CF 2M	GRADE 2 NS	GRADE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	17	14	19	19	NORMAL	NORMAL
ND170	56	F	Teacher	NO	-	N	-	N	6\12	6\18	GRADE 1 NS + PSC	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	22	21	18	16	NORMAL	NORMAL
ND171	39	F	Employee	NO	-	N	-	N	6\9	6\9	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	17	18	21	21	NORMAL	NORMAL
ND172	44	M	Employee	NO	-	N	-	N	6\12	6\18	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	23	19	19	19	NORMAL	NORMAL
ND173	76	F	Retired	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\60	6\24	GRADE 3 NS	PSEUDOPHAKIA	WNL	WNL	-	N	N	N	N	0	0	0	11	15	15	16	NORMAL	NORMAL
ND174	66	F	Housewife	GRADUAL, PROGRESSIVE	1 YEAR	N	-	N	CF 2M	CF 2M	GRADE 3 NS + PSC	GRADE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	18	19	22	21	NORMAL	NORMAL

ND175	55	F	Housewife	NO	-	N	-	N	6\9	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	19	22	16	15	NORMAL	NORMAL
ND176	56	M	Employee	NO	-	N	-	N	6\12	6\12	GRADE 2 NS	GRADE 2 NS	WNL	WNL	-	N	N	N	N	0	0	0	29	15	18	18	NORMAL	NORMAL
ND177	67	M	Retired	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	6\36	6\60	GRADE 2 NS + PSC	GRADE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	15	16	15	15	NORMAL	NORMAL
ND178	80	F	Housewife	GRADUAL, PROGRESSIVE	2 YEARS	N	-	N	CF 3M	CF 3M	GRADE 3 NS	GRADE 3 NS	WNL	WNL	-	N	N	Y	N	1	2	3	11	10	13	13	NORMAL	NORMAL
ND179	35	F	Employee	NO	-	N	-	N	6\9	6\9	CLEAR	CLEAR	WNL	WNL		N	N	N	N	0	0	0	18	17	18	18	NORMAL	NORMAL
ND180	43	F	Employee	NO	-	N	-	N	6\12	6\18	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	29	23	18	19	NORMAL	NORMAL
ND181	44	M	Employee	NO	-	N	-	N	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	33	29	21	21	NORMAL	NORMAL
ND182	56	F	Housewife	GRADUAL, PROGRESSIVE	2 MONTHS	N	-	N	6\18	6\18	GRADE 2 NS	GRADE 1 NS + CC	WNL	WNL	-	N	N	N	Y	1	1	2	14	14	17	18	NORMAL	NORMAL
ND183	56	M	Employee	NO	-	N	-	N	6\12	6\12	GARDE 1 NS + PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	18	16	17	17	NORMAL	NORMAL
ND184	77	F	Housewife	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	CF 3M	CF 3M	GRADE 3 NS	GRADE 3 NS	WNL	WNL	-	N	N	N	N	0	0	0	18	19	21	21	NORMAL	NORMAL
ND185	45	F	Employee	NO	-	N	-	N	6\18	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	16	17	17	20	NORMAL	NORMAL
ND186	55	F	Employee	NO	-	N	-	N	6\9	6\9	GRADE 1 NS	GRADE 1 NS + CC	WNL	WNL	-	N	N	N	N	0	0	0	23	23	17	18	NORMAL	NORMAL
ND187	56	F	Employee	NO	-	N	-	N	6\18	6\12	GRADE 1 NS + CC	GARDE 1 NS	WNL	WNL	-	N	N	Y	N	1	2	3	34	32	21	21	NORMAL	NORMAL
ND188	39	M	Employee	NO	-	N	-	N	6\12	6\9	GRADE 1 NS	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	11	15	14	16	NORMAL	NORMAL
ND189	56	F	Housewife	GRADUAL, PROGRESSIVE	5 MONTHS	N	-	N	6\12	6\12	PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	16	17	15	15	NORMAL	NORMAL
ND190	48	M	Employee	NO	-	N	-	N	6\12	6\18	PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	18	18	14	16	NORMAL	NORMAL
ND191	56	F	Employee	NO	-	N	-	N	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	19	21	22	22	NORMAL	NORMAL
ND192	67	M	Housewife	GRADUAL, PROGRESSIVE	7 MONTHS	N	-	N	6\60	6\60	GRADE 2 NS	GRADE 2 NS	HTN RETINOPATHY	HTN RETINOPATHY	-	N	N	N	N	0	0	0	22	25	21	21	NORMAL	NORMAL
ND193	55	M	Employee	NO	-	N	-	N	6\12	6\9	GRADE 2 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	21	18	14	15	NORMAL	NORMAL
ND194	57	F	Employee	NO	-	N	-	N	6\9	6\9	PSC	PSC	WNL	WNL	-	N	N	N	N	0	0	0	14	17	18	18	NORMAL	NORMAL
ND195	44	M	Employee	NO	-	N	-	N	6\18	6\12	GRADE 2 NS	GRADE 1 NS	WNL	WNL	-	N	N	N	N	0	0	0	17	18	17	17	NORMAL	NORMAL
ND196	67	M	Retired	GRADUAL, PROGRESSIVE	6 MONTHS	N	-	N	CF 1 M	6\60	GRADE 3 NS	GRADE 2 NS + PSC	WNL	WNL	-	N	N	Y	N	1	1	2	15	15	18	18	NORMAL	NORMAL
ND197	44	M	Employee	NO	-	N	-	N	6\12	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	-	Y	Y	Y	Y	2	2	16	4	4	5	4	REDUCED	REDUCED
ND198	66	M	Retired	GRADUAL, PROGRESSIVE	8 MONTHS	N	-	N	CF 3M	CF 3M	GRADE 3 NS + PSC	GRADE 2 NS + PSC	WNL	WNL	-	N	N	N	N	0	0	0	12	17	15	16	NORMAL	NORMAL
ND199	34	F	Employee	NO	-	N	-	N	6\9	6\9	CLEAR	CLEAR	WNL	WNL	-	N	N	N	N	0	0	0	16	17	21	21	NORMAL	NORMAL
ND200	45	F	Employee	NO	-	N	-	N	6\12	6\6	MILD PSC	MILD PSC	WNL	WNL	-	N	N	N	N	0	0	0	17	13	17	17	NORMAL	NORMAL

STUDY ID	AGE	GENDER	OCCUPATION	DIMINUTION OF VISION	DURATION	H/O DIABETES MELLITUS	DURATION		BCVA		ANTERIOR SEGMENT		FUNDUS EXAMINATION		ETDRS GRADE OF DIABETIC RETINOPATHY	SYMPTOMS				FREQUENCY	SEVERITY	SPEED SCORE	SCHIRMERS II TEST (mm)		TBUT (sec)		CORNEAL NERVE SENSITIVITY	
								TREATMENT Y/N	OD	OS	OD	OS	OD	OS		GRITTINESS	BURNING	IRRITATION	EYE FATIGUE				OD	OS	OD	OS	OD	OS
D1	56	M	BANK EMPLOYEE	NO	-	Y	18 YEARS	Y	6\9	6\6	PSC	GRADE 1 NS	Microaneurysm, dot blot hemorrhages	Microaneurysm, dot blot hemorrhages	BE Moderate NPDR	N	N	N	Y	1	1	2	6	8	10	10	REDUCED	REDUCED
D2	52	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	15 YEARS	Y	6\60	6\24	GRADE 3 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	Y	N	N	1	1	2	8	8	10	10	NORMAL	NORMAL
D3	80	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	N	PL+ Pracc	CF 0.5m	GRADE 3 NS + PSC	GRADE 3 NS	Microaneurysm, dot blot hemorrhages, Fibrovascular membranes +	Microaneurysm, dot blot hemorrhages, Fibrovascular membranes +	BE PDR	Y	Y	Y	N	2	2	12	4	5	8	8	REDUCED	REDUCED
D4	50	F	HOUSEWIFE	GRADUAL PROGRESSIVE	7 MONTHS	Y	5 YEARS	Y	6\60	6\18	GRADE 2 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	1	1	4	12	15	14	14	NORMAL	NORMAL
D5	50	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1.5 MONTHS	Y	RECENTLY DIAGNOSED	N	CF 3M	6\24	GRADE 3 NS	GRADE 2 NS + CC	Microaneurysms, dot blot hemorrhages	Microaneurysms, dot blot hemorrhages	BE Moderate NPDR	N	Y	N	Y	2	2	8	4	4	5	3	REDUCED	REDUCED
D6	60	M	FARMER	GRADUAL PROGRESSIVE	1 YEAR	Y	10 YEARS	Y	CF 3M	6\60	GRADE 3 NS + PSC	GRADE 3 NS	Dot blot hemorrhages, cotton wool spots	Dot blot hemorrhages, cotton wool spots	BE Moderate NPDR	N	N	Y	N	1	2	3	8	10	10	10	REDUCED	REDUCED
D7	52	M	DRIVER	NO	-	Y	RECENTLY DIAGNOSED	N	6\18	6\24	GRADE 2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	26	32	19	18	NORMAL	NORMAL
D8	68	F	HOUSEWIFE	GRADUAL PROGRESSIVE	4 MONTHS	Y	RECENTLY DIAGNOSED	N	CF 1M	6\9	GRADE 2 NS + PSC	PSEUDOPHAKIA	Dot blot hemorrhages, cotton wool spots	Dot blot hemorrhages, cotton wool spots	BE Moderate NPDR	N	N	N	N	0	0	0	14	15	17	17	REDUCED	REDUCED
D9	60	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	6 MONTHS	Y	CF 2M	6\60	GRADE 3 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	1	1	2	20	21	16	16	NORMAL	NORMAL
D10	56	M	TEACHER	GRADUAL PROGRESSIVE	5 MONTHS	Y	2 YEARS	Y	6\60	6\24	GRADE 2 NS + PSC	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	1	1	2	15	17	18	18	NORMAL	NORMAL
D11	76	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	10 YEARS	Y	6\60	6\60	PSEUDOPHAKIA	PSEUDOPHAKIA	Dot blot hemorrhages, cotton wool spots, Fibrovascular membrane +	Dot blot hemorrhages, cotton wool spots, Fibrovascular membrane +	BE PDR	Y	Y	Y	N	2	2	12	3	3	7	6	REDUCED	REDUCED
D12	45	F	EMPLOYEE	NO	-	Y	5 MONTHS	Y	6\24	6\24	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	18	18	24	22	NORMAL	NORMAL
D13	65	F	HOUSEWIFE	GRADUAL PROGRESSIVE	8 MONTHS	Y	RECENTLY DIAGNOSED	N	CF 2M	CF 2M	GRADE 3 NS	GRADE 3 NS	Microenurysms	WNL	RE Mild NPDR	N	N	N	Y	1	2	3	9	9	9	9	NORMAL	NORMAL
D14	44	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\18	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	19	19	20	21	NORMAL	NORMAL
D15	52	M	SHOPKEEPER	NO	-	Y	4 MONTHS	Y	6\36	6\36	GRADE 1 NS	GRADE 1 NS	Microaneurysms	Microaneurysms	BE Mild NPDR	N	Y	N	Y	1	1	2	13	13	15	16	NORMAL	NORMAL
D16	60	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	4 YEARS	Y	CF 3M	6\60	PSEUDOPHAKIA	PSEUDOPHAKIA	Microaneurysm, dot blot hemorrhages, hard exudates	Microaneurysm, dot blot hemorrhages, Fibrovascular membranes +	RE Severe NPDR, LE PDR	Y	Y	N	N	2	1	6	7	6	7	7	REDUCED	REDUCED
D17	55	F	TEACHER	NO	-	Y	RECENTLY DIAGNOSED	N	6\18	6\12	GRADE 1 NS	PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	1	1	2	8	8	10	10	NORMAL	NORMAL
D18	56	M	EMPLOYEE	GRADUAL PROGRESSIVE	3 MONTHS	Y	3 YEARS	Y	6\24	6\36	GRADE 1 NS	GRADE 1 NS	Microanurysms	Microanurysms	BE Mild NPDR	N	N	Y	Y	1	1	2	8	8	9	9	NORMAL	NORMAL
D19	55	M	EMPLOYEE	GRADUAL PROGRESSIVE	9 MONTHS	Y	5 YEARS	Y	6\36	6\24	GRADE2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	Y	N	Y	2	2	4	10	10	15	13	NORMAL	NORMAL
D20	67	F	HOUSEWIFE	GRADUAL PROGRESSIVE	2 YEARS	Y	10 YEARS	Y	CF 3M	CF 1M	GRADE 3 NS + PSC	GRADE 3 NS + PSC	Microaneurysms	Microaneurysms	BE Mild NPDR	Y	Y	N	N	2	2	8	8	6	9	9	REDUCED	REDUCED
D21	36	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	2	3	9	9	12	15	NORMAL	NORMAL
D22	58	F	HOUSEWIFE	GRADUAL PROGRESSIVE	6 MONTHS	Y	8 YEARS	Y	6\36	6\60	PSEUDOPHAKIA	PSEUDOPHAKIA	Dot blot hemorrhages, cotton wool spots, Fibrovascular membrane +	Microaneurysms, dot blot hemorrhages, hard exudates	RE PDR, LE Moderate NPDR	Y	Y	Y	N	2	2	12	4	4	7	6	REDUCED	REDUCED
D23	45	F	TEACHER	NO	-	Y	RECENTLY DIAGNOSED	Y	6\36	6\24	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	16	18	15	15	NORMAL	NORMAL
D24	48	F	EMPLOYEE	NO	-	Y	3 YEARS	Y	6\60	6\24	PSC	PSC	Microaneurysm, dot blot hemorrhages	Microaneurysm, dot blot hemorrhages	BE Moderate NPDR	N	N	Y	Y	1	1	4	8	8	10	8	REDUCED	REDUCED
D25	75	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	17 YEARS	Y	CF 2M	CF 3M	GRADE 3 NS	GRADE 3 NS + PSC	Dot blot hemorrhages, cotton wool spots	Dot blot hemorrhages, cotton wool spots	BE Moderate NPDR	Y	Y	N	N	2	2	8	5	6	5	6	REDUCED	REDUCED
D26	56	M	EMPLOYEE	GRADUAL PROGRESSIVE	6 MONTHS	Y	2 YEARS	Y	6\36	6\18	GRADE 2 NS + PSC	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	2	3	17	15	15	15	NORMAL	NORMAL
D27	55	M	BANK EMPLOYEE	GRADUAL PROGRESSIVE	6 MONTHS	Y	4 YEARS	Y	6\12	6\12	GRADE 1 NS	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	23	26	17	18	NORMAL	NORMAL
D28	67	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1.5 YEARS	Y	RECENTLY DIAGNOSED	N	CF 3M	CF 3M	GRADE 3 NS	GRADE 2 NS + CC + PSC	Microaneurysms	Microaneurysms	BE Mild NPDR	N	N	N	N	0	0	0	9	9	9	9	REDUCED	REDUCED
D29	59	F	HOUSEWIFE	NO	-	Y	7 YEARS	Y	6\18	6\12	GRADE 2 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	Y	Y	N	Y	1	2	9	5	4	6	5	REDUCED	REDUCED
D30	44	M	EMPLOYEE	NO	-	Y	1 MONTH	Y	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	Y	Y	Y	Y	2	2	16	3	3	5	5	REDUCED	REDUCED
D31	65	M	RETIRED	NO	-	Y	13 YEARS	Y	6\9	6\9	PSEUDOPHAKIA	PSEUDOPHAKIA	WNL	WNL	NO RETINOPATHY	Y	Y	Y	Y	3	2	20	3	4	7	8	REDUCED	REDUCED
D32	56	M	TEACHER	GRADUAL PROGRESSIVE	5 MONTHS	Y	6 YEARS	Y	6\24	6\24	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	7	7	10	10	NORMAL	NORMAL
D33	65	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	12 YEARS	Y	6\60	6\60	GRADE 2 NS + PSC	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	1	1	2	11	14	17	17	NORMAL	NORMAL
D34	66	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	7 YEARS	Y	6\60	6\36	GRADE 2 NS + PSC	GRADE 2 NS	Microaneurysms, dot blot hemorrhages	Microaneurysms, dot blot hemorrhages	BE Moderate NPDR	N	N	Y	Y	1	2	6	9	9	9	9	REDUCED	REDUCED
D35	48	F	EMPLOYEE	NO	-	Y	5 MONTHS	Y	6\24	6\18	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	8	9	8	10	NORMAL	NORMAL
D36	43	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	16	16	17	18	NORMAL	NORMAL

D37	54	M	EMPLOYEE	NO	-	Y	2 YEARS	Y	6\36	6\36	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	1	2	3	8	8	10	10	NORMAL	NORMAL
D38	50	F	EMPLOYEE	GRADUAL PROGRESSIVE	2 MONTHS	Y	RECENTLY DIAGNOSED	Y	6\12	6\9	MILD PSC	MILD CC	WNL	WNL	NO RETINOPATHY	Y	N	N	Y	1	2	6	6	6	8	8	NORMAL	NORMAL
D39	44	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	9	10	10	10	NORMAL	NORMAL
D40	67	M	RETIRED	GRADUAL PROGRESSIVE	8 MONTHS	Y	14 YEARS	Y	6\60	CF 2M	PSEUDOPHAKIA	GRADE 3 NS	Microaneurysm, dot blot hemorrhages, hard exudates	Microaneurysm, dot blot hemorrhages	BE Moderate NPDR	N	Y	Y	N	2	2	8	5	5	8	8	REDUCED	REDUCED
D41	45	M	EMPLOYEE	NO	-	Y	5 MONTHS	Y	6\12	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	18	19	16	16	NORMAL	NORMAL
D42	46	F	TEACHER	NO	-	Y	1 YEAR	Y	6\18	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	15	17	16	NORMAL	NORMAL
D43	56	F	HOUSEWIFE	GRADUAL PROGRESSIVE	7 MONTHS	Y	4 YEARS	Y	6\36	6\36	PSC	PSC	Dot blot hemorrhages	Microaneurysms	RE Moderate NPDR, LE Mild NPDR	N	N	Y	Y	2	2	4	9	9	10	10	REDUCED	REDUCED
D44	46	F	HOUSEWIFE	GRADUAL PROGRESSIVE	3 MONTHS	Y	3 MONTHS	Y	6\60	6\24	GRADE 2 NS + PSC	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	19	17	13	15	NORMAL	NORMAL
D45	55	M	DRIVER	GRADUAL PROGRESSIVE	7 MONTHS	Y	1 YEAR	Y	6\24	6\24	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	Y	N	N	2	2	4	8	9	8	8	NORMAL	NORMAL
D46	46	M	EMPLOYEE	NO	-	Y	4 MONTHS	Y	6\18	6\12	PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	Y	N	Y	2	2	8	4	4	6	5	REDUCED	REDUCED
D47	39	M	TECHNICIAN	NO	-	Y	RECENTLY DIAGNOSED	N	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	Y	Y	Y	1	2	9	8	8	7	7	REDUCED	REDUCED
D48	43	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\9	6\9	GRADE 1 NS	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	17	16	15	NORMAL	NORMAL
D49	55	F	HOUSEWIFE	GRADUAL PROGRESSIVE	5 MONTHS	Y	3 YEARS	N	6\36	6\60	PSC	PSC	Dot blot hemorrhages	Dot blot hemorrhages	BE Moderate NPDR	N	N	Y	Y	2	2	4	10	10	12	13	REDUCED	REDUCED
D50	65	M	RETIRED	GRADUAL PROGRESSIVE	9 MONTHS	Y	7 YEARS	Y	6\60	6\60	GRADE 2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	9	9	10	10	REDUCED	REDUCED
D51	60	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	6 MONTHS	Y	CF 2M	6\60	GRADE 3 NS + PSC	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	20	21	16	16	NORMAL	NORMAL
D52	68	F	HOUSEWIFE	GRADUAL PROGRESSIVE	4 MONTHS	Y	RECENTLY DIAGNOSED	Y	CF 1M	6\9	GRADE 3 NS	PSEUDOPHAKIA	Dot blot hemorrhages, cotton wool spots +	Dot blot hemorrhages, cotton wool spots +	BE Moderate NPDR	N	N	N	N	0	0	0	9	9	12	12	REDUCED	REDUCED
D53	52	M	DRIVER	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	N	CF 3M	CF 3M	GRADE 3 NS	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	2	2	4	26	32	19	18	NORMAL	NORMAL
D54	60	M	FARMER	GRADUAL PROGRESSIVE	1 YEAR	Y	10 YEARS	Y	CF 3M	6\60	GRADE 2 NS	GRADE 2 NS	Dot blot hemorrhages, cotton wool spots	Dot blot hemorrhages, cotton wool spots	BE Moderate NPDR	N	Y	N	Y	2	2	4	10	10	10	10	REDUCED	REDUCED
D55	50	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1.5 YEARS	Y	RECENTLY DIAGNOSED	N	6\60	6\18	PSC	PSC	Microaneurysms	Microaneurysms	BE Mild NPDR	N	Y	N	Y	1	2	6	4	4	5	3	REDUCED	REDUCED
D56	50	F	HOUSEWIFE	GRADUAL PROGRESSIVE	7 MONTHS	Y	5 YEARS	Y	6\60	6\36	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	2	2	4	12	15	15	14	NORMAL	NORMAL
D57	80	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	N	CF 0.5M	CF 3M	GRADE 2 NS	GRADE 2 NS	Fibrovascular membranes around disc, hemorrhages	Hemorrhages, cotton wool spots	RE PDR, LE Moderate NPDR	N	Y	Y	N	2	2	4	6	6	8	8	REDUCED	REDUCED
D58	52	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	5 MONTHS	Y	6\60	6\24	GRADE 2 NS	GRADE 1 NS + CC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	8	8	10	10	NORMAL	NORMAL
D59	44	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\36	6\24	GRADE 1 NS	PSC	WNL	WNL	NO RETINOPATHY	Y	Y	Y	Y	2	2	16	4	4	3	3	NORMAL	NORMAL
D60	54	M	TEACHER	GRADUAL PROGRESSIVE	6 MONTHS	Y	2 YEARS	Y	6\12	6\36	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	Y	N	N	2	2	4	7	8	9	9	NORMAL	NORMAL
D61	65	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	12 YEARS	Y	6\60	CF 3M	GRADE 2 NS	GRADE 3 NS	Dot blot hemorrhages	Dot blot hemorrhages, cotton wool spots +	BE Moderate NPDR	Y	Y	N	N	2	2	8	3	3	5	5	REDUCED	REDUCED
D62	66	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	5 YEARS	Y	CF 2M	CF 2M	GRADE 2 NS + CC	GRADE 2 NS	Fibrovascular membranes in the periphery, hemorrhages	Dot blot hemorrhages, cotton wool spots +	RE PDR, LE Moderate NPDR	Y	Y	N	Y	1	2	9	9	9	10	10	REDUCED	REDUCED
D63	76	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	15 YEARS	Y	6\18	CF 1M	PSEUDOPHAKIA	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	17	16	15	REDUCED	REDUCED
D64	54	M	EMPLOYEE	GRADUAL PROGRESSIVE	8 MONTHS	Y	10 YEARS	Y	6\12	6\18	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	11	16	21	19	NORMAL	NORMAL
D65	57	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	N	CF 3M	6\36	GRADE 3 NS	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	9	8	8	8	REDUCED	REDUCED
D66	43	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\18	6\12	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	16	16	15	14	NORMAL	NORMAL
D67	52	F	HOUSEWIFE	GRADUAL PROGRESSIVE	6 MONTHS	Y	8 YEARS	Y	CF 3M	6\24	GRADE 3 NS	PSC	Microaneurysms	Microaneurysms, dot bot hemorrhages	RE Mild NPDR, LE Moderate NPDR	N	Y	N	N	1	2	3	9	9	10	10	REDUCED	REDUCED
D68	55	M	TEACHER	GRADUAL PROGRESSIVE	1 YEAR	Y	5 YEARS	Y	6\24	6\36	PSC	PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	15	18	17	NORMAL	NORMAL
D69	67	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	15 YEARS	Y	CF 2M	CF 3M	GRADE 3 NS	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	10	10	14	15	NORMAL	NORMAL
D70	58	M	TEACHER	GRADUAL PROGRESSIVE	1 YEAR	Y	10 MONTHS	Y	6\60	6\36	GRADE 2 NS + CC	GRADE 2 NS	GLAUCOMATOUS	GLAUCOMATOUS	NO RETINOPATHY	N	N	N	N	0	0	0	17	19	15	15	NORMAL	NORMAL
D71	56	M	EMPLOYEE	GRADUAL PROGRESSIVE	6 MONTHS	Y	2 YEARS	Y	6\36	6\36	GRADE 2 NS	GRADE 2 NS	Microaneurysms, dot blot hemorrhages	Microaneurysms, dot blot hemorrhages	BE Moderate NPDR	N	N	N	N	0	0	0	6	6	8	8	REDUCED	REDUCED
D72	67	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	4 YEARS	Y	CF 1M	CF 3M	GRADE 3 NS	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	Y	Y	N	N	2	2	4	8	8	10	10	NORMAL	NORMAL
D73	77	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	12 YEARS	Y	6\60	6\60	PSEUDOPHAKIA	PSEUDOPHAKIA	Microaneurysms, dot blot hemorrhages in 2 quadrants, exudates in periphery	Microaneurysms, dot blot hemorrhages in 4 quadrants	RE Moderate NPDR, LE Severe NPDR	Y	N	Y	N	2	2	8	6	6	5	7	REDUCED	REDUCED
D74	56	F	TAILOR	GRADUAL PROGRESSIVE	1 YEAR	Y	5 YEARS	Y	6\36	6\36	GRADE 2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	12	14	16	16	NORMAL	NORMAL

D75	60	M	TEACHER	GRADUAL PROGRESSIVE	1 YEAR	Y	2 YEARS	Y	6\60	6\60	GRADE 2 NS + CC	GRADE 2 NS	Microaneuryms, dot blot hemorrhages in 1 quadrant	Microaneurysms, Dot blot hemorrhages in 2 quadrants	BE Moderate NPDR	N	N	N	N	0	0	0	7	8	7	7	REDUCED	REDUCED
D76	66	M	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	5 YEARS	Y	CF 3M	CF 2M	GRADE 2 NS + PSC	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	10	13	12	14	NORMAL	NORMAL
D77	52	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\24	6\18	GRADE 1 NS	GRADE 1 NS + CC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	19	21	18	18	NORMAL	NORMAL
D78	66	M	BANK EMPLOYEE	NO	-	Y	18 YEARS	Y	6\24	6\36	GRADE 2 NS	MILD PSC	Microaneurysms, dot blot hemorrhages	Microaneurysms, dot blot hemorrhages, venous beading	RE Moderate NPDR, LE Severe NPDR	N	N	Y	N	1	1	2	6	8	10	10	REDUCED	REDUCED
D79	72	M	RETIRED	GRADUAL PROGRESSIVE	9 MONTHS	Y	RECENTLY DIAGNOSED	N	6\12	CF 3M	PSEUDOPHAKIA	GRADE 3 NS	Microaneurysms, dot blot hemorrhages in 1 quadrant	Microaneurysms, dot blot hemorrhages in 1 quadrant	BE Moderate NPDR	N	N	N	N	0	0	0	9	9	10	10	REDUCED	REDUCED
D80	55	F	MAID	GRADUAL PROGRESSIVE	1 YEAR	Y	6 MONTHS	Y	CF 1M	CF 1M	GRADE 2 NS	GRADE 3 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	N	1	1	2	9	9	8	8	NORMAL	NORMAL
D81	53	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	Y	6\24	6\24	GRADE 1 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	1	1	4	12	15	16	16	NORMAL	NORMAL
D82	45	M	EMPLOYEE	NO	-	Y	2 MONTHS	Y	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	16	16	16	NORMAL	NORMAL
D83	67	M	RETIRED	GRADUAL PROGRESSIVE	2 YEARS	Y	12 YEARS	Y	CF 1 M	6\60	PSEUDOPHAKIA	PSEUDOPHAKIA	Microaneurysms, dot blot hemorrhages in 2 quadrants, exudates in periphery	Microaneurysms, dot blot hemorrhages in 2 quadrants, exudates in periphery	BE Moderate NPDR	Y	Y	Y	N	2	3	15	5	5	7	6	REDUCED	REDUCED
D84	56	M	TEACHER	GRADUAL PROGRESSIVE	8 MONTHS	Y	5 YEARS	Y	6\60	6\36	GRADE 2 NS	GRADE 2 NS + CC	Microaneurysms	Microaneurysms	BE Mild NPDR	Y	N	Y	N	2	2	8	9	9	10	10	REDUCED	REDUCED
D85	56	F	TAILOR	GRADUAL PROGRESSIVE	1 YEAR	Y	2 YEARS	Y	6\12	6\18	GRADE 1 NS	GRADE 1 NS + CC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	18	18	20	NORMAL	NORMAL
D86	78	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	10 YEARS	Y	CF 3M	CF 2M	GRADE 3 NS + PSC	GRADE 3 NS + PSC	Microaneurysms	WNL	RE Mild NPDR, LE No Retinopathy	N	N	N	N	0	0	0	11	14	16	16	REDUCED	REDUCED
D87	60	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	6 YEARS	Y	6\24	6\36	GRADE 2 NS + PSC	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	16	18	20	20	REDUCED	REDUCED
D88	56	M	EMPLOYEE	GRADUAL PROGRESSIVE	7 MONTHS	Y	RECENTLY DIAGNOSED	N	6\36	6\60	GRADE 1 NS	GRADE 1 NS	Microaneurysms, dot blot hemorrhages in 4 quadrants	Microaneurysms, dot blot hemorrhages in 1 quadrant	BE Moderate NPDR	Y	Y	Y	Y	2	2	16	4	4	6	5	REDUCED	REDUCED
D89	66	M	RETIRED	GRADUAL PROGRESSIVE	9 MONTHS	Y	1 YEAR	Y	6\60	6\60	GRADE 2 NS	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	15	18	19	REDUCED	REDUCED
D90	55	F	HOUSEWIFE	NO	-	Y	RECENTLY DIAGNOSED	N	6\18	6\18	GRADE 1 NS	GRADE 1 NS	Microaneurysms	Microaneurysms	BE Mild NPDR	N	N	N	Y	2	2	4	9	9	9	10	REDUCED	REDUCED
D91	78	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	10 YEARS	Y	CF 1M	CF 2M	GRADE 2 NS + PSC	GRADE 2 NS + CC	Microaneurysms	WNL	RE Mild NPDR, LE No Retinopathy	N	N	N	N	0	0	0	12	11	8	8	REDUCED	REDUCED
D92	56	M	BUSINESS	GRADUAL PROGRESSIVE	6 MONTHS	Y	2 YEARS	Y	CF 3M	6\60	GARDE 3 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	N	1	1	2	14	16	15	15	REDUCED	REDUCED
D93	64	M	RETIRED	GRADUAL PROGRESSIVE	3 MONTHS	Y	8 YEARS	Y	6\36	6\12	DENSE PSC	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	10	10	12	12	NORMAL	NORMAL
D94	62	M	MASON	GRADUAL PROGRESSIVE	2 MONTHS	Y	6 YEARS	Y	6\36	6\36	GRADE 2 NS	GRADE 2 NS	Microaneurysms, dot blot hemorrhages in 2 quadrants, exudates in periphery	Microaneurysms, dot blot hemorrhages in 2 quadrants, exudates in periphery	BE Moderate NPDR	N	N	Y	N	1	1	2	4	4	5	5	REDUCED	REDUCED
D95	68	F	HOUSEWIFE	GRADUAL PROGRESSIVE	8 MONTHS	Y	RECENTLY DIAGNOSED	N	CF 1M	6\18	GRADE 3 NS + PSC	PSEUDOPHAKIA	Microaneurysms, dot blot hemorrhages in 2 quadrant	Microaneurysms, dot blot hemorrhages in 2 quadrant	BE Moderate NPDR	N	Y	N	Y	2	2	8	8	6	9	9	REDUCED	REDUCED
D96	55	M	SHOPKEEPER	GRADUAL PROGRESSIVE	3 MONTHS	Y	RECENTLY DIAGNOSED	Y	CF 0.5M	CF 2M	GRADE 3 NS + PSC	GRADE 2 NS + CC	Microaneurysms, dot blot hemorrhages in 4 quadrants	Microaneurysms, dot blot hemorrhages	RE Severe NPDR, LE Moderate NPDR	N	N	Y	N	2	2	4	9	8	10	10	REDUCED	REDUCED
D97	65	F	HOUSEWIFE	GRADUAL PROGRESSIVE	6 MONTHS	Y	RECENTLY DIAGNOSED	Y	CF 2M	CF 1M	GRADE 3 NS	GRADE 3 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	9	8	8	9	REDUCED	REDUCED
D98	63	M	FARMER	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	Y	6\60	CF 3M	PSEUDOPHAKIA	GRADE 2 NS	Microaneurysms	Microaneurysms	BE Mild NPDR	N	N	Y	N	2	2	4	15	14	20	20	NORMAL	NORMAL
D99	65	F	HOUSEWIFE	GRADUAL PROGRESSIVE	5 MONTHS	Y	6 YEARS	Y	CF 3M	6\18	PSEUDOPHAKIA	PSEUDOPHAKIA	Vitreous hemorrhage	Dot blot hemorrhages in 4 quadrants	RE PDR , LE Severe NPDR	N	Y	Y	N	2	2	8	6	6	8	6	REDUCED	REDUCED
D100	55	F	HOUSEWIFE	GRADUAL PROGRESSIVE	10 MONTHS	Y	5 YEARS	Y	CF 3M	6\36	GRADE 3 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	N	2	2	4	5	5	8	8	REDUCED	REDUCED
D101	67	M	RETIRED	GRADUAL PROGRESSIVE	2 YEARS	Y	7 YEARS	Y	CF 2M	6\60	GRADE 2 NS + PSC	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	18	16	17	NORMAL	NORMAL
D102	48	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\18	6\12	GRADE 1 NS	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	10	10	9	10	NORMAL	NORMAL
D103	58	M	SHOPKEEPER	GRADUAL PROGRESSIVE	2 MONTHS	Y	5 YEARS	Y	6\36	6\60	GRADE 1 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	17	18	18	18	NORMAL	NORMAL
D104	66	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	7 YEARS	Y	6\60	6\60	GRADE 2 NS + PSC	GRADE 2 NS	Cotton wool spots	Microaneurysms	RE Moderate NPDR, LE Mild NPDR	Y	Y	Y	N	1	2	9	7	7	5	6	REDUCED	REDUCED
D105	59	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	6 MONTHS	Y	6\36	6\24	MILD PSC	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	19	15	15	NORMAL	NORMAL
D106	67	F	HOUSEWIFE	GRADUAL PROGRESSIVE	8 MONTHS	Y	2 YEARS	Y	6\60	CF 1M	GRADE 1 NS	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	13	16	17	17	NORMAL	NORMAL
D107	54	M	TEACHER	NO	-	Y	RECENTLY DIAGNOSED	Y	6\18	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	19	20	22	NORMAL	NORMAL
D108	39	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	Y	Y	N	N	2	1	6	8	8	9	10	NORMAL	NORMAL
D109	40	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\9	6\6	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	9	10	10	10	NORMAL	NORMAL
D110	50	M	SHOPKEEPER	GRADUAL PROGRESSIVE	4 MONTHS	Y	RECENTLY DIAGNOSED	Y	6\12	6\12	MILD PSC	MILD PSC	Microaneurysms	Microaneurysms	BE Mild NPDR	N	N	Y	N	2	2	4	8	8	6	7	NORMAL	NORMAL
D111	57	F	HOUSEWIFE	GRADUAL PROGRESSIVE	6 MONTHS	Y	10 MONTHS	Y	6\36	6\24	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	19	20	20	REDUCED	REDUCED
D112	46	M	DRIVER	NO	-	Y	RECENTLY DIAGNOSED	Y	6\12	6\18	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	15	16	16	NORMAL	NORMAL

D113	76	M	RETIRED	GRADUAL PROGRESSIVE	2 YEARS	Y	15 YEARS	Y	CFCF	CF 3M	GRADE 3 NS	GRADE 3 NS	Dot blot hemorrhages in 4 quadrants	Fibrovascular membrane	RE Moderate NPDR, LE PDR	Y	Y	Y	N	2	2	12	3	3	5	4	REDUCED	REDUCED
D114	37	M	TEACHER	NO	-	Y	RECENTLY DIAGNOSED	N	6\9	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	18	18	19	NORMAL	NORMAL
D115	60	M	FARMER	GRADUAL PROGRESSIVE	1 YEAR	Y	4 YEARS	Y	6\60	6\36	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	10	9	10	10	NORMAL	NORMAL
D116	65	M	FARMER	GRADUAL PROGRESSIVE	1 YEAR	Y	5 YEARS	Y	6\60	CF 3M	GRADE 2 NS	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	16	17	18	18	REDUCED	REDUCED
D117	70	F	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	10 YEARS	Y	6\60	6\36	PSEUDOPHAKIA	PSEUDOPHAKIA	Microaneurysms, dot blot hemorrhages in 4 quadrants	Microaneurysms, dot blot hemorrhages in 4 quadrants	BE Severe NPDR	Y	N	Y	N	2	2	8	4	4	7	6	REDUCED	REDUCED
D118	56	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	N	6\18	6\12	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	17	18	18	NORMAL	NORMAL
D119	55	F	HOUSEWIFE	GRADUAL PROGRESSIVE	7 MONTHS	Y	2 YEARS	Y	6\24	6\18	GRADE 2 NS	GRADE 1 NS + CC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	10	10	12	13	NORMAL	NORMAL
D120	45	M	TEACHER	NO	-	Y	RECENTLY DIAGNOSED	Y	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	Y	Y	Y	Y	2	3	24	3	3	6	6	REDUCED	REDUCED
D121	37	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\9	6\6	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	16	16	17	15	NORMAL	NORMAL
D122	44	F	EMPLOYEE	GRADUAL PROGRESSIVE	2 MONTHS	Y	RECENTLY DIAGNOSED	Y	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	13	14	18	18	NORMAL	NORMAL
D123	55	M	EMPLOYEE	GRADUAL PROGRESSIVE	6 MONTHS	Y	4 YEARS	Y	6\36	6\36	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	N	1	1	2	14	14	16	17	NORMAL	NORMAL
D124	67	M	RETIRED	GRADUAL PROGRESSIVE	9 MONTHS	Y	8 YEARS	Y	CF 3M	6\60	GRADE 2 NS + PSC	GRADE 2 NS + CC	Microaneurysm	Microaneurysm	BE Mild NPDR	N	Y	N	Y	1	1	4	9	9	9	9	REDUCED	REDUCED
D125	60	M	FARMER	GRADUAL PROGRESSIVE	1 YEAR	Y	1 YEAR	Y	6\60	6\24	GRADE 2 NS	GRADE 1 NS + CC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	13	13	18	19	NORMAL	NORMAL
D126	60	F	HOUSEWIFE	GRADUAL PROGRESSIVE	2 MONTHS	Y	RECENTLY DIAGNOSED	Y	CF 3M	CF 2M	GRADE 2 NS	GRADE 2 NS + PSC	Neovascularization, Hard exudates	Dot blot hemorrhages in 3 quadrants, Hard exudates	RE PDR, LE Severe NPDR	Y	Y	N	N	2	2	8	4	4	6	7	REDUCED	REDUCED
D127	42	M	EMPLOYEE	GRADUAL PROGRESSIVE	2 MONTHS	Y	5 YEARS	Y	6\9	6\18	MILD PSC	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	18	22	15	15	NORMAL	NORMAL
D128	60	M	RETIRED	GRADUAL PROGRESSIVE	6 MONTHS	Y	11 YEARS	Y	6\18	6\18	GRADE 2 NS	GRADE 2 NS	Dot blot hemorrhages in 1 quadrant	Dot blot hemorrhages in 1 quadrant, macular edema	RE Moderate NPDR, LE CSME	N	N	N	Y	1	1	2	2	4	8	8	REDUCED	REDUCED
D129	50	M	TEACHER	GRADUAL PROGRESSIVE	3 MONTHS	Y	RECENTLY DIAGNOSED	Y	6\12	6\18	GRADE 2 NS	GRADE 2 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	16	18	18	20	NORMAL	NORMAL
D130	54	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	8 MONTHS	Y	6\9	6\12	GRADE 1 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	Y	Y	Y	N	2	2	12	6	6	4	4	REDUCED	REDUCED
D131	67	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	12 YEARS	Y	6\12	CF 2M	PSEUDOPHAKIA	GRADE 3 NS + PSC	Dot blot hemorrahges in 1 quadrant	Dot blot hemorrahges in 1 quadrant	BE Moderate NPDR	N	N	Y	N	1	1	2	9	9	10	10	REDUCED	REDUCED
D132	38	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\6	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	12	12	16	16	NORMAL	NORMAL
D133	48	M	BANK EMPLOYEE	GRADUAL PROGRESSIVE	3 MONTHS	Y	RECENTLY DIAGNOSED	Y	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	18	17	18	NORMAL	NORMAL
D134	55	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	2 YEARS	Y	6\24	6\12	GRADE 1 NS + PSC	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	Y	N	Y	N	2	3	12	4	5	5	5	REDUCED	REDUCED
D135	67	F	HOUSEWIFE	GRADUAL PROGRESSIVE	2 YEARS	Y	8 YEARS	Y	6\60	CF 2M	GRADE 2 NS + PSC	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	10	10	13	14	NORMAL	NORMAL
D136	43	F	EMPLOYEE	NO	-	Y	2 MONTHS	Y	6\9	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	Y	Y	N	2	2	8	9	9	9	9	REDUCED	REDUCED
D137	48	M	EMPLOYEE	GRADUAL PROGRESSIVE	2 MONTHS	Y	RECENTLY DIAGNOSED	Y	6\18	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	16	16	17	19	NORMAL	NORMAL
D138	55	M	TEACHER	GRADUAL PROGRESSIVE	5 MONTHS	Y	1 YEAR	Y	6\24	6\24	GRADE2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	8	8	6	7	NORMAL	NORMAL
D139	56	F	HOUSEWIFE	GRADUAL PROGRESSIVE	3 MONTHS	Y	6 YEARS	Y	6\36	6\12	GRADE 2 NS + CC	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	N	1	1	2	10	14	16	16	NORMAL	NORMAL
D140	55	F	EMPLOYEE	GRADUAL PROGRESSIVE	8 MONTHS	Y	6 MONTHS	Y	6\12	6\24	GRADE 1 NS	GRADE 2 NS	Microaneurysms	Microaneurysms	BE Mild NPDR	N	Y	N	Y	1	2	6	9	9	9	9	REDUCED	REDUCED
D141	35	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	21	22	18	NORMAL	NORMAL
D142	42	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\9	6\12	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	Y	2	2	4	5	5	6	5	REDUCED	REDUCED
D143	58	M	DRIVER	GRADUAL PROGRESSIVE	5 MONTHS	Y	10 YEARS	Y	6\36	6\60	GRADE 2 NS + PSC	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	20	22	27	23	NORMAL	NORMAL
D144	45	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	6	5	7	7	NORMAL	NORMAL
D145	66	F	HOUSEWIFE	GRADUAL PROGRESSIVE	2 YEARS	Y	15 YEARS	Y	CF 3M	CF 3M	GRADE 3 NS	GRADE 3 NS	Dot blot hemorrhages in 1 quadrant	Dot blot hemorrhages in 1 quadrant	BE Moderate NPDR	N	N	Y	N	1	1	2	10	10	8	9	REDUCED	REDUCED
D146	56	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	6 YEARS	Y	6\24	6\60	GARDE 2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	23	25	25	28	NORMAL	NORMAL
D147	67	M	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	13 YEARS	Y	6\24	6\12	PSEUDOPHAKIA	PSEUDOPHAKIA	Cotton wool spots	Cotton wool spots, Dot blot hemorrhages	BE Moderate NPDR	Y	N	N	N	2	3	6	7	7	8	6	REDUCED	REDUCED
D148	45	M	TEACHER	GRADUAL PROGRESSIVE	8 MONTHS	Y	3 MONTHS	Y	6\36	6\18	GRADE 2 NS + PSC	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	Y	N	1	2	3	16	16	13	14	REDUCED	REDUCED
D149	63	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	7 YEARS	Y	CF 2 M	6\60	GRADE 2 NS	PSEUDOPHAKIA	Dot blot hemorrahges in 1 quadrant, cotton wool spots	Dot blot hemorrahges in 1 quadrant, cotton wool spots	BE Moderate NPDR	N	Y	N	N	1	2	3	9	9	8	8	REDUCED	REDUCED
D150	52	F	HOUSEWIFE	GRADUAL PROGRESSIVE	4 MONTHS	Y	9 MONTHS	Y	6\60	6\36	GARDE 2 NS	GRADE 1 NS + CC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	16	18	21	21	NORMAL	NORMAL

D151	41	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	6	5	6	6	REDUCED	REDUCED
D152	48	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 MONTH	Y	5 MONTHS	Y	6\9	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	N	1	1	2	17	17	18	19	NORMAL	NORMAL
D153	56	F	EMPLOYEE	GRADUAL PROGRESSIVE	7 MONTHS	Y	1 YEAR	Y	6\12	6\36	MILD PSC	GARDE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	16	19	21	NORMAL	NORMAL
D154	61	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	6 YEARS	Y	6\60	CF 1M	GRADE 3 NS	GARDE 3 NS + PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	2	2	8	9	8	9	9	REDUCED	REDUCED
D155	63	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	9 YEARS	Y	CF 2M	CF 3M	GRADE 2 NS	PSEUDOPHAKIA	Dot blot hemorrhages in 4 quadrants, hard exudates	Dot blot hemorrahges, cotton wool spots	BE Moderate NPDR	N	Y	Y	Y	1	1	6	10	10	12	13	REDUCED	REDUCED
D156	78	M	RETIRED	GRADUAL PROGRESSIVE	2 YEARS	Y	18 YEARS	Y	CF 3M	6\60	PSEUDOPHAKIA	PSEUDOPHAKIA	Neovascularization	Fibrovascular membrane	BE PDR	Y	Y	Y	Y	2	2	16	3	2	3	4	REDUCED	REDUCED
D157	69	F	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	6 YEARS	Y	6\60	6\36	GRADE 3 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	9	21	21	REDUCED	REDUCED
D158	76	F	HOUSEWIFE	GRADUAL PROGRESSIVE	2 YEARS	Y	20 YEARS	Y	6\60	CF 3M	PSEUDOPHAKIA	PSEUDOPHAKIA	Dot blot hemorrhages in 4 quadrants, cotton wool spots	Dot blot hemorrhages in 4 quadrants, cotton wool spots	BE Severe NPDR	N	Y	Y	N	2	2	16	7	7	4	6	REDUCED	REDUCED
D159	54	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	RECENTLY DIAGNOSED	N	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	23	24	17	17	NORMAL	NORMAL
D160	44	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	10	13	16	16	NORMAL	NORMAL
D161	55	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	4 MONTHS	Y	6\12	6\18	GRADE 1 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	17	16	14	16	NORMAL	NORMAL
D162	61	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	2 YEARS	Y	6\60	6\60	GRADE2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	17	18	16	16	NORMAL	NORMAL
D163	43	F	TEACHER	NO	-	Y	6 MONTHS	Y	6\9	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	15	16	19	19	NORMAL	NORMAL
D164	50	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	8 MONTHS	Y	6\18	6\24	GRADE 1NS	MILD PSC	MICROANEURYSMS	MICROANEURYSMS	BE Mild NPDR	N	N	Y	N	1	1	2	7	8	5	7	REDUCED	REDUCED
D165	51	M	EMPLOYEE	GRADUAL PROGRESSIVE	5 MONTHS	Y	1 YEAR	Y	6\12	6\24	GRADE 1 NS	GARDE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	20	22	21	21	REDUCED	REDUCED
D166	39	M	DRIVER	NO	-	Y	RECENTLY DIAGNOSED	Y	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	23	21	19	19	NORMAL	NORMAL
D167	44	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 MONTH	Y	RECENTLY DIAGNOSED	Y	6\12	6\9	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	N	1	1	2	7	8	9	9	REDUCED	REDUCED
D168	66	M	RETIRED	GRADUAL PROGRESSIVE	4 MONTHS	Y	15 YEARS	Y	CF 2M	6\60	GRADE 2 NS	GRADE 2NS	Dot blot hemorrhages in 1 quadrant	Dot blot hemorrhages in 1 quadrant	BE Moderate NPDR	N	Y	N	N	2	2	4	10	10	13	14	REDUCED	REDUCED
D169	34	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\9	6\6	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	17	19	19	NORMAL	NORMAL
D170	50	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	7 MONTHS	Y	6\24	6\60	GRADE 2 NS	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	Y	N	Y	1	1	4	9	9	10	10	REDUCED	REDUCED
D171	47	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\12	6\9	GRADE 1NS	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	18	18	20	NORMAL	NORMAL
D172	53	F	HOUSEWIFE	GRADUAL PROGRESSIVE	4 MONTHS	Y	1 YEAR	Y	6\24	6\60	GRADE 1 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	8	7	9	9	NORMAL	NORMAL
D173	65	M	HOUSEWIFE	GRADUAL PROGRESSIVE	3 YEARS	Y	12 YEARS	Y	CF CF	CF 2M	PSEUDOPHAKIA	PSEUDOPHAKIA	Fibrovascular membrane	Dot blot hemorrhages in 4 quadrants, cotton wool spots	RE PDR, LE Moderate NPDR	Y	N	N	Y	2	2	8	10	12	11	12	REDUCED	REDUCED
D174	33	F	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	N	6\12	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	16	21	19	NORMAL	NORMAL
D175	36	F	EMPLOYEE	NO	-	Y	5 MONTHS	Y	6\9	6\12	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	16	18	23	23	NORMAL	NORMAL
D176	48	M	EMPLOYEE	GRADUAL PROGRESSIVE	2 MONTHS	Y	3 YEARS	Y	6\24	6\36	GRADE 1 NS	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	9	9	8	7	NORMAL	NORMAL
D177	40	F	EMPLOYEE	NO	-	Y	6 MONTHS	Y	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	23	26	20	20	NORMAL	NORMAL
D178	56	F	RETIRED	GRADUAL PROGRESSIVE	4 MONTHS	Y	5 YEARS	Y	6\24	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	Y	Y	2	2	8	9	8	7	7	REDUCED	REDUCED
D179	76	M	RETIRED	GRADUAL PROGRESSIVE	5 YEARS	Y	23 YEARS	Y	CFCF	HMCF PL+ Pracc	PSEUDOPHAKIA	PSEUDOPHAKIA	Dot blot hemorrhages in 4 quadrants, cotton wool spots, macular edema	Fibrovascular membrane , vitreous hemorrhag	RE Severe NPDR, LE PDR	N	N	Y	Y	2	2	8	5	6	6	6	REDUCED	REDUCED
D180	33	M	EMPLOYEE	NO	-	Y	RECENTLY DIAGNOSED	Y	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	19	21	22	21	NORMAL	NORMAL
D181	45	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	2 YEARS	Y	6\12	6\9	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	19	21	22	19	NORMAL	NORMAL
D182	67	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	15 YEARS	Y	6\60	CF 3M	GRADE 2 NS	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	21	22	17	17	NORMAL	NORMAL
D183	87	F	HOUSEWIFE	GRADUAL PROGRESSIVE	2 YEAR	Y	6 YEARS	Y	HMCF PL+ Pracc	cf 3 m	PSEUDOPHAKIA	PSEUDOPHAKIA	fibrovascular membrane	Fibrovascular membrane	BE PDR	Y	Y	N	N	2	2	8	9	6	5	5	REDUCED	REDUCED
D184	56	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	8 YEARS	Y	6\60	6\36	GRADE 2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	15	18	18	18	NORMAL	NORMAL
D185	44	M	EMPLOYEE	NO		Y	RECENTLY DIAGNOSED	Y	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	22	21	21	18	NORMAL	NORMAL
D186	38	M	EMPLOYEE	NO		Y	RECENTLY DIAGNOSED	Y	6\9	6\12	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	7	8	9	9	REDUCED	REDUCED
D187	80	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	16 YEARS	Y	CF 3M	CF 3M	PSEUDOPHAKIA	PSEUDOPHAKIA	Fibrovascular membrane	Neovascularization	BE PDR	N	Y	Y	N	2	1	6	10	10	15	15	REDUCED	REDUCED
D188	72	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	9 MONTHS	Y	6\60	6\24	GRADE 3 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	18	17	19	14	REDUCED	REDUCED

D189	59	F	TEACHER	GRADUAL PROGRESSIVE	1 YEAR	Y	11 YEARS	Y	6\36	6\36	GRADE 2 NS	GRADE 2 NS + CC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	14	19	16	16	NORMAL	NORMAL
D190	61	F	HOUSEWIFE	GRADUAL PROGRESSIVE	8 MONTHS	Y	7 YEARS	Y	6\60	CF 2M	PSEUDOPHAKIA	GARDE 3 NS + PSC	Microaneurysms	WNL	RE Mild NPDR	N	N	N	N	0	0	0	9	8	9	10	REDUCED	REDUCED
D191	64	F	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	5 YEARS	Y	CF 2M	CF 3M	GRADE 2 NS + PSC	GRADE 3 NS	WNL	WNL	NO RETINOPATHY	Y	N	Y	N	1	1	2	9	9	9	9	NORMAL	NORMAL
D192	44	M	EMPLOYEE	NO		Y	RECENTLY DIAGNOSED	Y	6\12	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	21	24	16	16	NORMAL	NORMAL
D193	48	M	EMPLOYEE	NO		Y	5 MONTHS	Y	6\9	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	13	15	16	17	NORMAL	NORMAL
D194	65	M	HOUSEWIFE	GRADUAL PROGRESSIVE	1 YEAR	Y	2 YEARS	Y	6\24	6\60	GRADE 1 NS	GRADE 2NS	Dot blot hemorrhages in 1 quadrant	Dot blot hemorrhages in 1 quadrant	BE Moderate NPDR	N	N	Y	N	1	1	2	9	7	6	5	REDUCED	REDUCED
D195	55	M	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	3 YEARS	Y	6\18	6\24	GRADE 1 NS	GRADE 1 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	13	17	20	20	NORMAL	NORMAL
D196	51	F	TEACHER	GRADUAL PROGRESSIVE	5 MONTHS	Y	9 MONTHS	Y	6\9	6\12	MILD PSC	MILD PSC	WNL	WNL	NO RETINOPATHY	Y	Y	N	N	2	2	8	5	5	4	5	REDUCED	REDUCED
D197	39	F	EMPLOYEE	NO		Y	RECENTLY DIAGNOSED	Y	6\9	6\9	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	Y	1	1	2	15	17	19	19	NORMAL	NORMAL
D198	37	F	EMPLOYEE	GRADUAL PROGRESSIVE	3 MONTHS	Y	5 MONTHS	Y	6\12	6\12	CLEAR	CLEAR	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	9	9	9	9	REDUCED	REDUCED
D199	76	M	RETIRED	GRADUAL PROGRESSIVE	1 YEAR	Y	8 YEARS	Y	CF 3M	CF 2M	PSEUDOPHAKIA	PSEUDOPHAKIA	Cotton wool spots	Dot blot hemorrhages, cotton wool spots	BE Moderate NPDR	N	N	Y	N	1	1	2	8	8	9	9	REDUCED	REDUCED
D200	56	F	EMPLOYEE	GRADUAL PROGRESSIVE	1 YEAR	Y	5 MONTHS	Y	6\24	6\18	GRADE 2 NS	GRADE 2 NS	WNL	WNL	NO RETINOPATHY	N	N	N	N	0	0	0	9	6	7	6	REDUCED	REDUCED