"PREVALENCE OF DRY EYE IN TYPE 2 DIABETES PATIENTS

ATTENDING TERTIARY HOSPITAL IN SOUTH INDIA"

Submitted by

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Dissertation submitted to the

B. L. D. E. U'S SHRI B. M. PATIL MEDICAL COLLEGE AND RESEARCH

CENTER, VIJAYAPUR, KARNATAKA

In partial fulfillment of the requirements for the degree of

MS

IN

OPHTHALMOLOGY

Under the guidance of

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2015

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ACKNOWLEDGEMENT

This piece of work has been accomplished with the grace of almighty God. It gives me immense pleasure to express my heartfelt gratitude to all. I dedicate this page to each and everyone who have helped me to explore the expanses of knowledge.

I express my profound gratitude and sincere thanks to my guide, **Dr. M.H. PATIL** $_{M.S.}$, Professor, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Vijayapur, for his constant and unfailing support, professional insight, valuable suggestions, motivation and exemplary guidance to carry out and complete this dissertation. I am deeply grateful to him for providing me necessary facilities and excellent supervision to complete this work.

I offer my sincere thanks to **Dr. M. S.Biradar_{M.D.}**, Principal, and B.L.D.E.U's Shri. B.M.Patil Medical College, Vijayapur, for his support and inspiration.

I am deeply grateful to **Dr.Vijaykumar Kalyanappagol**, Medical Superintendent for providing me necessary facilities in the hospital premises.

I am deeply indebted and grateful to my Professor & Head, **Dr. Vallabha K**_{M.S.,D.OM.S.}, and Professor **Dr. Sunil. G Biradar**_{M.S.}, Department of Ophthalmology, B.L.D.E.U's Shri B.M.Patil Medical College, Vijayapur, who with their valuable suggestions and constant guidance supported me throughout the preparation of this dissertation work.

I express my heartfelt thanks to **Dr. Shadakshari S. Math_{M.S.}**, Associate Professor and **Dr. Raghavendra K. Ijeri_{M.S.}**, Assistant professor, and **Dr Jyoti R. C.**_{D.O.M.S} Department of Ophthalmology, B.L.D.E.U's Shri. B.M.Patil Medical College, Vijayapur, for their valuable suggestions and encouragement which have definitely helped me improve my research work.

I acknowledge my gratitude to my Colleagues **Dr. Harsha Nadgir** and **Dr. Gautam Beladiya**, Postgraduate Students, Department of Ophthalmology, B.L.D.E.U's ShriB. M. Patil Medical College, Vijayapur, for their advice and encouragement I extend my thanks to my seniors **Dr Sushma Hosmani**, **Dr Akshata D**, **Dr. Shravan**, **Dr. Darshan and Dr. Shilpa**, my junior **Dr. Vijay**, for their support and help in data collection. I also thank all friends in the department for their cooperation during the preparation of this dissertation.

I thank **Mr. Shahnawaz**, Statistician for his masterly guidance and statistical analysis. I sincerely acknowledge the support and kindness shown towards me by all the staff of Central Library, Shri B. M. Patil Medical College, Vijayapur, at all times.

I am thankful to all the Technical and non-teaching Staff of the Department of Ophthalmology, B.L.D.E.U's Shri B.M.Patil Medical College, Vijayapur for their cooperation.

I am grateful to **Preeti Net Zone**, Vijayapur for their timely and fantastic printing work.

My deepest sense of gratitude, to my parents Mr Sharanappa S Warad and Mrs. Smt Geeta S Warad,, my husband Dr Shivkumar Mutnal and my family members Mr Premnath Shirakoli, Mrs Smita Shorakoli and Sachi whose cherished blessings and countless sacrifices are behind whatever success I have achieved in my life.

Last but not the least, my sincere thanks to all the participants of this study for their cooperation without which this study would not have been possible.

Date: Place: Vijayapur **Dr. NEETA WARAD**

LIST OF ABBEREVIATIONS

ARMD	-	Age related macular degeneration.
CSME	-	Clinically significant macular oedema.
DM	-	Diabetes mellitus
FBS	-	Fasting blood sugar
HbA1c	-	Glycoselated haemoglobin
HIV	-	Human immunodefiency virus
H/O	-	History of
Ig	-	Immunoglobulin
mm	-	Millimeter
μm	-	Micro meter
Ν	-	Number
NIDIDD	_	Non proliferative diabetic retinopathy
NFDK		· · · · · · · · · · · · · · · · · · ·
OD	-	Right eye
OD OS	-	Right eye Left eye.
OD OS OSDI	-	Right eye Left eye. Ocular surface disease index.
OD OS OSDI PDR	-	Right eye Left eye. Ocular surface disease index. Proliferative diabetic retinopathy
OD OS OSDI PDR PPBS	-	Right eye Left eye. Ocular surface disease index. Proliferative diabetic retinopathy Post Prandial blood sugar
OD OS OSDI PDR PPBS QoL	-	Right eye Left eye. Ocular surface disease index. Proliferative diabetic retinopathy Post Prandial blood sugar Quality of life.
OD OS OSDI PDR PPBS QoL RBS	-	Right eye Left eye. Ocular surface disease index. Proliferative diabetic retinopathy Post Prandial blood sugar Quality of life. Random blood sugar
OD OS OSDI PDR PPBS QoL RBS Secs		Right eye Left eye. Ocular surface disease index. Proliferative diabetic retinopathy Post Prandial blood sugar Quality of life. Random blood sugar Seconds
OD OS OSDI PDR PPBS QoL RBS Secs SD	-	Right eye Left eye. Ocular surface disease index. Proliferative diabetic retinopathy Post Prandial blood sugar Quality of life. Random blood sugar Seconds Standard deviation.

ABSTRACT

BACKGROUND-

Dry eye is a very common disorder of tear film resulting from either decreased tear production or increased tear evaporation. It is not a common cause of vision loss, but it is still a serious issue for people who have it. The symptoms become progressively troublesome and exert an increasing burden on the patients as the disease progresses or increases in severity. If not detected early it can lead to sight threatening complications.

Diabetics often complain of dry eye symptoms such as burning sensation, foreign body sensation, heavy lids, redness etc.

Cataract and retinopathy are well known complications of diabetes, recently, problems involving the ocular surface, dry eye in particular have been reported in diabetics.

Diabetes patients suffer variety of complications due to dry eye which include superficial punctate keratopathy, trophic ulceration, and persistant epithelial defect.

Aim and objective of the study-

To find the prevalence of dry eye in type 2 diabetes patients and correlate with severity and duration of diabetes in patients attending BLDEU'S Shri. B. M. Patil Medical College, Hospital & Research Centre.

Materials and methods-

Its a prospective observational cross sectional study, consisting of 251 type 2 diabetes mellitus patients.

After applying inclusion and exclusion criteria all diabetic patients attending ophthalmology OPD of BLDEU'S Shri. B. M. Patil Medical College, Hospital & Research Centre were studied. All patients were given standard dry eye questioner (OSDI scores) and answers were documented. Detailed diabetic history, ocular signs and symptoms were taken.

Patients were subjected for complete ocular examination of anterior and posterior segment.

Tear film function tests included Schirmer's test, TBUT, and Lissamine green staining. On basis of these test results and OSDI scores patients were labelled to have presence of absence of dry eye.

RESULTS

A total of 251 patients included in the study, 130 (41.03%) were found to be positive for dry eye.

Hence prevalence of dry eye in type 2 diabetics in this study was found to be 41.03%.

Of 251 patients included in study 155 were males and 96 were females.

Mean age group of study population was 67.5 years. 33.5% of study population was between age group of 51 to 60 years.

High OSDI scores were found to correlate significantly with prevalence of dry eye p value < 0.05%.

Significant correlation was found between duration of diabetes and dry eye prevalence, with increasing duration of diabetes there was increase in the prevalence of dry eye.

With increased severity of diabetic retinopathy there was increase in prevalence of dry eye. And patients with retinopathy changes had increased prevalence of dry eye when compared to those who did not have retinopathy changes.

Lissamine green stain was found to be very effective alternative to Rose Bengal stain and we were able to detect even pre dry eye cases. Even before the signs and symptoms of dry eye appeared, stain showed the damage to the ocular surface.

There was significant correlation between HbA1c levels and prevalence of dry eye.

CONCLUSION-

In this study prevalence of dry eye was 41.03%.

Dry eye is significantly more common in diabetes patients.

Poor glycemic control correlates with increase prevalence in dry eye in diabetes patients.

The declined tear film function is severe in patients with diabetic retinopathy changes than those without retinopathy changes.

Examination for dry eye should be integral part of the assessment of diabetic eye disease.

Key words- Dry eye, Diabetes, OSDI score, TBUT.

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INTRODUCTION

DEFINITION

The national eye institute / industry workshop of clinical trials in dry eye chaired by Michael. A. Lemp defined dry eye as 'The disorder of tear film due to tear deficiency or excessive tear evaporation which causes damage to the interpalpebral ocular surface and is associated with symptoms of ocular discomfort.^{1,2,3}

The term 'dry eye' can be attributed to the Swedish Ophthalmologist Henrik Sjogren, who described the triad of dry eye, dry mouth, and joint pains in the year 1933.⁴

The term used commonly to denote dry eye in clinical practice is "keratoconjunctivitis sicca". Keratoconjunctivitis sicca, also known as dry eye syndrome, dry eye disease, chronic dry eye disease, or keratitis sicca, refers to disorders of the tear film caused by reduced tear production, poor tear quality or excessive tear evaporation⁵. These disorders are associated with such symptoms of ocular discomfort as irritation, foreign body sensation, or redness and may cause disease of the ocular surface like ocular surface keratinisation, corneal dellen, scarring, vascularisation; microbial or sterile corneal ulceration with possible perforation and severe visual loss⁶.

Dry eye is a chronic, multifactorial condition characterized by disturbances in the tear film and the ocular surface. It can be caused by deficiency of anyone or more of the tear film components, or can be a component of systemic diseases, including Sjogren's syndrome, lupus and Stevens-Johnson syndrome. Additionally, factors such as contact lens wear and adverse environmental exposures such as arid environments, windy conditions or visual tasking can exacerbate the symptoms of dry eye. Also prevalence of dry eye increases with age. It is estimated that nearly 75% of people over 65 will experience dry eyes syndrome.⁵

The World health organisation (WHO) estimates prevalence of Diabetes worldwide currently is 220 million. India is known as capital of diabetes with prevalence of 62.4 million as of 211 stated by International diabetes federation⁷. Recently problems involving the ocular surface, dry eyes in particular have been reported in diabetic patients⁸. Diabetic patients have lower values of tear secretion and lower values of tear break up time test (TBUT) than normal subjects⁹.

Dry eye can lead to varying degree of complications as mentioned above so early diagnosis and treatment of dry eye syndrome in diabetic patients is important¹⁰. Better understanding of the key and important presenting symptoms, the external and systemic factors contributing to dry eye in diabetics and the help in early diagnosis of this chronic condition, with more efficient and effective treatment and long term patient satisfaction.

AIMS AND OBJECTIVES

 To find the prevalence of dry eye in type 2 diabetes patients and correlate with severity and duration of diabetes in patients attending BLDEU'S Shri. B. M. Patil Medical College, Hospital & Research Centre.

REVIEW OF LITERATURE

In study by Seifart u et al in 1994, they compared diabetics [type 1 and 2] and non diabetics with age and sex matched.

A general ophthalmic examination was performed, main points of comparison were subjective complaints, objective findings on conjunctiva and cornea, break up time [BUT], basal secretion, impression cytology of conjunctiva and grade of diabetic retinopathy. They found that 52.8% of diabetics complained of dry eye symptoms, as against 9.3% in controls. Basal secretion test lower than 5mm was observed in 26% of diabetics, pathological conjuctival epithelium was found in 86% of diabetics, a correlation was found between HbA1c values and presence of dry eye syndrome, higher the values higher the incidence of dry eye¹¹.

- In 2000 study by Goebbels M, he did comparative study between insulin dependent diabetics with retinopathy and control group. He did fluorophotometry of tear secretion, the schirmer test, TBUT, and impression cytology of conjunctiva. He found that schiermer readings were 37% lesser than control group, significant more frequent and pronounced signs of conjuctival metaplasia was seen in diabetics. None of the other values differed between groups¹².
- In 2004 a study by Li HY et al, they studied tear film functions of patients with type 2 diabetes and compared with controls.

Dry eye score was calculated with results of schirmer test 1, TBUT, corneal fluorescein staining, tear film lipid layer observation with tear scope.

Dry eye score was found to be higher in diabetics compared to control group, break up time was faster for diabetics group than controls, patients with background diabetic retinopathy and proliferative diabetic retinopathy had higher scores compared to diabetics without retinopathy changes, those patients who underwent photocoagulation therapy had higher scores than patients who had not undergone photocoagulation. There was good correlation between dry eye and diabetic retinopathy changes and photocoagulation¹³.

- In 2005 study by Igor Kaiserman et al, they compared use of lubrication in Diabetes and non diabetic patients. They found that significant higher percentage of diabetes patients received lubricants (20.6%) than non diabetics (13.8%). Use of ocular lubrication increased with poor glycemic control. Keratoconjuctivitis sicca is significantly more common in diabetes patients and poor glycemic control correlates with increased artificial tear use in diabetes patients^{14.}
- A study done in 2008 by Masoud Reza Manaviat. In this study 199 patients of type 2 diabetes were considered. They found that 108 (54.3%) patients suffered from dry eye. There was significant association between dry eye disease and duration of diabetes.

They also correlated dry eye with grades of diabetic retinopathy. There was significant relation between age, sex, duration of diabetes and diabetic retinopathy^{15.}

In 2008 study by Yu L et al, they investigated correlation between diabetes mellitus and tear film function.

Tear film functions performed were TBUT, fluorescein staining, schirmer 1 test, rise Bengal staining, total tear protein detection, tear sodium dodecly sulphate polyacrylamide gel electrophoresis, TMS-4 corneal topography.

They found that in PDR group, BUT and schirmer 1 test values were reduced than in controls and NPDR group. Corneal fluorescein staining scores, positive rate of rose Bengal staining, surface regularity index, surface asymmetry index were also higher in PDR group. Concentrations of lactoferrin and tear specific pre albumin were lower in PDR group.

They concluded that declined tear film function is severe in PDR patients¹⁶.

- In a study by Indu Gupta et al, in 2010 case control study, they included 100 eyes of 50 patients of type 2 diabetes mellitus. And 40 eyes of 20 healthy individuals were included. They performed Schirmers' 1 test, Tear film break up time (TBUT) and conjuctival imprint cytology on each subject in both groups. They foun that mean values of Schirmer 1 test and TBUT were significantly decreased in diabetes patients. The conjuctival imprint cytology revealed pronounced degree of metaplasia with loss of goblet cells in diabetes patients. They also found decreased tear production and unstable tear film in diabetes patient¹⁷.
- In a study by Shoba Pai at el in 2011, it was a comparison study between diabetes patients and normal population to know the changes in tear film function. They took 50 diabetes patients and 50 normal individuals. Their results showed that Tear film break up time was significantly shorter in diabetics, total and basal tear secretions were significantly lower in diabetes patients. This study indicates that patients with diabetes have decreased corneal sensitivity, decreased tear stability and secretions, suggestive of ocular surface disease¹⁸.
- In 2010 study by Gupta N et al in Delhi, they estimated the prevalence of dry eye among Indian patients. 400 subjects were enrolled in the study. They

found that over all prevalence was 29.25% and there was considerable age and gender related variation in this parameter. Subjects more or equal to age of 80 years had higher prevalence (41.2%), Females had more prevalence compared to males 27% v 12%. Grittiness was the commonest complaint reported¹⁹.

- In 2012 study by Basak SK et al, on hospital based population study was a cross sectional study with 3023 subjects. They found that dry eye prevalence was more in females (51.9%) than in men (48.1%). Symptom based dry eye was diagnosed in 1234 subjects (40.8%). Prevalence was dry eye was in 786(26%) subjects. Different grades of Meibomian gland dysfunction was noted in 957 (31.7%) subjects. Primary Sjogren syndrome was noted in 21.5% subjects, 10.9% of dry eye patients had some form of systemic collagen vascular disorder^{20.}
- In 2007 study by James McCulley, et al examined the stain patterns in 22 patients with varying degrees of dry eye and in 11 patients without ocular disease, who served as control subjects. In addition to revealing the progressive pattern, the research also underscored the value of using lissamine green stain instead of the more commonly used fluorescein stain, which does not easily identify damage until it is more progressed. And concluded that a few properly placed drops of lissamine green can reveal staining patterns that are key to diagnosing dry-eye syndrome earlier than possible with other methods²¹.

OCULAR SURFACE

Anatomy: ²²

The bulbar conjunctiva is a thin and translucent structure. It is tied to the subjacent structures by areolar tissue and is mobile to allow ocular movements. The conjunctival structure varies from region to region and also differs with age. The neonatal conjunctiva is pristine.

Conjunctival epithelium consists of different layers. The deepest layer consists of cylindrical cells (as in epidermis), with intermediate layers of polyhedral cells. The most superficial layer is flat but indented. Goblet cells are absent at muco-cutaneous junction , begin to appear and are very numerous beyond the subtarsal folds the fornix to epithelium becomes less glandular, losing its goblet cells, and more epidermal in type, but never keratinised. At the limbus, the epithelium is stratified and papille form giving the deep aspect a charatcterstic sinuous profile.

Goblet cells are most dense nasally, least dense in the upper temporal at the palpebral mucocutaneous junction and limbus. They are chief source of mucin. They arise from the basal layer of epithelium and tend to retain attachment to its basement membrane. They are round to oval in shape, $10-20\mu$ m wide, with flat basal nuclei Cells become larger and more oval as they approach the surface where they develop a stoma and discharge their mucin content. Electron microscopy shows that they are attached by desmosomes to the neighbouring epithelial cells. The density of goblet cells is 10+/-3 cells/mm².

Cytology :²³

Bulbar conjunctiva is composed of stratified columnar epithelium. The cells are round, pyramidal or elongated cylindrical measuring 15-25µm in diameter.

The nucleus is single, round to oval, eccentric, measuring $6-9\mu m$ in diameter . It has smooth regular nuclear membrane and moderately course granular chromatin that is uniformly distributed . Nucleolus is single, small, red and round.

ANATOMY AND PHYSIOLOGY OF LACRIMAL GLAND²⁴

The main lacrimal gland consists of a large orbital part and a small palpebral part which are continuous with each other around the lateral edge of the aponeurosis of levator palpebrae superioris. The larger orbital part is almond shaped and lies in the lacrimal fossa on the anterior and lateral part of the roof of the orbit. The smaller palpebral part (about 1/3 the size of orbital part) lies below the aponeurosis of levator palpebrae superoris and extends into the upper lid.

There are also present in the conjunctival stroma about sixty accessory lacrimal glands which open into the epithelial surface of the conjunctiva. About forty of these are attached to the main lacrimal gland which are known as glands of Krause. Along the superior margin of the tarsal conjunctiva, are located a group of slighty larger glands, the glands of wolfring. A majority of the lacrimal gland fluid enter the fornices superotemporally. From the fornices, lacrimal fluid travels even in the absence of blinking, into the marginal tear strips. The distribution of this fluid into the marginal tear strips to the pre-ocular film depends on the blink²⁵.

Aqueous tears flow out of the ductal openings of the lacrimal glandsand are either hypotonic or isotonic, collect primarily in the forniceal spaces in two strips adjacent to the upper and lower lids and in the thin preocular tear film. The general movement of the tear film is from the outer reaches of the interpalpebral space towards the medial canthus. Most of this flow occurs along the lacrimal rivers and is driven by the muscular action of the orbicularis muscle of the eyelid. Tears reach the superior and inferior puncta through which they are drained into the canaliculi during relaxation.²⁶

NEUROGENIC CONTROL

The lacrimal gland is innervated by both parasympathetic and sympathetic nerve fibers.

It is primarily innervated by the parasympathetic fibers that originate in the cells of the lacrimatory nucleus. The postganglionic secretory nerve fibers pass to the zygomatic nerve, then through a connecting branch they enter the lacrimal nerve. The lacrimal nerve innervates the lacrimal gland.

The sympathetic fibres originate in the hypothalamus relay in superior cervical ganglion and travel in the carotid plexus. They join the deep petrosal nerve, nerve of the pterygoid canal, the maxillary nerve, the zygomatic nerve, the zygomaticotemporal nerve, and finally lacrimal nerve.

Parasympathetic fibers travelling with the lacrimal nerve stimulate lacrimal gland secretion. 1 - adrenergic agonists also stimulate tear secretion. The role of sympathetic nerve fibers to the gland remains somewhat unresolved.

The classic interpretation of the lacrimal secretory system, as first suggested by jones in 1966, has divided the system both anatomically and functionally into two parts

- 1. The basic secretors are composed of goblet cells, accessory lacrimal glands and oil glands.
- 2. The 'reflex' secretors composed of main lacrimal gland. ^{26.27.}

Effect of drugs on tear production -

Certain drugs like anticholinergics, antidepressants, -blockers, and antihistaminics are known to decrease the tear production.²⁸

Bromhexine hydrochloride administered systemically increases tear secretion.

ANATOMY AND PHYSIOLOGY OF THE TEAR FILM ^{25, 29.}

The tear film is a three layered structure composed of lipid, aqueous and from anterior to posterior. It is more appropriate to think that tear film is a two layered structure ; a thin lipid film floating on a large aqueous lake. The mucin layer more appropriately belongs to the corneal and conjunctival epithelium to which it is closely attached. Thickness estimates ranges between 7 and 40 μ m, the range being difficulty in visualisation of tear film for measurement. The film is thickest after a blink, measuring about 9 μ m. The thickness then decreases in a linear manner until at 30 seconds it has decreased to its minimal thickness of 4 μ m.

FUNCTIONS OF TEAR FILM

- 1. Forms and maintains a smooth refracting surface over the cornea.
- 2. Maintains a moist environment for the epithelial cells of the cornea and conjunctiva.
- 3. It has bactericidal properties
- 4. Lubricates the lids.
- 5. It transfers oxygen from air to the cornea
- 6. It dilutes and washes away noxious irritants.

The lipid layer^{29, 30}

The lipid layer was first postulated by wolff and subsequently described by McDonald. This is the most superficial layer of the tear film, 0.1µm in thickness. It is produced primarily by the meibomian glands. These are modified sweat glands

present in the tarsal plate about 30-40 in upper lid and 20-30 in lower lid. They secrete sebaceous material at the mucocutaneous.

Junction of the lid margin. The secretions contain hydrocarbon, wax esters, cholesterol esters, triglycerides.³¹

The lipid layer consists polar and nonpolar lipids. The polar lipids are in contact with the aqueous phase of the tear film and provide structural stability to the tear film, while nonpolar lipids are at the air interface .^{32, 33.}

The melting point of the lipids is $19-32^{\circ}$ C that ensures that it is always fluid on the ocular surface.

Functions of lipid layer

- 1. Prevents spill over of the tears and contains tears within the palpebral opening.
- 2. Inhibits evaporation of tears, especially under the conditions of low humidity and turbulent airflow.
- 3. Prevents damage to lid margin skin by tears.
- 4. The smooth layer of lipid provides an excellent dioptric element for light refraction into the eye and sharp retinal image formation.
- 5. Acts as a hydrophobic barrier and prevents the aqueous layer from getting contaminated with polar lipids that could rupture the tear film prematurely.

Aqueous layer

The aqueous layer is about 6.5μ m in thickness. It comprises about 60% of the tear film. It is secreted by the main lacrimal gland and accessory lacrimal glands of Krause and Wolfring. The layer is an aqueous solution of low viscosity, containing ions of inorganic salts,glucose, urea and various biopolymers such as enzymes,proteins and glycoproteins. Lysozyme, lactoferrin, tear specific prealbumin and secretory Ig A are also present.³¹

A majority of the lacrimal gland fluid enters the fornices superotemporally . from the fornices, lacrimal gland fluid travels, even in the absence of blinking, into the marginal tear strips. The distribution of this fluid in the marginal strips to the preocular film depends on the blink.³⁴

Functions of aqueous layer

- 1. It provides oxygen to the epithelium.
- 2. Washes away debris and noxious irritants.
- 3. It prevents infection due to the presence of antibacterial substances like lysozyme and betalysin.

Mucin layer

The mucin layer is produced by the conjunctival goblet cells. Holly and Lemp estimated that mucus layer is $0.002 - 0.005 \ \mu m$ thick²⁶. But recent studies indicate that it is considerably thicker about $30 \mu m^{22}$. It is made up of glycoprotein's and mucopolysaccharides.²⁷

Goblet cells are interspersed among the stratified squamous epithelial cells of the conjunctiva³⁵. These are distributed singly or in clusters which are identified as mucous crypts³⁰.

Some goblet cells secrete their mucus directly onto the ocular surface and others secrete into the crypts which rise to the ocular surface³⁵.

Kersing has shown that goblet cell densities vary over the ocular surface with the highest density in the inferonasal quadrant. Kersing, in his study found goblet cell densities of 400/sq mm and 1,599/sq mm on the interpalpebral bulbar and inferior palpebral conjunctiva respectively.

Conjunctival goblet cells have typically been identified by alcian blue and periodic acid Schiff stains. These stain the mucus within the secretory granules but not in the remainder of the cell. These do not identify the goblet cells that have Recently secreted mucus.

Functions of the mucin layer -

- 1. The mucin of the glycocalyx renders the whole of the ocular surface hydrophilic and allows even spreading of the aqueous layer over the eye^{36} .
- 2. An adequate layer of mucin masks lipid molecules arriving at the corneal surface and thereby maintains its hydrophilic character.

An estimate of the effectiveness of the mucin layer can be made by measuring the tear film break up time or by performing a goblet cell count.

The composition of tear film is as follows ³¹

Contents	Concentration
Water	98.2
Electrolytes	
Sodium	145mEq/L
Potassium	20mEq/L
Chloride	128mEq/L
Bicarbonate	26mEq/L
Calcium	2.11mEq/L
Magnesium	Trace
Zinc	Trace

METABOLITES

3mg/dl
1-5mmol/L
present
7.20mg/dl
0.6-2gm/100ml
1-2gm/L
trace
14-24mg/100ml
17mg/100ml
250mg/100ml
present
present

ENZYMES

Lysozyme	present
LDH	high levels
Peroxidise	10 ³ U/L

LIPIDS

Cholesterol 200mg%

Glucose: 27

The glucose concentration present in the tear film is too low to satisfy the needs of corneal epithelium. Corneal glucose is obtained from aqueous humour.

PROTEIN COMPONENTS –

These include tear specific prealbumin , -lysin, lactoferrin , lysozyme , immunoglobulins , complement . These lower the surface tension of the tears, thus maintaining a continuous tear film over the cornea.

LYSOZYME

Lysozyme in human tears was first described by Fleming in 1922²⁹. lysozyme(muramidase) destroys bacterial cell membranes . Tear lysozyme levels have been shown to be decreased in keratoconjunctivitis sicca , lupus erythematosus , trachoma and herpes simplex. The lysozyme test is the most sensitive test for the diagnosis of sicca syndrome.

LACTOFERRIN²⁹

This is both a bacteriostatic and bacteriocidal iron binding protein that accounts for upto 25% of human tear proteins .The normal concentration of lactoferrin is 1.4mg/ml.

IMMUNOGLOBULINS AND COMPLEMENT

All the immunoglobulins are present in tears, but only IgA is present in significant quantities about 14-24mg/100ml. Yamamoto and Allansmith showed that the entire complement pathway is present in normal human tears.

The average tear flow rate in humans is about 1.2μ L/minute and ranges from 0.5-2.2 μ L/minute. It is lowest during sleep and highest during emotional stimuli or fall of irritants such as foreign bodies.

In the total 7μ L of tear film, 1μ L is in the preocular tear film within the palpebral fissure, 2.9μ L within the marginal tear film strips and about 4.5μ L within the fornices.

PHYSICAL PROPERTIES OF TEAR FILM ³¹.

- Thickness of tear film average thickness varies from 4-8μm. However recent confocal microscopy has shown that tear film is about 40μm thick.
- Volume of tear film average volume of tear film is 7μl with a range from 4-13μl during basal conditions.
- 3. Rate of tear secretion in non-stimulated subjects the average rate of tear secretion is 1.2μl/min, with a total 24 hour secreting volume of about 10cu ml.
- 4. Turnover rate is 18%/min.
- 5. Refractive index refractive index of tear film is about 1.357.
- 6. pH of tears usual range is from 7.3 to 7.7.
- 7. Osmolarity of the normal human tear film averages 302 ± 6 (SD)mOsm/L.³⁷
- Oxygen tension in the normal tear film under basal conditions, po₂ varies from 40-160mm Hg.

BLINKING AND TEAR FILM STABILITY

Tears are produced normally at a rate of $1-2\mu$ l/minute and an average volume of 5 to 10 μ l is in the conjunctival cul-de-sac ^{38.}

The normal involuntary blink takes about one fourth of a second, and occurs an average once every 5 seconds. However, blink rate does reduce with activities that require concentration such as reading, driving and watching television²⁶.

The human tear film is a constantly changing fluid membrane with flow occurring only in the aqueous layer. The lipid layer remains intact between blinks. The mucin layer remains adherent to the epithelium^{27.}

DYNAMIC EVENTS DURING BLINKING³⁹

Eyelid motion, globe movement, tear distribution and tear drainage are all intimately related in serving the crucial function of maintaining a clean, stable tear film layer over the corneal surface.

Because most normal, non conscious blinks are incomplete, an area of the inferior cornea is not wiped and wetted as frequently as the remainder of the corneal surface.

Incomplete blinks are faster (that is, last shorter time, from start to finish) than complete blinks, because the eyelids do not move that far and the time that the eyelids are stationary is shorter.

It is downward motion of the upper eyelid that wipes the cornea clear of any accumulated debris, and a few blinks will usually carry such material into the lower tear meniscus where it remains when the eyelid rises. Then, the nasally directed horizontal movement of the lower lid during the next few blinks moves the debris toward the medial canthus. Once there, it will exit either via the punctual canaliculi drainage system, or collect in the region of the eyelid junction, where it can be removed by the finger. The horizontal translation of the lower lid appears to play a role in the normal tear turn over process. The descent of the upper lid reaches reaches its maximum speed at about the time that it crossed the visual axis, generally in the range of 17-20cm/sec, but occasionally reaching a speed of over 40cm/sec.

The motion of the lower eyelid is horizontal, in a nasally directed movement, with a total displacement in the range of 2-5mm.

Burton found that the upper eyelid exerts a squeeze force of 50-70g.

Miller reported that average pressure developed during a blink was 10.3mmHgwhich would ensure intimate contact between the inner surface of the upper eyelid and the cornea.
DRAINAGE OF TEARS⁴⁰

Tears flow along the upper and lower marginal strips and enters the upper and lower canaliculi partly by capillary and partly by a reduction of the pressure in the system. About 70% of tears drain through the lower canaliculus and the remaining through the upper.

With each blink, the pretarsal orbicularis oculi compresses the ampullae, shorten the horizontal canaliculi and moves the puncta medially .Simultaneously, the lacrimal part of the orbicularis oculi, which is attached to the fascia of the lacrimal sac, contracts and expands the sac , thereby creating a negative pressure which sucks the tears from the canaliculi into the sac. When the eyes open the muscles relax, the sac collapses and a positive pressure is created which forces the tears down the nasolacrimal duct into the nose .The puncta move laterally, the canaliculi lengthen and fill with tears. Tears drain without the aid of gravity ²².

DRY EYE CLASSIFICATION

National eye institute/industry workshop in 1995 gave the classification of dry eyes as follows.¹





M.A Lemp has divided the causes into:

- 1. Aqueous tear deficiency
- 2. Mucin deficiency
- 3. Lipid abnormalities
- 4. Lid surfacing abnormalities
- 5. Epitheliopathy

By far, the aqueous deficiency is the most common cause of dry eye syndromes.

DRY EYE TYPES

Tear deficient dry eye

Patients with tear deficient dry eye develop ocular surface disease called keratoconjunctivitis sicca.

This group of disorders can be further classified into Sjogren's and Nonsjogren's etiology.

SJOGREN'S SYNDROME TEAR DEFICIENCY

This clinical syndrome named after Henrik Sjogren is characterised by the combination of aqueous tear deficiency with keratoconjunctivitis sicca and dry mouth. At present, two clinical types have been recognised.

Primary - sicca syndrome alone or combined with xerostomia.

Secondary – sicca syndrome associated with connective tissue disorders. Women: men = $9:1^{41}$

The pathogenesis of the tear deficiency is caused by infiltration of the lacrimal gland by B and CD4 lymphocytes and by plasma cells, with subsequent fibrosis. Profound aqueous tear deficiency develops in sjogren's syndrome with significantly lower schirmer's test values, more severe ocular rose bengal and fluorescein staining scores. Patients with secondary Sjogren's syndrome are associated with autoimmune diseases like Rheumatoid arthritis, Systemic lupus erythematosus, progressive systemic sclerosis, hashimoto's thyroiditis, polymyositis, polyarteritis nodosa, and Waldenstrom's macroglobulinemia.

NON-SJOGREN'S SYNDROME TEAR DEFICIENCY

- Congenital alacrimia The most common condition associated with alacrimia is riley-day syndrome, in which there is abnormal parasympathetic innervation of the lacrimal gland. Patients produce a reduced amount of tears while crying. The lacrimal glands are histologically normal.
- Secondary lacrimal gland deficiency ⁴² infiltrative /infectious diseases that replace the secretory lacrimal gland tissue like sarcoidosis, lymphoma, and amyloidosis, may cause dry eye. Dry eye was detected in 21% of a group of patients with AIDS.

Vitamin A deficiency ⁴³

Vitamin A deficiency has been reported to cause dry eye by two different mechanisms .The first mechanism is by causing mucin tear deficiency termed 'xeropthalmia' due to loss of conjunctival goblet cells and other sources of ocular surface mucin.

The second mechanism is by causing decreased aqueous tear production which may be related to systemic protein deficiency.

There is impaired dark adaptation and night blindness. An oval or triangular patch of the temporal part of the conjunctiva has a dry, non wettable xerotic appearance that becomes covered with a foamy substance (Bitot's spots).

Lacrimal gland ablation

Removal of the main lacrimal gland results in persistent unilateral dry eyes^{42.}

3. Neural causes of dry eye^{42} –

Sensory - Heigle and Plugfelder reported that patients with neurotrophic keratitis have significantly reduced tear secretion. After trigeminal ablation, there is decreasesd conjunctival goblet cell density, decreased corneal epithelial collagen and ocular surface changes.

Loss of corneal sensation is a feature of contact lens wear which causes dryness of eyes. A disturbance of lipids in the tear film may be the most frequently encountered contact lens related tear film disturbance. Reduced tear volume and tear flow in a contact lens wearer with a dry eye decreases the movement of a contact lens and results in rapid appearance of dry spots. There is also associated abnormality of the mucin layer with excess mucus debris. Blinking rate is also affected in contact lens wearers.

Motor denervation 42 - damage to the secretomotor fibres of the lacrimal gland secondary to seventh nerve palsy involving the greater superficial petrosal nerve (eg-posterior fosssa tumours) results in dry eyes.

4. Lacrimal obstructive diseases -

 $Trachoma^{42}$ – a cell mediated response which develops in the conjunctiva leads to scarring and occlusion of lacrimal gland ductules , aqueous tear deficiency and keratoconjunctivitis sicca .

Ocular cicatrial pemphigoid – there is fibrotic occlusion of the ducts of lacrimal glands and also reduced conjunctival goblet cell density.

Erythema multiforme and stevens- Johnson syndrome⁴²- stevens-johnson syndrome, also called erythema multiforme major characteristically involves two or

more mucous membranes, including the conjunctiva. After the acute episode, the conjunctival epithelium may develop squamous metaplasia with keratinisation and loss of conjunctival goblet cells.

Chemical burns ⁴² - chemical injuries cause dry eye by various mechanisms like loss of corneal innervation, loss of accessory lacrimal glands, scarring of the ducts and reduced goblet cell density.

EVAPORATIVE DRY EYE

This category of dry eye is characterized by normal lacrimal gland secretory function, but abnormal tear dynamics resulting in increased tear evaporation which can be due to increased palpebral fissure width or meibomian gland dysfunction.

True lipid deficiency is seen in conditions of severe anhidrotic ectodermal dysplasia, a very rare condition in which the meibomian glands are congenitally absent.

Meibomian gland dysfunction can be seen in various types of blepharitis. The bacteria that invade the meibomian glands secrete lipases that hydrolyse the normal lipids to produce various types of free fatty acids which are extremely surfaceactive and are capable of rupturing on contact with an otherwise stable tear film . These free fatty acids may be directly toxic to the corneal epithelium or may damage it via formation of dry spots.

Defective spreading of the tear film(lid surfacing abnormalities)

Tear film evaporation is positively correlated with ocular surface area. Mechanical or neurological disorders resulting in increased palpebral fissure width (in cases of exposure keratitis) may lead to instability of the tear film and dry eye. The most common causes of exposure keratitis include proptosis associated with thyroid ophthalmopathy and facial nerve palsy. Dry eye due to Bell 's palsy can be either due to corneal exposure secondary to inadequate lid closure or absent corneal sensation resulting from exposure hypoesthesia.

Abnormalities of the eyelids such as ectropion, entrpion, symblepharon, large lid notches and keratinised lid margin can interfere with effective spreading of tears across the cornea and cause drying of the ocular surface. Dellen are areas of locally thinned cornea adjacent to limbal or conjunctival elevations. These areas are not resurfaced by the lid are not able to support tear film without the benefit of an adequate hydrophilic mucus layer.

DIAGNOSIS OF DRY EYE⁴²

Symptoms form an important part of assessment of any disease process and dry eye is no exception. Surveys on population based prevalence of dry eye have shown that symptoms are present in 25-35% of people. However, studies have also shown a poor association between the signs and symptoms of dye dyes.⁴⁴

The full spectrum of symptoms include heaviness of the lids, blurring and fluctuating vision, excess ropy mucus, burning, itching, scratchiness, foreign body sensation, photophobia, tearing and pain. The symptom most frequently encountered is foreign body sensation or sandy sensation.

Often the patients volunteer information about their intolerance to drafts and winds, intolerance to air conditioning.

Reading is often difficult for dry eye patients since the blink frequency decreases during tasks requiring concentration. As the blink frequency goes down, the length of the time the eys is left exposed to the atmosphere becomes longer and drying may increase.⁴²

Patients may complain of night time awakening especially in case of blepharities of lagophthalmos. Sleep decreases tear production and compromises the eye with regard to tear flow and produces nocturnal symptoms.

Smoke is universally intolerable to tear-deficient patients since there is particulate bombardment of the ocular surface by the smoke which is actually suspension of solid in air.

History of skin diseases should always be asked which plays an important part in entities like scleroderma, scurvy, the facial rash in lupus, old scars from stevensjohnson syndrome, and acne rosacea.

History of drug intake is asked. Thiazide diuretics, antidepressants, -blockers, anticholinergics, antihistaminics, anti-parnkinsonian drugs, benzodiazepines, antihypertensives are known to cause dry eye.

It is helpful to have a list of standardized questions to ask patients, using defined terms. Numerous studies have been done to find the most common symptom and to formulate a valid questionnaire.

The National Eye Institute visual function questionnaire (NEI VFQ – 25) is one such questionnaire to assess the symptoms of ocular disease. However, it surveys the general ocular health and is not reliable to capture the broad range of symptoms unique to a certain ocular disorder $.^{45}$

The ocular surface disease index questionnaire (OSDI), is a 12 item questionnaire designed to provide a rapid assessment of symptoms of ocular irritation consistent with dry eye disease and their impact on vision related functioning .The questions were generated based on patients comments from several years of clinical studies . Each symptom is given an individual score and the final calculation takes into account the number of questions answered and the cumulative scores . The reliability and validity of this questionnaire has been tested in a sample of 109 dry eye patients where it has been found to have excellent test-retest reliability and validity effectively discriminating.

Between normal, mild to moderate, and severe dry eye disease as defined by both the physician's assessment of severity and a composite disease Severity score⁴⁶. Like it has been with other trials, OSDI too has shown to have moderate co-relation with clinical signs among patients with dry eye disease who have tear deficiency. But, it has demonstrated good sensitivity and specificity in distinguishing between normal subjects and patients with dry eye.

OSDI is scored on a scale of 0 to 100, with higher scores representing greater disability. Due to these reasons, this questionnaire has been employed in the present study.

EXAMINATION

Non-ocular examination

General physical examination is undertaken to note any arthritic changes, facial skin changes, salivary gland enlargement. The mouth is examined for evidence of xerostomia.

Ocular examination

One of the most remarkable features of early dry eye syndrome is that the eye appears to be perfectly normal.

Decreased visual acuity that varies with blinking is one of the first signs encountered^{47.}

The initial slit lamp examination is to be done without any topical anaesthesia or special stains into the eye.

The configuration of the lid margin, its approximation to the ocular surface and the completeness of the voluntary lid closure are noted.

The palpebral fissure width is to be examined which is important in the understanding of dry eye because the tear film evaporation is proportional in part to the ocular surface exposed.

The presence of ectropion , entropion ,trichiasis, lid erythema , telangectasia, poliosis, loss of lashes, colarettes, foamy discharge or inspissated material from meibomian gland are noted. The bulbar and palpebral conjunctiva is examined for dilated conjunctival vessels and tenacious stings of mucus which are common in keratoconjunctivitis sicca. Redundant, thickened and loose superior bulbar conjunctiva is seen in superior limbickeratoconjunctivitis. Conjunctival subepithelial fibrosis , keratinisation, symblepharon, and vascularisation are often seen in cicatrizing disease such as ocular Cicatrial pemphigoid and Stevens-johnson syndrome.

Inferior marginal tear film strip-

The size of the inferior marginal tear strip is an indirect indication of tear film volume. The height of the marginal tear strip is measured between the upper margin of the lower lid and the globe. A normal strip is 1-2mm above the lid margin of the lower lid with a concave anterior surface. The slightly deficient strip less than 1mm above lid margin, an enlarged strip will have a convex surface and more than 2mm in size. The deficient inferior marginal strip appears absent with little evidence of tears in the juncture between the lower lid and the globe. The size of the inferior marginal strip is not an absolute sign of dry eye.

Pre-corneal tear film²⁹–

The pre-corneal tear film should be examined before manipulating the lids with the slit lamp microscope. Mucus particles and debris floating up and down against a background of focal gray epithelial dots may be seen in the interpalpebral area suggestive of keratoconjunctivitis sicca. Meniscus floaters are tiny bits of debris being carried along in the upper and lower tear menisci. They are extremely common in dry eye patients.

Mucus strands present in the pre-corneal tear film are actually strings of lipid contaminated mucus that have rolled up and been pushed into the cul-de-sac by the shearing action of lids. These are common in aqueous deficient states, but can become rather spectacular in the mucin-deficient states.

Cornea³⁸-

The epithelial abnormalities commonly found are dry spots and punctuate epithelial keratopathy which are best seen with the slit-lamp as gray dots localised over the inferior surface of the cornea. Cornea is also examined in the interpalpebral area to detect localised elevations and corneal thickness, which is reduced at the centre in patients with dry eye.Filaments are short(<2mm), discrete, translucent, bulbous strands of mucus intertwined with desquamated cells and cellular debris that dangle from the corneal surface and stain with rose bengal. These are characteristically located on the inferior one-third of the cornea. Blinking produces severe pain because the filaments are firmly attached to the richly innervated epithelium.

CLINICAL DIAGNOSTIC TESTS

Tests to assess tear function can be broadly classified as tests that measure tear secretion, those that measure tear film stability and those that measure clearance.

TESTS OF TEAR SECRETION

1. Schirmer's tests

This test is intended to provide a measure of tear production per unit time.

This was the most common technique for the assessment of tear secretion which was originally described in 1903^{42} .

Schirmer's 1:

This can be done with or without topical anaesthesia which measure only basic and combined basic and reflex (total) secretion respectively.

It is performed by using No.41 Whatman filter paper that is 35mm long and 5mm wide. A notch is present at 5mm from one end and indicates the position of the lid fold that will help hook paper onto the lower lid. It is placed at the junction of the middle and lateral 1/3 over the lower lid. The patient with the eyes open, in a dimly lighted room, looks straight and blinks normally. Both eyes are tested simultaneously. Care should be taken not to touch the cornea. After a full five minutes, the strip is removed and the wetted length is measured from the fold. Normal values without

anaesthesia are >/10mm wetting /5 min.

Basic secretion is measured by anaesthetising the conjunctiva by 4% xylocaine. Normal values with anaesthesia are >/5mm wetting /5 min. The difference in the basic and total secretion gives the amount of reflex secretion.

Pathological values

Borderline dry eye	- <10mm/5min	
Hyposecretive dry eye	-	<5mm/5min

Schirmer's 2⁴²:

This is performed as in schirmer's 1, but after the filter is in place a dry cotton bud is placed in the nares to irritate the nasal mucosa. The rationale for this test is that the ocular surface receptors are fatigued due to the constant stimulation in a dry eye state and therefore the stimulus of the filter paper does not induce a reflex secretion.

Stimulating the nasal mucosa irritates the trigeminal nerve and since this is another afferent stimulus for reflex tear secretion, results in tear production .this test is very uncomfortable for the patient. It involves vigorous stimulation of nasal mucosa. Wetting of the strip in response to this test is reduced in Sjogren's syndrome. Less than 15mm wetting indicates failure of reflex secretion.

Schirmer's 3:

This is seldom performed and involves the use of a strong photic stimulus to produce reflex tearing due to a retinal reflex.

Lucca et al.,⁴² evaluated the sensitivity and specificity of the schirmer's test and found a 25% sensitivity and 90% specifity for this test using history , symptoms and clinical examination .

It forms one of the important diagnostic tests in the evaluation of dry eye syndrome, although a diagnosis of dry eye cannot be made or excluded on the basis of

this test alone.

Hamano et al., developed the phenol red thread test in an attempt to overcome some of the disadvantages of schirmer's tests. 3mm of dye impregnated 15mm cotton thread is placed under the lateral 1/5th of inferior palpebral lid margin. It is allowed to absorb tears for 15seconds. Its colour changes to bright orange from tear contact . asian population show a lessened wet length response .

The Japanese diagnostic criteria uses a cut-off value of 10mm for the phenol red thread test 48 .

TEAR MENISCOMETRY

It measures the characteristics of tear meniscus. Tear meniscus volume is reduced in aqueous deficient dry eye as indicated by reduced height and radius of curvature. Radius of curvature can be measured by slit image photography or by technique of reflective meniscometry to provide non invasive method of diagnosis.

MEASUREMENT OF TEAR FILM STABILITY:

Tear film break up time (TBUT)^{42, 38}

Tear film break up time was originally described in 1969 by Norn. Lemp and Hamill in 1973 popularised the concept. It is a practical method of assessing the stability of pre-ocular tear film.

A fluoroscein strip, moistened slighty with balanced salt solution or similar ocular irrigant is touched against the inferior tarsal conjunctiva and the patient is asked to blink several times to distribute the dye throughout the tear film. The examiner should encourage the patient to stare straight ahead without blinking, while the cornea is observed through the slit-lamp using diffuse illumination with the cobalt blue filter. The time between the last blink and the appearance of the first randomly distributed dry spot in the fluroscein film is noted in seconds.

Normal tear film break up time is greater than or equal to 10seconds. It is susceptible to several variables such as

- **1.** Lid holding and use of topical anaesthesia which reduces the tear break-up time.
- 2. Environmental conditions such as humidity and air flow.

Tear break up time does not vary with age and gender.

The breakup of the tear film should occur in a random pattern so that no one area consistently shows dry spots. An area that consistently breaks up indicates localised corneal surface irregularities. Tear film break up time is reduced in patients with mucin deficiency and in patients who have severe aqueous deficiency. The normal break up time varies from one individual to another and also may vary in the same individual at different times of the day.

Holly suggested that after each blink, the pre-corneal tear film thinssecondary to evaporation and retracts towards fornices. Meanwhile the superficial lipid layer diffuses through the aqueous layer to the mucin surface converting it into a hydrophobic surface. The aqueous film then retracts from the contaminated areas, forming a dry spot. These appear usually within 15-30seconds after a blink at scattered locations on the corneal surface.

In normal individuals, the blinking action of the eyelids which usually occurs before the formation of dry spots is required to reform the layer and hence, the blink interval should be shorter than the break up time.

Non invasive breakup time⁴²

This can be measured using a keratometer and a toposcope which was invented by Tonge in London.

Ocular surface staining

Epithelial damage to the exposed surface of the eye can be demonstrated with vital and supravital stains.

Fluroscein⁴⁸

Fluroscein staining is the standard method used to demonstrate ocular surface damage. This water soluble dye when penetrates the intercellular spaces and stains the ocular surface indicates increased epithelial permeability. This orange dye, which fluoresces green when excited by blue light, is applied to the eyes with a strip wetted with a drop of saline. Excess of the dye is shaken from the strip prior to application. Usually staining is confined to the exposed interpalpebral area of the ocular surface, but in severe dry eye, it may well extend to the upper bulbar conjunctiva.

Fluorescein instillation is very well tolerated and causes minimal irritation. Results are recorded on a corneal diagram. Before damaged cells are revealed by the stain, the precorneal tear film is coloured by the dye and it is possible to examine it for normal variations in uniformity or for the appearance of dark patches when the layer breaks up clinically. Fluorescein differs from rose bengal because it stains areas of epithelial cell loss and not devitalised epithelium⁴⁹.

Lissamine green:

Lissamine Green does not itch and is not painful to use. Rose bengal does itch at positive staining of the eye. Thus Lissamine Green – vital staining can be used for even further indications.

Lissamine Green stains exactly like rose bengal, i.e. devitalized and dead cells as well as mucus. If there is a minimum of difference, Lissamine Green would stain a little more than rose bengal at same concentration.

Method:

The ideal would be 0, 01 ml 1% Lissamine Green dripped from a single-dose ampule in the lower eyelid to achieve the right amount of staining of the outer eye. Eyedrops guarantee a constant concentration of lissamine.

Second best and for practical application it is perhaps easier to use a Lissamine Green strip (Lissaver-Plus), which contains 1.5 mg of dye. One drop unpreserved, sterile Saline solution 0,9% from a single-dose ampule is applied to the strip. Let the drop fall into the fornix inferior, or, as the enclosed directions for use suggest: touch conjunctiva or fornix with the moisturized tip of the strip. The directions recommend to let the patient blink the eye several times after the application and to use one or two drops. I would use just one drop and reduce the blinking to avoid any surplus running down the skin around the eyes and the cheeks. This would also reduce the amount of dye lost through the tear canals (or spray out on spectacles).

The vital staining is read interpalpebrally in a slitlamp with white light. Read it in three parts: Corneal, nasal and temporal conjunctival parts, where you give 1 point for few spots, 2 points for several separate spots and 3 points for conflurative spots. A maximum of 9 points can be reached. The test is pathological at minimum 4 points.



FIGURE NO 1-Lissamine green score.

In addition to revealing the progressive pattern, the research also underscored the value of using lissamine green stain instead of the more commonly used fluorescein stain, which does not easily identify damage until it is more progressed. If the most commonly used stain, which is fluorescein is used, than it is most likely to miss the first two stages of the development of dry eye and, consequently, miss a lot of diagnoses.

Early diagnosis of dry eye is important because more treatment options are available in the beginning stages of the syndrome, further progression of the disease can be prevented, and other conditions signaled by dry eye, such as lupus or rheumatoid arthritis, can be identified sooner using Lissamine green stain.

Rose Bengal stain

Rose Bengal is a water-soluble red aniline dye which is the tetraiodotetrachloro derivative of fluorescein ^{29,38,28}. Fenestra and Tseng ,demonstrated that rose Bengal stains healthy epithelial cells when they are not protected by a healthy layer of mucin . This has the unique property of evaluating the protective status of the preocular tear film.

Rose Bengal does not stain the pre-ocular tear film as does fluorescein. It seems to precipitate at the bottom of the meniscus. It neither penetrates into the corneal stroma nor diffuses into the intercellular spaces of the epithelium like fluoroscein. It stains mucus particles, strands, filaments and plaques more vividly than does fluorescein. It appears that staining depends on loss of cell surface glycoprotein that normally contributes to the glycocalyx and enables the mucous layer to attach to the ocular surface.³⁴

Rose bengal is available as 1% ophthalmic solution and as dye impregnated strips.The strips are used by first applying unpreserved saline to it and then touching the wet strip to the inferior palpebral conjunctiva. The interpretation of rose Bengal staining in dry eye is based on two factors intensity and location.

Van Bijstervald developed a scoring system for rose bengal dye that divides the ocular surface into three zones: nasal bulbar conjunctiva, cornea and temporal bulbar conjunctiva. Each zone is given a score ranging from 0 to 3 with 0 indicating no staining and three indicating confluent staining. Scores for each eye are totalled and in any eye, scores of 4 or greater are taken to indicate a positive test for keratoconjunctivitis sicca.



FIGURE No 2 : Modified Van bijstervald rose bengal grading map

Two false positives can be seen with rose bengal stain. A small amount of stain over the body of pterygium or pinguecula is a common normal finding. Also if a schirmer's test has been performed before the use of a rose bengal stain , the conjunctiva will pick up the dye in that area of contact between the conjunctiva and paper strip . False negative results are seen in mild dry eye syndrome. In this system , normal eyes were accurately distinguished from abnormal eyes with 4% false positive and 5% false negative results.

The classic location for rose bengal staining in aqueous tear deficiency is the interpalpebral conjunctiva which appears in the shape of two triangles whose bases are at the limbus . The staining pattern characteristic of dry eye should involve the exposure zone more than the non-exposure zone.

Patterns of staining in different dry eye conditions

- 1. In keratoconjunctivitis sicca from lacrimal gland dysfunction, the conjunctiva stains more than the cornea. In the early disease, staining is limited to the nasal bulbar conjunctiva within the exposure zone. In moderate disease, there is staining of nasal and temporal bulbar conjunctiva within the exposure zones and the nasal staining is greater than temporal staining. Later in the disease cornea stains inferiorly which then progresses to involve the whole of it.
- 2. Meibomitis and meibomian gland dysfunction initially, there can be either no

rose bengal staining or staining of the inferior or superior bulbar conjunctiva under the eyelids and outside the exposure zones. With more severe inflammation, the staining spreads and affects the cornea within the exposure zone. Cornea stains more when compared to conjunctiva.

The major disadvantage with rose bengal is its irritation. Care should be taken to flush the eyes thoroughly after staining.

LABORATORY TESTS

Tear film osmolarity

Measurement of tear film osmolarity is the gold standard test². It is the most sensitive and specific diagnostic test for dry $eye^{26,31}$.

When 311mOsm/L is used as the upper limit of normal, elevated tear osmolarity is 95% sensitive and 94% specific for dry eye.

Tear lysozyme:

The lysozyme test is the most sensitive test for the diagnosis of sicca syndrome. **Meyer** showed that the tear lysozyme concentration is measured viscometrically was decreased in keratoconjunctivitis sicca. His observation was confirmed by **Regan**. **Thygeson** and **kimura** showed that a decrease in concentration of lysozyme preceded all other symptoms of sicca syndrome.

One of the common methods for measuring tear lysozyme is the method of agar diffusion which was introduced by **Van Bijstervald** in 1969¹.

More recently, a radial immunodiffusion technique has been described 28 .

The main disadvantage of tear lysozyme as a diagnostic test for dry eye is its 'lack of specificity'.

Lactoferrin

This is a protein produced by the lacrimal gland the level of which correlates with tears volume. It has significant antibacterial activity ⁴⁷.

In the early 1980s, **Stutchell** and colleagues measured lactoferrin levels in dry eye patients and normal controls using electrophoresis and reported some interesting facts.

Lactoferrin is measured using lactocard or lactoplate.

Ocular ferning test

The ocular ferning test is based on the fact that conjunctival mucus from a normal eye crystallizes in the form of 'ferns' when placed on a dry glass slide and observed under the microscope.

PSYCHOLOGICAL EFFECTS OF DRY EYE:

Dry eye syndrome is not a common cause of vision loss, but it is still a serious issue for people who have it. The symptoms become progressively troublesome and exert an increasing burden on the patients as the disease progresses .These types of patients have various of health related QoL impairment, can become frustrated with their treatment course, repeatedly visit doctors seeking treatment changes , and may seek alternative treatments leading to significant utilization of medical resources. Studies have shown that these patients are reported to have significant loss of productivity each year, often losing approximately 5 work days and working an average of 208 days with dry eye symptoms.⁵¹

It reduces the functional visual acuity of the patient and also leads to his life long dependence on his doctor. sufferes of dry eye syndrome are more likely to report problems with daily activities, like reading using a computer, driving and watching television, than people without dry eye syndrome. The signs do not manifest till late stage of the disease. If not detected early dry eye can lead to complications which are as follows:

COMPLICATIONS:⁵²

- 1. Sterile stromal ulcers: the corneal melt which occurs is typically an oval, non infiltrated ulcers situated at or just below the visual axis with its longest dimension horizontal. The ulcer tends to progress quickly and then perforate.
- 2. Blepharitis and conjunctivitis : there is an increased incidence of infection due

to loss of normal antibacterial tear substances, lysozyme, lactam due lactoferrin.

- 3. Band keratopathy
- 4. Keratinisation
- 5. Corneal vascularisation.

TREATMENT 53

Ocular therapy for tear deficiency is directed towards the following goals:

- 1. Replacement using tear substitutes
- 2. Decreased tear drainage 3.Decreased tear evaporation
- 3. Improved surfacing by the tear film
- 4. Treatment of underlying disease

Tear substitutes

The goal of using tear substitutes is to increase humidity at the ocular surface and to improve lubrication with subsequent secondary benefits. Artificial tears smooth the corneal surface an effect that contributes to improved vision.

Dry eye tear substitutes contain 97% to 99% water. They also contain various electrolytes designed to maintain the osmolarity of the nascent aqueous tear (approximately 300mOsm/L) and the precorneal tear film (approximately 303 to 310mOsm/L), or to a lower osmolarity without causing discomfort. Mucilages like methylcellulose increases the residence time of artificial tears.

Current therapy of dry eye disease is determined by the severity of the condition. In mild cases, in which there are no signs of damage to the conjunctiva or cornea, may be successfully managed with artificial tears applied upto 4 times per day. In moderate cases, with mild damage to the cornea artificial tears can be used upto 12 times per day. In severe dry eye with features like keratinisation

of conjunctiva, superficial punctuate keratopathy in addition to artificial tears and lubricating ointment, tear conserving therapies are required.

An artificial tear has obvious limitations. Firstly they cannot duplicate the composition of natural tears. Secondly the preservatives in them disrupt the precorneal tear film and damage the epithelial surface, worsening the ocular surface disease. The ideal tear substitute is that which approximates the normal electrolyte composition of the tear film, has low surface tension, is well tolerated , is non-irritating, contains no toxic preservatives, and has a long residence time on the cornea and conjunctiva.

Preservation of tears:

- 1. **Punctual occlusion** helps in reducing the tear film osmolarity, increasing tear volume, and prolonging residence of artificial tears. Temporary occlusion using collagen implants, silicone pulgs . Temporary. ooclusion can be done to ascertain if the punctal blockage will help reduce symptoms and also to rule out excessive tearing due to such blockage. Permanent occlusion is done by using thermal or electric cauterization of puncta or canaliculi or by argon laser photocoagulation of the puctal opening.
- Moisture chambers and room humidifiers the concept behind moisture chambers is to enclose the eye so that evaporative loss of tears is reduced. Moisture chamber spectacles or goggles can be used.

Anti-inflammatory therapy

Anti-inflammatory therapy should be considered for patients with severe keratoconjunctivitis sicca who have intolerable irritation, blurred vision or sight threatening corneal complications.

1. Topical Cyclosporine A (CsA)

Immunosuppressive mechanism of cyclosporine relate to binding of specific nuclear proteins required for initiation of T-cell activation, thus preventing T-cell production of inflammatory cytokines such as IL-2 and thereby disrupting immune mediated processes. Patients treated with cyclosporine A 0.05%, 1%, showed significant improvement in two objective signs of dry eye(corneal fluroscein staining and schirmer value).

2. Topical Corticosteroids

Corticosteroids are potent inhibitors of many inflammatory pathways. Among their multiple biologic activities, they inhibit inflammatory cytokine and chemokine production, and decrease synthesis of matrix metalloproteins , decrease expression of cell adhesion molecule and stimulate lymphocyte apoptosis. They have been reported to improve both signs and symptoms of dry eye in several clinical studies.

Unfortunately the long term side effects of corticosteroid, including cataract and steroid responsive glaucoma, preclude their use for long term treatment of dry eye.

3. Oral tetracycline

Indicated in patients with meibomianitis. The mechanism of tetracycline action could be by inhibition of bacterial lipases. This inhibits the breakdown of meibomian lipids into potentially inflammatory fatty acids. Tetracycline 250mg orally 4 times a day, tapered over 3 months or doxycycline 100mg twice a day for upto 2 months before tapering to a maintenance dose of 100mg a day as long as needed.

4. Hot Compresses

Indicated in patients with meibomian gland dysfunction. A clean washcloth heated with hot water is applied to closed lids for 2 to 10 minutes.

The warm compresses is followed by eyelid massage to express the secretions. Cleaning of the eyelid margins with dilute soaps such as baby shampoo may help in cases with seborrhoea.

Surgical treatment

1. Tarsorrhaphy

It can substantially reduce the exposed surface area of the cornea, thus reducing evaporation of tears. In patients with severe ocular surface disease, particularly persistent epithelial defects, and non-infectious corneal ulcers it can be extremely helpful.

2. Conjunctival transplantation

Cicatrising ocular surface disoreders associated with symblepharon, trichiasis, or cicatrising lagophthalmos can be treated by conjunctival or limbal grafting and mucuous membrane transplantation.

3. Keratoprosthesis

Corneal prosthesis has been employed in the management of severe types cicatricial disease such as ocular cicatricial pemphegoid, stevens-johnson. Syndrome, severe trachoma, and chemical burns in which scarring has been excessive and the prognosis for corneal grafting very poor.

Course of the disease

Dry eye disease is a chronic disease, the treatment of which necessarily involves the patient compliance to a major extent. The patients of dry eye syndrome may remain symptomatic over the years with little progression of the disease.

Patients may go through periods of helplessness and depression due to the chronic nature of the problem which the ophthalmologist must recognise and encourage their patients to continue to pursue their normal activities and living.

MATERIALS AND METHOD

SOURCE OF DATA:

This study was carried out in patients attending or admitted B.L.D.E.U'S Shri

B.M.Patil Medical College, Hospital and Research centre, Vijayapur .

Study duration-

It was a hospital based prospective study from October 2013 to march 2015.

METHOD OF COLLECTION OF DATA:

With the prevalence rate of dry eye in Diabetes as 20.6% 15 , at 95% confidence interval and at \pm 5 margin of error the sample size will be 245.

SAMPLE SIZE:

$$n = (\underline{1.96})^2 \times p \times q$$

$$d^2$$

$$p = Prevalence$$

$$q = 100-p$$

$$d = margin of error.$$

Hence a minimum of 245 cases of type 2 diabetes will be included in the

study.

Statistical analysis:

Data will be analyzed by following methods

- 1. Diagrams
- **2.** Mean \pm SD
- **3.** Statistical tests like't' and X^2 tests.

Inclusion Criteria:

1. Patients diagnosed with type 2 Diabetes Mellitus

Exclusion Criteria:

- History of mucoid or watery discharge suggestive of keratoconjuctivitis, conjunctivitis.
- 2. Impaired eye lid function like Bell's palsy, ectropion
- 3. Contact lens users
- 4. Patients who have undergone ocular surgery in last 6 months
- 5. Patients with systemic infections like HIV, HTLV
- 6. Trachoma
- Patients on treatment with blockers, Anti depressants, Antihistaminics, Diuretics, Systemic retinoids.

Methodology

Study was conducted on 251 patietns who were diagnosed with type 2 DM.

Written informed consent was taken before enrolling the patients in the study. An OSDI questionnaire was administered to all participants to assess the symptoms of dry eye and correlate them with the signs.

A complete slit-lamp examination of the lid margins, tear meniscus, conjunctiva, cornea and tear film was done. Relevant examination of other important ocular structures was done.

Following this, tests to diagnose dry eye were performed. These are tear break up time (TBUT), lissamine green staining, and schirmer's tests. Participants were labelled as having dry eye if atleast two out of these three diagnostic tests were positive. This criteria of two diagnostic tests to diagnose dry eye was adopted to increase the detection rate of dry eye and hence arrive at an accurate prevalence.

DIAGNOSTIC TESTS:

 Schirmer's test: this test was performed before the other tests as it had to be done before instillation of anaesthesia.

Procedure:

It was done using 5×35 mm sterile strips of Whatman No.41 filter paper Patient was made to sit in relatively dark room with fan switched off. Terminal round end of strip was folded at the pre marked area along 90⁰ angle. Touching the paper directly with the finger was avoided in oeder to avoid contamination of skin oils. The patient was then asked to look up, lower lid retracted and test paper inserted in lower cul de sac at the junction of medial $2/3^{rd}$ and lateral $1/3^{rd}$ of lid. Adequate care was taken during the procedure to ensure that paper did not touch cornea to avoid reflex tearing.

The patient was advised to blink normally. At the end of 5 minutes, the strips were removed and the length of filter paper moistened was measured in mm starting from the fold.

Interpretation: Measurements of <10mm were considered to be positive. Readings >/= 10mm were considered as negative.

2. Tear film break up time(TBUT) : The TBUT is the time in seconds between the last blink and the appearance of the dry spot.

Procedure:

The patient was seated at the slit lamp. After instilling a drop of 2% fluorescein into the right eye, the patient was asked to blink a few times and place his head in the slit lamp. Then he/she was asked to look straight ahead without blinking. the tear film was observed by moving the beam of slit lamp from limbus to limbus watching for area of tear film rupture manifested by Black Island with in the green sea

of fluorescein.time elapse between last blink and appearance of first black spot was termed tear film break up time and was noted in seconds.this kind of measurement was taken for three successive blinks and mean of this was taken as final reading.

Interpretation-

Break up time of less than 10 seconds was considered positive, indicative of dry eye.

Greater than or equal to 10 seconds was considered negative.

3. lissamine green staining

Lissamine Green stains exactly like rose bengal, i.e. devitalized and dead cells as well as mucus.

Staining was done last after 15 mins of fluorescien staining.

Procedure

Patient seated on slit lamp comfortable position.

Commercially available lissamine green strips were used, Lissamine Green

strip (Lissaver-Plus), which contains 1,5 mg of dye.

One drop unpreserved, sterile Saline solution 0,9% from a single-dose ampule is applied to the strip.

Drop was allowed to fall into inferior fornix or as the enclosed directions for

use suggested. Conjunctiva or fornix touched with the moisturized tip of the strip.

Patient blinked the eye several times after the application.

The vital staining was read interpalpebrally in a slit lamp with white light.

Interpretation-

Staining was read it in three parts: Corneal, nasal and temporal conjunctival parts, where we gave 1 point for few spots, 2 points for several separate spots and 3 points for conflurative spots. A maximum of 9 points can be reached. The test was pathological at minimum 4 points.

RESULTS

TABLE 1 Characteristics of study population.

characteristics	number
Total no of patients	251
Age group	25-85
	years
Schirmer's test	102
TBUT	96
Lisamine green staining	137
Dry eye present	103
Dry eye abesent	148

Total no of patients examined in the study were 251.

Age group varied between of 25 to 85 years.

With average age of 67.5 years.

Age (Yrs)	Ν	Percent
25-40	16	6.4
41-50	66	26.3
51-60	84	33.5
61-70	60	23.9
>70	25	10
Total	251	100

 TABLE 2- Age distribution of study population.

GRAPH 1 Age distribution of study population in years.



The study population was divided into subgroups and number of patients in each age group was calculated. Youngest patient was aged 25 and oldest patient aged 85 years.

We had maximum no of patients in age group between 51 to 60 years i.e. 84 (33.5%) patients.

Sex	Ν	Percent
Male	155	61.8
Female	96	38.2
Total	251	100

TABLE 3- Sex Distribution of Study Population

GRAPH 2- Sex distribution study population.



Among 251 study population we had 155 [61.8%] male patients and 96 [38.2%] female patients.

Duration of Diabetes (Yrs)	Ν	Percent
0-5	150	59.8
6-10	50	19.9
11-15	37	14.7
>15	14	5.6
Total	251	100

 TABLE 4: Distribution of study population based on duration of diabetes in

years.

GRAPH 3-Duration of diabetes in study population.



Study population was divided based on duration of history of diabetes mellitus, we had more than half of study population with history of diabetes between 0 to 5 years i.e 150 patients [59.8%].
TABLE 5 Distribution of study population on HbA1c levels.

HbA1c	N	Percent
Good	92	36.7
Fair	89	35.5
Bad	70	27.9
Total	251	100

GRAPH 4 - Distribution of study population on HbA1c levels



Study population was distributed based on their HbA1c levels as good between 5.5 to 6.8%, fair between 6.8 to 7.6%, poor above 7.6%.

HbA1c levels indicate glycemic control.

TABLE 6 Distribution of study population on basis of presence or absence of dry

Dry Eye	N	Percent
Negative	148	59
Positive	103	41
Total	251	100

GRAPH 5 – Study population with presence or absence of dry eye.



Study population were diagnosed with dry eye or no based on OSDI scores and testes performed.

103 patients were positive for dry eye, 148 were diagnosed with no Dry eye disease.

Dry Eye	Ν	Percent		
Negative	148	59		
Mild	24	9.6		
Moderate	50	19.9		
Severe	29	11.6		
Total	251	100		

 TABLE 7: Distribution study population based on severity of dry eye.

GRAPH 6- Severity of dry eye among study population.



Among 103 patients positive for dry eye, they were divided into subgroups as mild, moderate , severe based on OSDI scores and tests performed. We have maximum no of patients diagnosed with moderate dry eye 50, i.e. 19.9%

TABLE 8- Distribution of study population based on OSDI scores.

OSDI score	N	Percent
0-33	172	68.5
34-66	51	20.3
67-100	28	11.2
Total	251	100

GRAPH 7 Distribution of study population based on OSDI scores.



Of 103 patients which were positive for dry eye we divided them based on their OSDI scores, 0 was taken as negative, 1- 33 was taken as mild, 34-66 was taken as moderate, 66 to 100 was taken as severe.

H/O SPECTACLE USE	Ν	Percent
No	114	45.4
Yes	137	54.6
Total	251	100

 TABLE 9 – Distribution of study population based on history of spectacle use.

Graph 8 Percentage of study population based on history of spectacle use.



We had more than half of study population who were spectacle users.

TABLE 10 - Distribution of study population based on schirmer's test results.

SCHIRMER'S TEST	Ν	Percent
Negative	149	59.4
Positive	102	40.6
Total	251	100

GRAPH 9- Showing results of Schirmer's test.



Schirmer's test was taken positive when the wetting was less than 10mm of No. 41 Whatman filter paper. We had 102 patients positive for test and all these patients were positive for dry eye.

TBUT	Ν	Percent
Negative	155	61.8
Positive	96	38.2
Total	251	100

TABLE 11 – Distribution of study population on TBUT results.





Tear film break up time was taken positive when it was less than 10 seconds.

We had 96 patients positive for the test and all of them were positive for dry eye.

TABLE 12 – Distribution of study population based on Lissamine green staining

LISSAMINE GREEN STAINING	Ν	Percent		
Negative	114	45.4		
Positive	137	54.6		
Total	251	100		

results.

GRAPH 11– Showing results of Lissamine green stain



Lissamine green staining was taken positive based on staining pattern, when more than 4 spots were noted in interpalpabral area on slit lamp examination, they were labelled as positive for the test.

TABLE 13: Distribution of study population based on diabetic Retinopathy

Retinopathy Changes	N	Percent
Normal	130	51.8
Mild NPDR	30	12
Moderate NPDR	40	15.9
Severe NPDR	30	12
PDR	14	5.6
Others	7	2.8
Total	251	100

Changes

GRAPH-12 Showing distribution of study population based on diabetic

retinopathy changes.



Duration of		HbA1c								
Diabetes	F	air	G	ood		Bad]	otal	n voluo	
(Yrs)	Ν	%	Ν	%	N	N %		%	p value	
0-5	69	75.0	19	55.1	3	45.7	15	59.8		
0-5	07	75.0	77	55.1	2	45.7	0	57.0		
6.10	14	15.2	15	16.0	2	30.0	50	10.0		
0-10	14	13.2	15	10.7	1	30.0	50	17.7		
11-15	8	87	19	21.3	1	14.3	37	14.7	< 0.05	
11-13	0	0.7	17	21.5	0	14.5	51	17.7		
>15	1	1.1	6	6.7	7	10.0	14	5.6		
	92	100.0	89	100.0	7	100.0	25	100.0		
Total)2	100.0	0)	100.0	0	100.0	1	100.0		

TABLE 14 : Correlation between Duration of Diabetes (Yrs) and HbA1c levels





There was slight increase in the HbA1c levels as there was increase in duration of diabetes which signifies the control of diabetes was bad as the duration of diabetes increased.

	Duration of Diabetes (Yrs)										
Dry Eyes	0-5		6-10		11-15		>15		Total		1
	N	%	N	%	N	%	N	%	N	%	p value
Negative	143	95.3	5	10.0	0	0.0	0	0.0	148	59.0	
Positive	7	4.7	45	90.0	37	100.0	14	100.0	103	41.0	< 0.05
Total	150	100.0	50	100.0	37	100.0	14	100.0	251	100.0	

 TABLE 15 : Correlation between Dry Eyes and Duration of Diabetes (Yrs)

GRAPH 14 –Showing correlation between duration of diabetes and dry eye



syndrome.

There was significant increase in drye eye syndrome with increasing duration of diabetes. P value < 0.05

All patients above 11 years duration of diabetes were positive for dry eye disease based on tests performed.

SCHIMEDS	Duration of Diabetes (Yrs)											
SCHIMERS	(-5 6-10		11-15		>15		Total		р		
IESI	Ν	%	N	%	N	%	N	%	Ν	%	value	
Negative	14 3	95.3	6	12.0	0	0.0	0	0.0	14 9	59.4		
Positive	7	4.7	4 4	88.0	3 7	100. 0	1 4	100. 0	10 2	40.6	< 0.05	
Total	15 0	100. 0	5 0	100. 0	3 7	100. 0	1 4	100. 0	25 1	100. 0		

TABLE 16 : Correlation between Schirmer's test and Duration of Diabetes

(Yrs)

GRAPH 15- Showing correlation between Schirmer's test and duration of



diabetes.

With increase in duration of diabetes there was increase in positive schirmer's test.

All patients with duration of diabetes more than 11 years were tested positive for schirmer's test.

		Duration of Diabetes (Yrs)													
TBUT	0-5		6-10		11-15		>15		Total		n valuo				
	Ν	%	Ν	%	Ν	%	N	%	Ν	%	p value				
Negative	143	95.3	12	24.0	0	0.0	0	0.0	155	61.8					
Positive	7	4.7	38	76.0	37	100.0	14	100.0	96	38.2	< 0.05				
Total	150	100.0	50	100.0	37	100.0	14	100.0	251	100.0					

TABLE 17: Correlation between TBUT and Duration of Diabetes (Yrs)

GRAPH 16 – Showing correlation between duration of diabetes and TBUT.



There was significant increase in positive TBUT with increasing duration of diabetes.

All patients with history of diabetes more than 11 years were positive for the test.

TABLE 18: Correlation between Lissamine green staining and Duration of

LISSAMINE	Duration of Diabetes (Yrs)										
GREEN	0-5			6-10		11-15		>15		otal	n valuo
STAINING	N	%	Ν	%	Ν	%	N	%	Ν	%	p value
Negative	112	74.7	2	4.0	0	0.0	0	0.0	114	45.4	
Positive	38	25.3	48	96.0	37	100.0	14	100.0	137	54.6	<0.05
Total	150	100.0	50	100.0	37	100.0	14	100.0	251	100.0	-

Diabetes (Yrs)

GRAPH17 - Showing correlation between Lissamine green staining and duration



of diabetes.

Lissamine green staining was positive for maximum patients with duration of diabetes more than 6 years.

Staining was positive in 34 patients who were negative for other 2 tests and had no signs and symptoms of dry eye.

SCHIMERS		Dry Eyes										
TEST	Ne	gative	Po	ositive]	Fotal	n valua					
	N	%	Ν	%	Ν	%	p value					
Negative	148	100.0	1	1.0	149	59.4						
Positive	0	0.0	102	99.0	102	40.6	< 0.05					
Total	148	100.0	103	100.0	251	100.0						

TABLE19: Correlation between Schirmer's test and presence of Dry Eyes

GRAPH 18- Showing correlation between Schirmer's test and dry eye disease.



Except for 1 patient, all those patients who were positive for Schemer's test were positive for dry eye disease.

Specificity of test was 100%, sensitivity was 99.03%.

Positive predictive value of test was found to be 100%.

		Dry Eyes											
TBUT	Negative		Po	ositive]	Fotal	n voluo						
	N	%	Ν	%	N	%	p value						
Negative	148	100.0	7	6.8	155	61.8							
Positive	0	0.0	96	93.2	96	38.2	< 0.05						
Total	148	100.0	103	100.0	251	100.0							

 TABLE 20 - Correlation between TBUT and presence of Dry Eyes

GRAPH 19 – Showing correlation between dry eye disease and TBUT



There was significant correlation between presence of dry eye and positive TBUT .

Sensitivity of test was found to be 93.20% and specificity was 100%

Positive predictive value of test was found to be 100%.

TABLE21: Correlation between Lissamine green staining and presence of Dry

LISSAMINE		Dry Eyes										
GREEN	Negative		Po	ositive]	Total	n value					
STAINING	N	%	N	%	Ν	%	p vulue					
negative	114	77.0	0	0.0	114	45.4						
positive	34	23.0	103	100.0	137	54.6	< 0.05					
Total	148	100.0	103	100.0	251	100.0						

GRAPH 20 – Showing correlation between Lissamine Green Staining and

presence of Dry Eyes



There was significant correlation between positive staining and presence of dry eye.

Sensitivity of test was found to be 100%, specificity was 77.02%.

Positive predictive value was 75.18%.

TABLE 22- Correlation between absence of dry eye and positive Lissamine

LISSAMINE	No Dry Eyes					
GREEN STAINING	N	%				
positive	34	23.0				
Total	148	100.0				

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21 UUI	Stamme	
0	C	

GRAPH 21 – Showing correlation between Lissamine green staining and dry eye.



There were 34 patients who did not have dry eye disease but were positive for lissamine green staining. This indicates the strength of stain to identify the pre dry eye status and damage to ocular surface before signs and symptoms of dry eye appear.

H/O	Dry Eyes										
SPECTACLE	Ne	gative	Po	ositive	J	Fotal	n value				
USE	N	%	Ν	%	Ν	%	p vulue				
No	84	56.8	30	29.1	114	45.4					
Yes	64	43.2	73	70.9	137	54.6	< 0.05				
Total	148	100.0	103	100.0	251	100.0					

TABLE23 Correlation between H/O Spectacle Use and presence of Dry Eyes

GRAPH 22– Showing correlation between history of spectacle use and presence



of dry eye

There was significant correlation between history of spectacle use and presence of dry eye. P value < 0.05%

Dry		OSDI score												
Eves		0-33		34-66		>66	T	otal	n value					
	N	%	N	%	Ν	%	Ν	%	p varae					
Negative	147	85.5	0	0.0	1	3.6	148	59.0						
Positive	25	14.5	51	100.0	27	96.4	103	41.0	< 0.05					
Total	172	100.0	51	100.0	28	100.0	251	100.0						

TABLE24: Association of Dry Eyes and OSDI score

GRAPH 23– Showing correlation between OSDI scores and dry eye.



As there was increase in OSDI score there was increase in presence of dry eye.

Significant correlation was noted between OSDI scores and presence of dry eye.

		OSDI score												
HbA1c	0-33			34-66		>66		Total						
	Ν	%	Ν	%	N	%	N	%	_ p value					
Fair	82	47.7	10	19.6	0	0.0	92	36.7						
Good	58	33.7	23	45.1	8	28.6	89	35.5						
Bad	32	18.6	18	35.3	20	71.4	70	27.9	<0.05					
Total	172	100.0	51	100.0	28	100.0	251	100.0						

TABLE 25: Association of HbA1c and OSDI score

GRAPH-24 Showing correlation between HbA1c levels and OSDI scores



There was significant increase in presence of dry eye with increasing HbA1c levels.

Hence with poor glycemic control there is increase in the incidence of dry eye.

Duration	OSDI score											
of	0-33		3	4-66		>66	T					
Diabetes (Yrs)	N	%	N	%	N	%	N	%	p value			
0-5	144	83.7	5	9.8	1	3.6	150	59.8				
6-10	25	14.5	20	39.2	5	17.9	50	19.9				
11-15	2	1.2	25	49.0	10	35.7	37	14.7	< 0.05			
>15	1	0.6	1	2.0	12	42.9	14	5.6				
Total	172	100.0	51	100.0	28	100.0	251	100.0				

TABLE26: Correlation between Duration of Diabetes (Yrs) and OSDI score

GRAPH-25 Correlation between OSDI scores and duration of diabetes.



There was significant correlation between duration of diabetes and OSDI scores. P value < 0.05%

OSDI scores were higher with increase in the duration of diabetes.

Duration	Retinopathy Changes														
of Diabetes	Normal		N	Aild PDR	Mo N	derate PDR	Se	evere PDR	F	PDR	0	thers	Т	otal	p
(Yrs)	N	%	N	%	N	%	N	%	N	%	N	%	N	%	value
0-5	116	89.2	9	30.0	8	20.0	10	33.3	1	7.1	6	85.7	150	59.8	
6-10	12	9.2	10	33.3	15	37.5	7	23.3	5	35.7	1	14.3	50	19.9	
11-15	1	0.8	10	33.3	13	32.5	8	26.7	5	35.7	0	0.0	37	14.7	< 0.05
>15	1	0.8	1	3.3	4	10.0	5	16.7	3	21.4	0	0.0	14	5.6	
Total	130	100.0	30	100.0	40	100.0	30	100.0	14	100.0	7	100.0	251	100.0	

TABLE27: Association of Duration of Diabetes (Yrs) and Retinopathy Changes

GRAPH 26- Showing association of duration of diabetes and retinopathy

changes.



With increasing duration of diabetes more of retinopathy changes were noted.

		Retinopathy Changes														
HbA1c	Normal		Mild NPDR		Moderate NPDR		Severe NPDR		PDR		Others		Total		р	
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	value	
Fair	73	56.2	11	36.7	4	10.0	1	3.3	0	0.0	3	42.9	92	36.7		
Good	39	30.0	16	53.3	19	47.5	9	30.0	4	28.6	2	28.6	89	35.5	<0.05	
Bad	18	13.8	3	10.0	17	42.5	20	66.7	10	71.4	2	28.6	70	27.9	<0.05	
Total	130	100.0	30	100.0	40	100.0	30	100.0	14	100.0	7	100.0	251	100.0		

TABLE 28: Association of HbA1c and Retinopathy Changes

GRAPH 27- Showing association between HbA1C Levels And Retinopathy



Changes.

With increasing HbA1c levels there were more retinopathy changes.

Poor glycemic control increases the chances of retinopathy changes.

		Retinopathy Changes														
Dry Eyes	No	rmal	N	Aild PDR	Mo N	derate PDR	Se N	evere PDR	F	DR	0	thers	Т	otal	p value	
	Ν	%	N	%	N	%	N	%	N	%	Ν	%	Ν	%		
Negative	118	90.8	10	33.3	5	12.5	7	23.3	2	14.3	6	85.7	148	59.0		
Positive	12	9.2	20	66.7	35	87.5	23	76.7	12	85.7	1	14.3	103	41.0	< 0.05	
Total	130	100.0	30	100.0	40	100.0	30	100.0	14	100.0	7	100.0	251	100.0		

TABLE 29: Association of Dry Eyes and Retinopathy Changes

GRAPH 28- Showing correlation between dry eye and retinopathy changes.



There was significant correlation between presence of dry eye and retinopathy changes.

Incidence of dry was more in patients with retinopathy changes when compared to patients who did not have retinopathy changes.

		Dry Eyes													
Age Groups	Ne	gative	Po	ositive]	Total		Positive							
	N	%	N	%	N	%	p value	and negative Dry eye							
25-40	15	10.1	1	1.0	16	6.4		0.1							
41-50	43	29.1	23	22.3	66	26.3	-	0.5							
51-60	46	31.1	38	36.9	84	33.5	0.004	0.8							
61-70	35	23.6	25	24.3	60	23.9	0.004	0.7							
>70	9	6.1	16	15.5	25	10.0		1.8							
Total	148	100.0	103	100.0	251	100.0									

TABLE 30- Correlation between age and prevalence of dry eye.

GRAPH 29 Showing correlation between age and prevalence of dry eye.







		Dry Eyes													
Sex	Ne	gative	Po	sitive]	Fotal	n voluo								
	Ν	%	Ν	%	Ν	%	p value								
Male	86	58.1	69	67.0	155	61.8									
Female	62	41.9	34	33.0	96	38.2	0.154								
Total	148	100.0	103	100.0	251	100.0									

TABLE 31- Sex wise distribution of dry eye.

GRAPH 31 – Showing sex wise distribution of dry eye



We noticed prevalence of dry eye more in male patients than in female patients.

This observation may be biased due to more number of male patients 61.8% in the study population.

DISCUSSION

Prevelance of dry eye-

In total sample of 251 patients, we found 103 positive for dry eye based on their OSDI scores and three objective tests (schirmer's test, TBUT, lissamine green staining) conducted.

Prevalence Of Dry Eye In This Study Was Found To Be 41.03%.

In a study by Igor Kaiserman et al, it was cohort study they reported incidence of dry eye in type 2 diabetes patients to be 20.6%.¹⁴

Study by Masoud Reza Manviat et al they reported incidence of dry eye to be 54.3% among type 2 diabetes patients.¹⁵

In a study by Goebbels M he reported incidence of dry eye in diabetics patients was 33% as compared to control group.¹²

The vast disparity in dry eye prevalence stems mainly from the different dry eye diagnostic criteria employed and different cut-off values for the objective dry eye tests.

The high prevalence in some studies is also because objective dry eye tests have been performed in patients with positive symptom score (thereby introducing a selection bias)or in patients in rheumatoid arthritis and Sjogren's syndrome, which have proven dry eye components.

Correlation of dry eye with age.

Study population was divided into 5 subgroups based on age.

The prevalence of dry eye was found to significantly increase with increase in age of the patients (p=0.028) and was found to be significantly higher in persons aged more than 60 years. This corresponds to the study by

Moss et al⁵⁸ which showed an association between older age and an increase in dry eye dry symptoms.

In our study also percentage of patients tested positive for dry eye above 60 years was high.

Sex wise distribution of dry eye-

We found a higher prevalence of dry eye in men compared to women.

In a study by Ibtesam Nasimul Hasan et al, they had incidence of dry eye more in men 16.7% compared to women 11.4%.⁵⁹ The prevalence of dry eye has been seen to affect females than males⁶⁰. Also women who used hormone replacement therapy had greater risk 69% of developing dry eye syndrome^{61.}

OSDI scores-

it has been proposed time and again that there is poor correlation between subjective symptoms and objective signs of dry eye. Thus emphasizing the need for objective testing in all patients at risk for developing dry eye. The OSDI scoring system was used in our study as it can classify the dry eye into mild, moderate, and severe varieties. (Table 7)

An OSDI scoring of 67-100 which corresponds to severe dry eye, was found to correlate significantly with objective tests of dry eye, Simpson TL et al ⁵⁴ have found that this scoring system is highly sensitive in differentiating symptomatic and asymptomatic subjects of dry eye.

We also were able to demonstrate that a large number of patients with dry eye do show symptoms and the symptoms correlate with signs and objective tests for dry eye.

Refractive errors and dry eye

We found a significant correlation between the presence of refractive errors and dry eye.

Our findings are consistent with other studies (Sahai et al)⁵⁵ which have shown that prevalence of dry is more in patients with refractive error than emmetropes.

Prevalence of dry eye was higher in those with corrected and uncorrected refractive errors study by Jie et al⁵⁶ has shown that there was significantly higher incidence of dry eye among people with uncorrected refractive errors.

It has been postulated that persons with refractive errors have an increased tendency to rub their eyes which apart from introduction of infective material, sebum and sweat could cause the lodgement of foreign body into the eye that predispose tear film instability. Also people with uncorrected refractive errors have more tendencyto squeeze the eye causing instability of tear film, predisposing to dry eye^{57.}

Tests performed for detection of dry eye

Three diagnostics tests were performed on all patients.

Positivity in 2 tests out of 3 was necessary to label them as having dry eye.this criteria was adopted for diagnosis in order to increase detection rate hence to arrive at an accurate prevalence.

Among all tests lissamine gren staining showed highest sensitivity 100%.

Schirmer's test and TBUT showed 100% specificity. 100% positive predictive value was observed in TBUT and schirmer's test.

100% negative predictive value was observed in lissamine green staining.

Schirmer's test apart from being one of the most frequent tests used in dry

eye clinical practice, other studies have also shown sensitivity and specificity of up to 85% which correspond to the results of our study.

In Goebbels M study diabetics showed lower schirmer test reading compared to control, similar results are observed in our study also⁹.

In another study by Li HY et al¹³ TBUT values were less than 10secs in diabetics when compared to controls. We found similar results in our study too. They also had increase in dry eye score in patients with diabetic retinopathy when compared to diabetics who did not have retinopathy changes. In our study also there was increase in incidence of dry eye in patients with retinopathy changes when compared to those who did not have retinopathy changes.

Dry eye and duration of diabetes.

The prevalence of diabetic microvascular complications is higher in patients with longer duration of diabetes^{62.}

These individuals are at higher risk of developing dry eye syndrome.

Seifart and associates demonstrated that diabetic patients had increased rate of Keraticonjuctivitis Sicca, which may be attributed to decreased corneal sensitivity, neuropathy involving innervations of lacrimal glands and loss of goblet cells.⁶³ similar results were found in current study.

Dry eyes and HbA1c levels.

We noted that there was significant correlation between prevalence of dry eye and HbA1c levels.

Increased HbA1c levels there was increase in the incidence of dry eye.

In study by Seifart U et al they had found a positive correlation between HbA1c levels and dry eye sundrome¹¹.

This indicates poor glycemic control predisposes patients for dry eye disease.

CONCLUSION

- Dry eye is an under-diagnosed ocular disorder. This is because diagnosis and assessment of dry eye are complicated by the considerable variation in disease symptoms and signs and lack of definitive diagnostic tests.
- One should be alert for occurrence of dry eye and its common occurrence in diabetics.
- Age of the patient is an important consideration as dry eye is more common in elderly patients.
- While considering the diagnosis of dry eye, other factors which are to be taken for consideration include gender, presence of refractive error, duration of diabetes, control of diabetes (HbA1c levels), diabetic retinopathy changes and associated systemic diseases like Rheumatoid arthritis as dry eye has positive correlation with these factors.
- Dry eye evaluation with appropriate and standard questionnaire along with standard tests for dry eye helps in diagnosis and treatment.
- A good control of diabetes will prevent the occurrence of dry eye hence prevents its complications .so control of diabetes should be preventive measure.
- This will go long way in effective treatment and management of diabetics with dry eye, especially as the disease is chronic and needs long term treatment and follow up like diabetes.
- Early and appropriate management will provide ocular comfort and satisfaction with a better quality of life.

SUMMARY

- Among 251 patients studied, prevalence of dry eye in type 2 diabetics was as high as 41.03%.
- Prevalence of dry eye increased with increasing age of patient and was significantly higher among patients more than 60 years of age.
- Prevalence was higher among male patients than in females.
- As duration of diabetes increased there was increase in the prevalence of dry eye.
- Prevalence of dry eye was more in patients who had diabetic retinopathy changes when compared to those who did not have retinopathy changes.
- Dry eye prevalence increased in patients with higher HbA1c levels which indicated poor glycemic control.
- There was a good correlation between the objective tests performed and prevalence of dry eye.
- Significant correlation was noted between the OSDI scores and prevalence of dry eye.
- Prevalence of dry eye increased with history of spectacle use.
- Lissamine green stain was positive in few patients who did not have signs and symptoms of dry eye nor they had retinopathy changes, indicating for preventive measures.

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<u>ANNEXURE – I</u>





B.L.D.E. UNIVERSITY'S SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103 INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2013 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance. Title Dreval abafes Babien tertian Hospit BOUGH TH Name of P.G. student × leefa Catava Depart neut 00 Name of Guide/Co-investigator Dr M d8500 De 2 6 0

> DR.TEJASWINI, VALLABHA CHAIRMAN INSTITUTIONAL ETHICAL COMMITTEE BLDEU'S, SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization

1) Copy of Synopsis/Research project.

2) Copy of informed consent form

3) Any other relevant documents.

<u>ANNEXURE – II</u>

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR – 586103, KARNATAKA

FITLE OF THE PROJECT:	PREVALENCE OF DRY EYE IN
	TYPE 2 DIABETES PATIENTS
	ATTENDING TERTIARY HOSPITAL
	IN SOUTH INDIA.
PRINCIPAL INVESTEGATOR:	Dr. NEETA WARAD
	Department of Ophthalmology
	Email: dr.neeta6@gmail.com
PG GUIDE:	Dr. M.H.Patil _{M.S.}
	Professor of Ophthalmology
	B.L.D.E. U's Shri B.M. Patil
	Medical College, Hospital & Research
	Centre, Sholapur Road,
	Vijayapur - 586103

PURPOSE OF RESEARCH:

I have been informed that this study will evaluate the nature and prevalence of dry eye in Type 2 Diabetes patients.

I have been explained about the reason for doing this study and selecting me as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

Relevant clinical history, detailed clinical examination and investigations according to Performa will be done.

RISKS AND DISCOMFORTS

I understand that I have to undergo a complete ocular and systemic examination as required. I may have to undergo the various tests required and to expect a time delay for all the various test reports to come.

BENEFITS:

I understand that my participation in this study will help to know the prevalence of dry eye in Type 2 Diabetes patients and its correlation with severity and duration of Diabetes mellitus.

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr. Neeta Warad is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. And that a copy of this consent form will be given to keep for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Neeta Warad will terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to ______ the

purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr. M.H.Patil

(Guide)

Dr.Neeta Warad

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Neeta Warad has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

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

(Participant)

Date:

(Witness to above signature)

ANNEXURE-III

CASE SHEET PROFORMA

Name:		Date:	
Age:			
Sex:			
Address:			
Occupation:			
H/o Diabetes:	Yes		No
Duration of Diabetes-			
H/o Spectacle use:	Yes		No

If Yes, duration:

Treatment History:

Symptoms of dry eye:

(Ocular Surface Disease Index –OSDI questionnaire)

Have you experienced any of the	All the	Most of	Half the	Sometimes	Never
following during the last week :	time	the time	time		
1. Eyes that are sensitive to					
light					
2. Eyes that feel gritty					
3. Painful or sore eyes					
4. Blurred vision					
5. Poor vision					
Have problems with your eyes	All the	Most of	Half the	Sometimes	Never
limited you in performing any of	time	the time	time		
the following during the last					
week:					
6. Reading					
7. Driving at night					
8. Working with a					
computer/bank					
machine/any near work.					
9. Watching TV					
Have your eyes felt	All the	Most of	Half the	Sometimes	Never
uncomfortable in any of the	time	the time	time		
following situations during the					
last week:					
10. Windy conditions					
11. Places or areas with low					
humidity					
12. Areas that are air					
conditioned					

OSDI= [(sum of scores for all questions answered)x100] / [(total number of questions answered)x4].

Ocular examination:

RIGHT EYE

LEFT EYE

Visual acuity

Pin hole

With glasses

Visual axis

Extra ocular movements

Adnexa:

Eye lids

Eye lashes

Meibomian gland openings

Lacrimal puncta

Tear film meniscus height-

Conjunctiva:

Palpebral

Bulbar

Congestion

Chemosis

Cornea

Sclera

Anterior chamber

Iris

Pupil

Lens

Fundus

Investigations:

Right eye

Left eye

- 1. Schirmer test
- 2. Tear film break up time
- 3. Lessamine green test
- 4. Blood investigations
 - FBS, PPBS / RBS-
 - HbA1C LEVELS-

Diagnosis:

COLOUR PLATES

FIGURE 3 – SCHIRMER TEAR TEST STRIP



FIGURE 4 – Clinical PHOTOGRAPH OF SCHIERMER'S TEST



FIGURE 5 FLUORESCEIN STAINING STRIPS



FIG URE 6 – CLINICAL PHOTOGRAPH OF FLUORESCEIN STAINING (TBUT TEST)



FIGURE 7 LISSAMINE GREEN STAIN STRIPS



FIGURE 8 – CLINICAL PHOTOGRAPH OF LISSAMINE GREEN STAINING ON OCULAR SURFACE



KEY TO MASTER CHART

ARMD	-	Age related macular oedema.
Ac Dac	-	Acute dacryocystitis.
BRAO	-	Branch retinal artey occlusion
CC	-	Cortical cataract
Chr Dacr	-	chronic dacryocystitis
CN	-	Cranial Nerve
CRVO	-	central retinal venous occlusion
CSME	-	clinically significant macular oedema.
HbA1c	-	glycoselated haemoglobin
LE	-	Left eye
Mon	-	months
Mod	-	moderate
NS	-	Nuclear sclerosos (grade 1,2,3,4)
NPDR	-	Non proliferative diabetic retinopathy.
PDR	-	proliferative diabetic retinipathy.
PSC	-	posterior subcapsular cataract
POAG	-	Primary open angle glaucoma.
Pseudo P	-	Pseudophakia
RE	-	right eye
Sev	-	severe
Yrs	-	years

MASTER CHART

SL NO.	NAME	AGE/SEX	IP/OP NO.	Duration of DM	HBAIC	FBS/PPBS		Vision		RETINA	SISCIND FIG			SCHIKMEK'S LEST IN mm		TBUT IN secs		LISSAMINE GREEN STAINING	OSDI SCORE	HYPOGYLCEMICS	DRY EYE	H/O SPECTACLE USE
							RE	LE	RE	ΓE	RIGHT EYE	LEFT EYE	RIGHT EYE	LEFT EYE	RIGHT EYE	LEFT EYE	RIGHT EYE	LEFT EYE				
1	Yallappa	41/M	103948	1 yr	7.4	262/305	6/9	6/9	Normal	Normal	Normal	Normal	35	35	40	42	NEGATIVE	NEGATIVE	4.16	oral	no	yes
2	Chidanand	75/M	17887	15yrs	7.1	178/234	6/9	6/6	PDR	PDR	PDR	PDR	7	8	8	7	positive	positive	27.08	oral	mild	yes
3	Mohanchanda	48/M	134428	18yrs	6	108/150	6/12	6/12	Normal	Normal	Pterygium	Pterygium	8	8	9	9	POSITIVE	POSITIVE	22.91	ORAL+INJ	mild	yes
4	Gangabai	55/F	176678	3yrs	6.6	156/235	6/36	6/36	Normal	Normal	CC	CC	22	24	15	20	positive	positive	3.5	oral	no	yes
5	B B Jadhav	65/M	156049	7yrs	7	141/229	6/6	6/9	Normal	Normal	Normal	Normal	10	12	9	8	positive	positive	56.3	oral	moderate	yes
6	Gurappa Gundadraj	52/M	176303	6yrs	6	112/123	6/6	6/6	Normal	Normal	PSC	Normal	30	28	24	30	NEGATIVE	NEGATIVE	0	oral	no	no
7	Annasaheb	46/M	9382	3yrs	6.8	156/186	6/36	6/36	Normal	Normal	Pterygium + PSC + Mild NPDR	Pterygium + PSC+ Mild NPDR	24	20	40	45	NEGATIVE	NEGATIVE	0	oral	no	yes
8	Vitabai	55/F	9704	5yrs	6.6	266/281	6/24	6/9	MILD NPDR + ARMD	MILD NPDR + ARMD	Mild NPDR	Mild NPDR	5	5	12	10	positive	positive	22.6	oral	mild	yes
9	Bhimanna	75/M	176490	17yrs	7	237/337	6/36	6/12	Mild NPDR	Mild NPDR	Mild NPDR	Mild NPDR	4	4	8	8	positive	positive	49.1	oral	moderate	no
10	S M Shirhatti	55/M	136431	8yrs	7.5	160/300	CF 3	6/9	Mod NPDR	Mod NPDR	Mod NPDR	Mod NDPR	4	5	10	10	positive	positive	19.6	oral	mild	yes
11	Sundrawwa	45/F	132313	бyrs	6	299/388	6/9	6/6	Normal	Normal	Normal	Normal	5	5	10	9	positive	positive	20.5	oral	mild	yes
12	Basanna Naik	58/M	111224	10yrs	6	150/192	6/6	6/6	Normal	Normal	Normal	Normal	15	15	42	42	negative	negative	0	oral	no	no
13	S S Kedachur	70/M	284098	30yrs	7.5	170/310	6/60	6/36	Mod NPDR	Mod NPDR	PSC + Mod NPDR	PSC + Mod NPDR	1	1	4	3	positive	positive	80.2	oral	severe	yes
14	Basayya Hiremath	62/M	294043	5mon	7.8	156/220	6/36	6/60	Mod NPDR	Mod NPDR + tributary artery occlusion	Mod NPDR	Mod NPDR+ tributary arterial occlusion	4	4	9	9	positive	positive	22.7	oral	mild	yes
15	M R Bhavikatti	80/M	291776	4yrs	6	130/198	6/36	6/12	Normal	Normal	PSC	PSC	22	24	42	40	NEGATIVE	NEGATIVE	0	oral	no	yes
16	Mohan Ksharasagar	38/M	192220	2yrs	6	103/163	6/6	6/6	Normal	Normal	CC	CC	28	25	28	34	NEGATIVE	NEGATIVE	0	oral	no	no
17	Kamanna Malagani	61/M	132290	5yrs	6	236/266	6/36	6/36	Normal	Normal	Pterygium	Normal	5	5	9	10	positive	positive	21.9	oral	mild	yes
18	Rayappa Mucchandi	47/M	132242	1 yr	6	110/134	6/6	6/6	Normal	Normal	Normal	Normal	28	25	25	30	NEGATIVE	NEGATIVE	0	oral	no	no
19	Shivani.murti. Hiremath	48/F	107762	2yrs	6.6	82/200	6/6	6/6	Normal	Normal	Normal	Normal	26	30	29	36	negative	negative	0	oral	no	no
20	Uma	62/F	161798	13 yrs	6.4	150/312	CF- 3m	PL	Mod NPDR + CSME	Mod NPDR	Mod NPDR + CSME	Mod NPDR	8	8	9	10	positive	positive	38.6	oral	moderate	no
21	Chayavva	35/F	8688	6yrs	6.6	356/500	6/60	CF- 3m	Mild NPDR	Mild NPDR	Blepharitis+cataract+ mild NPDR	Blepharitis + cataract + mild NPDR	5	5	8	8	positive	positive	22.2	oral	mild	yes

22	Dundavva	55/F	7192	8vrs	8.8	198/309	6/36	CF-	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	4	5	10	10	positive	positive	27.7	oral	mild	ves
23	Umarmath	58/F	81443	12 yrs	6.5	168/200	6/36	3m 6/36	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	6	5	8	10	nositive	nositive	49.1	oral	moderate	
23	Javalaxmi	58/F	81502	3vrs	6.6	101/222	6/60	6/9	Normal	Normal	Normal	Normal	30	28	30	32	negative	negative	0	oral	no	no
25	Gangabai Poti	55/F	139499	10vrs	6.1	152/232	6/60	6/18	Normal	Normal	Normal	Normal	5	5	7	7	positive	positive	39.5	oral	moderate	ves
26	Hulawwa Bhagwati	52/F	139519	10915	6	96/117	6/9	6/9	Normal	Normal	Normal	Normal	35	35	38	35	negative	negative	0	oral	no	no
27	Kusuma Alabal	59/F	139521	3vrs	6.4	180/263	6/12	6/12	Normal	Normal	POAG	POAG	32	31	44	48	negative	negative	0	oral	no	no
28	Sharda Gundanawwa	50/F	139520	4vrs	6	227/340	6/6	6/6	Normal	Normal	Normal	Normal	10	10	10	10	positive	positive	30.1	oral	mild	no
29	Vijavlaxmi Savanth	40/F	139588	10vrs	6	113/237	6/6	6/6	Normal	Normal	Ptervgium+CC	Ptervgium	28	24	24	20	positive	positive	43.8	oral	mild	no
30	Sujatha Mundgal	35/F	139482	2vrs	6.2	223/343	6/6	6/6	Normal	Mild NPDR	Ac Dacr+CC	Stye+CC+Mild	32	30	30	32	positive	positive	0	oral	no	no
												NPDR					1					
31	chandrakala	40/F	139441	4yrs	6.6	209/334	6/6	6/6	Normal	Normal	Normal	normal	15	13	10	10	positive	positive	23.7	oral	mild	no
32	Shardabai	62/F	134970	1 yr	7.8	145/205	6/60	CF- 3m	Normal	Normal	PSC	PSC + NS3	30	30	24	30	negative	negative	0	oral	no	yes
33	Mahadevi Kuwali	60/F	192240	8yrs	7.8	174/324	6/36	6/36	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	4	5	6	8	positive	positive	56.3	oral	moderate	yes
34	Shantabai Gadave	45/F	176293	7yrs	9	136/222	6/6	6/6	Sev NPDR	Sev NPDR	sev NPDR	sev NPDR	2	3	6	5	positive	positive	79.3	oral	severe	no
35	Gangabai Walikar	55/F	176678	3yrs	6.6	125/239	6/6	6/6	Mild NPDR	Mild NPDR	Mild NPDR	Mild NPDR	18	20	15	18	positive	positive	6	oral	no	no
36	Mahadev Hiremath	48/M	20765	6mon	6.1	95/115	6/6	6/6	Normal	Normal	Normal	Normal	26	28	35	38	negative	negative	0	oral	no	no
37	Kantabai	60/F	3085	20yrs	9.7	247/437	6/36	6/12	Sev NPDR	Sev NPDR	CC + sev NPDR	CC+ Sev NPDR	2	2	4	4	positive	positive	80.8	oral	severe	yes
38	Hanumanth Bankalgi	61/M	37324	5yrs	8.8	166/240	6/60	CF- 1.5m	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	4	5	8	6	positive	positive	45.2	oral	moderate	yes
39	Prabhavathi	41/F	7103	2yrs	6.4	164/347	6/6	6/6	Normal	Normal	Normal	Normal	28	32	40	35	negative	negative	0	oral	no	no
40	Mallawwa	54/F	7146	5yrs	7.6	130/190	6/24	6/24	Mod NPDR	Mod NPDR	CC + Mod NPDR	CC+ Mod NPDR	4	4	6	7	positive	positive	59.7	oral	moderate	yes
41	Basavraj Kannur	66/F	2494	15yrs	7.5	121/230	6/12	6/18	Normal	Normal	Stye+Conjunctivitis	Chr Dacr+CC	3	5	6	7	positive	positive	60.5	oral	moderate	yes
42	Jamabai chavan	46/F	347658	1.5yrs	8.5	159/229	6/6	6/6	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	22	21	30	30	negative	negative	0	oral	no	no
43	Chandappa	60/M	330212	5yrs	7	69/89	6/60	cf- 1.5m	Mild NPDR	Mild NPDR	Pseudo P + mild NPDR	Mild NPDR	4	4	9	7	positive	positive	18.5	oral	mild	yes
44	Neelawwa Sarawad	60/F	338940	4yrs	6.9	130/240	6/12	6/12	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	5	5	8	8	positive	positive	20.6	oral	mild	yes
45	Kasturi	62/F	27488	20yrs	9.4	217/335	6/36	CF- 2m	Mod NPDR	Mod NPDR	Mod NPDR	Pterygium+Mod NPDR	1	1	4	3	positive	positive	82.6	oral	severe	yes
46	Mallikarjun Itangihal	45/M	339252	10yrs	8.4	196/268	6/36	6/6	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	3	3	5	5	positive	positive	69.4	oral	severe	yes
47	Rajashekhar	65/M	34682	5yrs	8.8	140/266	6/12	6/12	Normal	Normal	Normal	ARMD	4	4	7	6	positive	positive	40.2	oral	moderate	yes
48	Shivaputrayya Hallur	60/M	325647	1 yr	6.2	116/225	6/60	6/60	Normal	Normal	Normal	Normal	17	18	41	45	negative	negative	0	oral	no	yes
49	Hanumantayya	55/M	3524	5yrs	8.2	233/298	6/60	6/36	Mod NPDR	Mod NPDR	CC+ Mod NPDR	CC+ Mod NPDR	5	5	8	9	positive	positive	49.6	oral	moderate	yes
50	Mallappa Biradar	65/M	3641	4yrs	6.5	160/290	6/12	6/12	Normal	Normal	Mild NPDR	Mild NPDR	17	20	22	26	positive	positive	0	oral	no	yes
51	Parvathi Mendiger	76/F	100049	20yrs	7.8	123/189	CF- 3m	6/36	Mod NPDR	Mod NPDR	CC+ Mod NPDR	CC+ Mod NPDR	2	2	4	4	positive	positive	78.3	oral	severe	yes
52	Lalita	59/F	6914	2yrs	6	113/140	6/24	6/24	CRVO	Normal	CRVO	Normal	24	24	29	30	negative	negative	0	oral	no	yes
53	Gurappa Janabagi	70/M	312549	22yrs	9.2	196/256	PL	6/18	Normal	Sev NPDR	PSC	PseudoP + sev NPDR	1	1	3	4	positive	positive	80.3	oral	severe	yes
54	Basappa Bilagi	55/F	28701	6yrs	8.2	261/296	6/24	6/24	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	5	6	10	9	positive	positive	30.1	oral	moderate	yes
55	Mohan Singh	52/M	8884	6yrs	7.8	254/373	6/36	6/24	Normal	Normal	Normal	Normal	5	5	8	8	positive	positive	59.3	oral	moderate	yes
56	S C Pattar	63/M	103852	5.5yrs	6.4	90/124	6/24	6/9	Normal	Normal	Normal	Normal	32	31	40	40	positive	positive	0	oral	no	yes
57	Yallappa	41/M	103948	1 yr	6	96/126	6/6	6/6	Normal	Normal	Normal	Normal	20	26	27	25	negative	negative	0	oral	no	no
58	B S Biradar	80/M	103793	20yrs	6.3	95/110	6/24	6/24	Normal	Normal	Normal	Normal	1	1	3	4	positive	positive	69.4	oral	severe	yes
59	Nagappa	45/M	7958	9yrs	6.3	156/256	CF3m	CF2m	Mild NPDR	Sev NPDR	Mild NPDR	Sev NPDR	5	4	6	7	positive	positive	48.3	oral	moderate	yes

60	Sugandha Jadhav	60/F	329227	2yrs	6	120/136	6/6	6/9	Normal	Normal	Normal	Normal	21	26	32	30	negative	negative	0	oral	no	no
61	Shama Manguli	65/F	30673	4yrs	6	190/230	6/18	6/18	Normal	Normal	Xanthelesma	Xanthelesma	22	26	30	26	positive	positive	0	oral	no	yes
62	Neelamma Patil	66/F	330005	4yrs	6.3	214/355	6/24	6/12	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	21	23	21	26	positive	positive	0	oral	no	yes
63	Parvathi	60/F	24578	2yrs	6	140/248	6/18	6/18	Normal	Normal	Normal	Normal	30	29	30	35	negative	negative	0	oral	no	yes
64	Ramesh	41/M	18259	1 yr	7.8	245/460	6/9	6/18	Normal	Normal	Normal	Normal	21	20	28	24	negative	negative	0	oral	no	yes
65	Shankar Angwadi	56/M	2478	1.5yrs	6.2	149/273	6/12	6/12	Normal	Normal	Normal	Normal	29	27	30	31	negative	negative	0	oral	no	no
66	Basavaraj Angadi	56/M	13726	12yrs	6.2	150/222	6/36	6/24	Normal	Normal	Cortical Cataract	Cortical Cataract	4	5	8	6	positive	positive	48.3	oral	moderate	yes
67	Basanna Borawath	60/M	13656	4yrs	6	97/139	6/36	6/36	Normal	Normal	Blepharitis	Blepharitis	19	18	24	25	positive	positive	0	oral	no	no
68	Bhagiratbai	80/F	18316	бyrs	8.2	130/216	6/60	6/36	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	4	4	9	9	positive	positive	19.4	oral	mild	yes
69	Sujata Kannur	68/F	2496	4yrs	6.1	130/251	6/12	6/18	Normal	Normal	Normal	Normal	21	23	25	22	positive	positive	0	oral	no	yes
70	M S Biradar	70/M	363000	20yrs	6.6	90/142	6/18	6/18	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	2	1	4	4	positive	positive	80.3	oral	severe	no
71	Basavaraj Hanamani	36/M	49199	3yrs	6.1	140/220	6/6	6/6	Normal	Normal	CC	Normal	22	23	45	44	negative	negative	0	oral	no	no
72	Vithal Telli	61/M	35276	5yrs	6.1	150/185	CF- 2m	CF- 3m	Normal	Normal	CC	CC	19	18	25	22	positive	positive	0	oral	no	no
73	Mallikarjun Hapanad	67/M	254366	11yrs	6.1	108/202	6/9	6/9	Mild NPDR	Mild NPDR	Blepharitis + Mild NPDR	Chr Dacr+Mild NPDR	5	4	7	9	positive	positive	47.8	oral	moderate	yes
74	Rudrappa Dodamani	74/M	2637	20yrs	6.2	169/196	6/36	CF- 2m	Normal	Normal	PSC	CC	2	3	5	4	positive	positive	78.4	INJ+ORAL	severe	yes
75	Rafiq	56/M	33563	5yrs	6.2	116/176	6/60	6/36	Normal	Normal	Normal	Normal	23	20	30	30	positive	positive	0	oral	no	yes
76	Yamanappa	65/M	329893	3yrs	6.8	308/423	6/18	6/18	Normal	Normal	Normal	Normal	33	36	28	26	negative	negative	0	oral	no	yes
77	Appasaheb Indi	66/M	330302	1 yr	6.3	150/188	CF- 2m	CF- 2M	Normal	Normal	NS2	NS2	24	27	45	44	negative	negative	0	oral	no	yes
78	Kalappa Badiger	48/M	29756	4yrs	6	166/189	6/9	6/12	Normal	Normal	Normal	Normal	5	5	12	10	positive	positive	23.5	oral	mild	yes
79	Ramesh Arjungi	25/M	29647	New	7	200/330	6/9	6/9	Normal	Normal	Normal	Normal	35	35	35	36	negative	negative	0	oral	no	
80	Paradappa	65/M	29846	4yrs	6.5	180/220	6/60	6/60	Normal	Normal	Xanthelesma +NS	Xanthelesma +NS	23	24	40	40	negative	negative	0	oral	no	no
81	B B Budhihal	51/M	44964	10yrs	8.8	160/220	6/18	6/9	Sev NPDR	Severe NPDR	CC + sev NPDR	CC + sev NPDR	4	5	7	8	positive	positive	45.9	oral	moderate	yes
82	Rabiya Inamdar	55/F	40730	1.5yrs	6	112/180	6/6	6/6	Normal	Normal	Normal	Normal	20	20	28	25	negative	negative	0	oral	no	no
83	Mallangouda	55/M	41024	4yrs	6	113/186	6/6	CF-F	Normal	Normal	Stye	CC	17	16	31	34	negtive	negative	0	oral	no	no
84	M S Patil	55/M	41062	4yrs	6.2	136/180	CF- 3M	CF- 2M	Normal	Normal	CC	CC	7	8	9	9	positive	positive	18.5	oral	mild	no
85	Appanna	80/M	37309	10yrs	8.8	188/218	6/24	6/36	CSME	CSME	CC +CSME	CC + CSME	2	2	4	3	positive	positive		INJ+ORAL	severe	yes
86	G H Matti	62/M	44893	3yrs	6	115/158	6/6	6/6	Normal	Normal	Normal	Normal	16	18	20	20	negative	negative	0	oral	NO	no
87	Appa saheb	60/M	4545	10yrs	7.9	96/170	6/18	6/12	Mod NPDR	Mod NPDR	Pseudo P + Mod NPDR	Pseudo P + Mod NPDR	5	5	7	8	positive	positive	43.7	oral	moderate	yes
88	Basappa Kori	66/M	2252	10yrs	8.6	230/330	6/24	6/24	PDR	PDR	CC+ PDR	CC+ PDR	2	1	5	4	positive	positive	66.7	oral	SEVERE	yes
89	Siddappa	64/M	21358	4yrs	6	137/157	6/12	6/18	Normal	Normal	Chalazion	Chalazion	13	16	21	22	positive	positive	0	oral	NO	yes
90	Basavraj t	47/M	4148	4 months	8.3	137/190	6/6	6/6	Normal	Normal	CC	CC	35	35	40	40	negative	negative	0	oral	NO	no
91	Vishalaxmi Badiger	50/F	4046	14yrs	9	110/210	6/60	6/60	PDR	PDR	PDR	PDR	2	2	5	4	positive	positive	71.4	oral	SEVERE	yes
92	Krishna	50/M	34221	8yrs	9.7	170/224	6/6	6/18	Sev NPDR + CSME	Sev NPDR + CSME	Sev NPDR + CSME	Sev NPDR + CSME	3	5	6	6	positive	positive	56.3	INJ+ORAL	moderate	yes
93	Awarappa	60/M	22118	1 yr	6	123/156	6/6	6/6	Normal	Normal	Normal	Normal	22	19	20	32	negative	negative	0	oral	NO	no
94	Digambabavva	65/F	17061	12yrs	6.9	167/210	6/18	CF- 3m	Mod NPDR	Mod NPDR	Pseudo P+ Mod NPDR	Cortical Cataract+Mod NPDR	4	3	9	8	positive	positive	43.7	oral	moderate	yes
95	Mahadevi	68/F	9581	3yrs	5.9	126/180	6/18	6/18	Normal	Normal	PSC	PSC	23	22	32	32	negative	negative	0	oral	NO	yes
		-		-	-		-								-				-			

96	S Mulimani	48/M	34619	15YRS	8.5	136/196	6/24	6/24	Sev NPDR +CSME	Sev NPDR+ CSME	Sev NPDR +CSME	Sev NPDR+ CSME	2	2	5	5	positive	positive	69.3	oral	SEVERE	no
97	Savithri	37/F	24733	6yrs	7.6	126/172	6/12	6/12	PDR	PDR	PDR	PDR	5	5	8	9	positive	positive		oral	MILD	yes
98	Shivanand	58/M	19790	13yrs	8.2	260/310	6/12	6/24	PDR +CSME	PDR	Pseudo P+ PDR +CSME	Pseudo P+ PDR	2	1	4	5	positive	positive	76.4	oral	SEVERE	yes
99	Anand	47/M	5037	2yrs	6.8	160/250	6/36	6/36	Normal	Normal	Normal	CC	23	24	22	19	negative	negative	0	oral	NO	yes
100	Sushadevi	60/M	20537	2yrs	7.2	110/168	6/24	6/24	Myopic fundus	Myopic fundus	CCl + NSII	CC +NSII	15	16	30	30	negative	negative	0	oral	NO	yes
101	GURULINGAMMA	80/f	230761	1.5yrs	7	116/156	6/9	6/18	Normal	Normal	Act Dacr	Cor Ulcer	18	17	27	24	negative	negative	0	oral	NO	yes
102	M B Patted	74/M	36258	12yrs	6.9	121/143	6/60	6/36	Mild NPDR	Mild NPDR	Mod NPDR	Mod NPDR	3	4	8	8	POsitive	positive	49.2	oral	moderate	yes
103	Shashikala Bhustell	35/F	26970	2yrs	6	122/180	6/6	6/6	Normal	Normal	CC	Normal	28	30	33	34	negative	negative	0	oral	NO	no
104	Sanabai Pathad	70/F	268812	2yrs	6.3	101//150	6/60	6/60	Normal	Choroidal atrophy	Normal	Choroidal atrophy	20	22	39	36	negative	negative	0	oral	NO	no
105	Sidangouda	50/F	270108	5yrs	6	115/170	6/6	6/6	Normal	Normal	Normal	Normal	12	13	19	18	positive	positive	12.4	oral	NO	no
106	Sumashi Gajanar	47/F	238663	11yrs	10.2	80/220	CF- 3m	CF- 3m	PDR	PDR	PDR	PDR	2	2	4	5	positive	positive	72.5	oral	SEVERE	yes
107	Annapurna	60/F	223043	2yrs	7.1	86/200	CF- 2m	cf- 1.5m	Normal	Normal	CC	CC	24	23	21	23	negative	negative	0	oral	NO	no
108	Heeranna	60/M	19817	1.5yrs	6	90/123	6/60	6/60	Normal	Normal	Conjunc+SIMC	Conjunc+SIMC	35	35	30	30	negative	negative	0	oral	NO	no
109	Rajaram	75/M	19290	5yrs	6	104/140	6/6	6/6	Normal	Normal	Normal	Normal	5	6	11	9	positive	positive	12.9	oral	MILD	no
110	Geeta Patil	43/F	215514	New	6.6	183/225	6/6	6/6	Normal	Normal	Normal	Normal	22	24	31	33	negative	negative	0	oral	NO	no
111	Annsuyabai	58/F	215507	4yrs	6.3	124/217	6/9	6/9	Normal	Normal	Normal	Normal	12	15	26	23	positive	positive	6.3	oral	NO	yes
112	Paramanna Puttli	80/M	18547	14yrs	8.9	103/140	6/9	6/9	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	2	4	5	7	positive	positive	57.2	INJ+ORAL	moderate	yes
113	Basamma Ukkali	70/F	215524	6yrs	6.8	126/186	CF- 3M	CF- 3m	Normal	Normal	Normal	Normal	5	5	8	10	positive	positive	27.3	oral	MILD	no
114	Bhimappa Hanchanal	65/M	19563	7yrs	6	116/166	6/60	6/60	Mild NPDR	Mild NPDR	PSC+Mild NPDR	PSC+ Mild NPDR	4	5	10	9	positive	positive	17.4	oral	MILD	no
115	Drakshayani	55/F	215560	2yrs	6.4	145/222	6/12	6/9	Normal	Normal	Normal	Normal	3	3	8	6	negative	negative	0	oral	NO	no
116	Shrinivas Deshpande	83/M	215522	14yrs	6.1	146/286	6/12	6/9	Mod NPDR	Mod NPDR	PSC+Mod NPDR	PSC + Mod NPDR	3	4	7	8	positive	positive	59.3	oral	moderate	yes
117	Shantappa Hippargi	73/M	21549	2yrs	6	95/116	6/24	6/24	Normal	Normal	CC + PSC	CC	28	25	30	27	negative	negative	0	oral	no	yes
118	Siddhangouda	50/M	21573	1 yr	6	96/159	6/6	6/6	Normal	Normal	Normal	Normal	22	22	31	32	negative	negative	0	oral	nO	no
119	S M Biradar	50/M	19855	5yrs	6	108/150	6/6	6/6	Normal	Normal	CRAO	Normal	5	5	9	9	positive	positive	29.5	oral	mild	no
120	Siddlingapathi	45/M	230777	4yrs	6.2	100/156	6/6	6/6	Mild NPDR	Normal	Mild NPDR	Normal	5	5	10	10	positive	positive	13.8	oral	mild	no
121	Lakshmibai	55/F	231130	3yrs	6.2	116/178	6/18	6/12	Normal	Normal	Normal	Normal	10	12	12	12	positive	positive	28.1	oral	mild	yes
122	Siddappa	49/M	27842	4yrs	6.6	93/201	CF- 2m	CF- 2M	Normal	Normal	PSC	PSC	5	5	8	10	positive	positive	23.8	oral	mild	no
123	Neermala Patil	51/F	208120	12yrs	6.6	131/208	6/6	6/6	Normal	Normal	Normal	Normal	2	3	6	5	positive	positive	57.6	oral	moderate	no
124	Sharanappa Domanal	71/M	207843	5yrs	8.6	188/227	6/24	6/36	Sev NPDR	Sev NPDR	Pseudo P + sev NPDR	PSC + CC+ Sev NPDR	4	5	9	10	positive	positive	25.8	oral	mild	yes
125	Hazarthi Chowdhari	60/F	18167	New	6.2	90/180	6/9	6/9	Mild NPDR	Mild NPDR	Stye + Mild NPDR	Stye + Mild NPDR	22	22	26	24	negative	negative	0	oral	no	no
126	Sharanappa Hirekurabal	65/M	17692	1 yr	6.9	178/340	CF- 3M	CF- 3m	Normal	Normal	CC	CC	30	32	33	37	negative	negative	0	oral	no	no
127	Shantabai Dalawal	60/F	211153	2yrs	6.2	110/178	6/6	6/6	Normal	Normal	Normal	Normal	25	22	31	32	negative	negative	0	oral	no	no
128	Basavraj Honnamani	47/F	210226	1 yr	6.2	121/205	6/6	6/6	Normal	Normal	Normal	Normal	30	30	40	40	negative	negative	0	oral	no	no
129	Mallikarjun	55/M	17378	New	8.6	230/319	6/36	6/24	Normal	Normal	Normal	POAG	32	29	34	37	negative	negative	0	oral	no	yes
130	Sangappa Gaddi	75/M	19090	8yrs	7.2	230/345	6/36	6/60	Normal	Normal	Normal	Normal	4	4	7	8	positive	positive	48.3	oral	moderate	yes
131	Laxmibai Doddamani	54/F	215487	7yrs	6.2	110/160	CF- 3M	6/24	Normal	CRVO	Normal	CRVO	5	5	9	9	positive	positive	22.04	oral	mild	yes
132	Shatawaj	54/M	215527	0.5yrs	6.6	120/190	6/24	6/12	Normal	Normal	Normal	Normal	22	27	40	45	negative	negative	0	oral	no	yes
133	Shankar Halagami	55/M	215516	12yrs	9.4	171/310	6/6	6/6	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	2	2	3	3	positive	positive	71.7	ORAL+INJ	severe	no
134	Shatawaji Shinde	64/M	215527	20yrs	6.6	165/215	6/24	6/12	sev NPDR	sev NPDR	Sev NPDR	Sev NPDR	2	2	3	4	positive	positive	69.3	oral	severe	yes
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135	Shivappa	55/M	21455	5yrs	7.5	210/346	6/18	6/6	Normal	Normal	Normal	Normal	5	4	5	8	positive	positive	43.2	oral	moderate	yes
136	Jagdeesh	57/M	253484	9.5Yrs	8.8	130/235	6/9	НМ	Normal	Normal	Blepharitis	Blepharitis	1	2	1	2	positive	positive	78.3	oral	severe	no
137	Mallikarjun Manvi	50/M	299298	11 yrs	8.8	115/230	6/18	6/18	Mild NPDR	Mild NPDR	Mild NPDR	Mild NPDR	1	1	2	4	positive	positive	70.3	oral	severe	yes
138	Saydamma	50/F	24468	5yrs	6.2	110/160	6/6	6/6	Normal	Normal	Stye	Stye	5	4	8	10	positive	positive	18.4	oral	mild	no
139	Kamalabai	60/F	299337	11yrs	11.2	120/180	6/9	6/6	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	1	2	3	4	positive	positive	79.5	INJ+ORAL	severe	no
140	Y Y Kabade	68/M	301104	3yrs	8.9	100/148	6/36	6/24	PDR	PDR	PDR	PDR	22	28	38	35	positive	positive	12.7	oral	no	yes
141	Parimala	58/F	296026	2yrs	6	69/113	6/12	6/9	Normal	Normal	CC	CC	24	21	26	32	negative	negative	0	oral	no	yes
142	B G Stavarmath	49/M	306900	4.5Yrs	6	115/196	6/6	6/9	Normal	Normal	Blepharitis	Blepharitis	5	4	9	10	positive	positive	18.9	oral	mild	yes
143	Sonabai Rathod	70/F	306917	1 yr	8	101/136	6/60	6/60	Normal	Normal	Normal	Normal	28	29	33	29	negative	negative	0	oral	no	yes
144	S Y Malinmani	48/M	306900	New	9.6	200/290	6/6	6/6	Sev NPDR	sev NPDR	Stye + Sev NPDR	Chr Dacr+Sev NPDR	19	27	38	34	negative	negative	0	oral	no	no
145	S A Nadagouda	50/M	305403	6yrs	6.1	140/190	6/12	6/12	Normal	Normal	Ulcer	Normal	5	4	9	8	positive	positive	27.5	oral	mild	no
146	Shrisashmanna	65/M	306994	3yrs	6.9	100/156	CF- 3M	CF- 2m	Mild NPDR	Mild NPDR	CC+ Mild NPDR	CC+ Mild NPDR	17	19	38	33	negative	negative	0	oral	no	no
147	Sangamesh	45/M	26507	2yrs	6.4	96/160	6/6	6/6	Normal	Normal	Normal	Xanthelesma	24	27	42	40	negative	negative	0	oral	no	no
148	Madiwalamma	50/F	27219	11yrs	6	100/164	6/36	6/60	Normal	Normal	Normal	Normal	3	4	8	9	positive	positive	47.8	oral	moderate	yes
149	Chandrakant Shinde	50/M	310965	9yrs	8.4	160/260	6/12	6/9	Normal	Normal	Normal	Normal	5	3	7	6	positive	positive	59.4	oral	moderate	yes
150	Rajendra Paranakar	58/M	21339	4yrs	8.3	220/360	6/6	6/6	Normal	Normal	Pterygium	Recurrent Styes	4	4	9	9	positive	positive	30.1	oral	mild	no
151	Siddappa	70/M	22675	4yrs	6.3	103/201	CF- 3M	CF- 2M	Normal	Normal	Normal	Normal	3	5	9	11	positive	positive	22.3	oral	mild	no
152	Geetabai	50/F	313297	1 yr	6.1	110/176	6/24	6/12	Normal	Normal	Pterygium	Perforation	20	30	40	38	negative	negative	0	oral	no	yes
153	Narmada Patil	67/F	313658	7yrs	6.7	116/200	6/9	6/9	Mild NPDR	Normal	Mild NPDR	Normal	3	5	9	10	positive	positive	24.5	oral	mild	yes
154	R B Hadagli	54/F	312004	10yrs	8.2	160/260	6/12	6/12	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	2	4	6	4	positive	positive	38.5	oral	moderate	yes
155	Sumithra	59/F	28453	New	6.8	96/146	6/6	6/6	Normal	Normal	Normal	Xanthelesma	16	16	30	32	negative	negative	0	oral	no	no
156	Gurappa Jambagi	70/M	312549	2yrs	8.8	116/176	PL	6/18	Normal	Sev PDR with CSME	Mature cataract	Sev PDR with CSME	13	16	36	40	negative	negative	0	oral	no	yes
157	Sunanda	50/F	296825	10yrs	7	140/240	6/12	6/12	Normal	Normal	Normal	Normal	4	3	5	8	positive	positive	45.9	oral	moderate	yes
158	Channappa Kamble	48/M	299955	1 yr	8.6	223/289	6/24	6/24	Sev NPDR + CSME	Sev NPDR + CSME	Sev NPDR + CSME	Sev NPDR + CSME	19	22	41	39	negative	negative	0	oral	no	yes
159	Madivalemma	50/F	27219	13yrs	7.4	166/238	6/36	6/60	Normal	Normal	Normal	Stye	4	5	9	10	positive	positive	59.4	oral	moderate	yes
160	Kasturibai	65/F	305407	14 yrs	7.2	136/196	6/24	6/24	Mod NPDR	Mod NPDR	Pseudo P + Mod NPDR	Pseudo P + Mod NPDR	3	4	7	8	positive	positive	65.1	oral	moderate	yes
161	Annaraj	65/M	19404	5yrs	6.9	110/170	CF- 1m	cf- 1.5m	Mild NPDR	Mild NPDR	PSC+NSII+Mild NPDR	PSC+NSII+Mild NPDR	3	3	10	10	positive	positive	16.4	oral	mild	no
162	Vishwanath Patil	41/M	139603	5yrs	7	116/160	6/9	6/9	Normal	Normal	Normal	Normal	5	5	9	7	positive	positive		oral	mild	yes
163	Shantabai Halamani	60/F	139639	бyrs	6.7	183/410	CF- 3M	CF- 3m	Mild NPDR	Mild NPDR	CC+Mild NPDR	Stye+CC+Mild NPDR	5	4	9	9	positive	positive	29.4	oral	mild	no
164	Mallappa Thandikatti	55/M	139470	4yrs	8.6	170/280	6/36	6/18	Normal	Normal	Cortical cataract	Cortical cataract	20	20	34	30	negative	negative	0	oral	no	yes
165	Danakka Biradar	57/F	155196	0.5yrs	6.6	170/204	6/9	6/9	Normal	Normal	Normal	Normal	30	30	42	50	negative	negative	0	oral	no	no
166	Sadashiv	55/M	265431	4yrs	6.6	186/240	6/36	6/36	Normal	Normal	Blepharitis	Blepharitis	17	12	20	22	negative	negative	0	oral	no	yes
167	Ashok	48/M	274335	6yrs	8	120/168	6/9	6/9	Normal	Normal	Normal	Normal	2	4	8	6	positive	positive	60.3	oral	moderate	yes
168	Sangappa Rashdi	53/M	274317	7yrs	8.2	160/266	6/24	CF- 3m	PDR	PDR+CSME	CC+ PDR	CC+PDR+CSME	3	5	7	9	positive	positive	55.9	oral	moderate	yes
169	Sharada	51/F	272777	6yrs	6.6	171/290	6/6	6/6	Mild NPDR	Mild NPDR	Mild NPDR	Mild NPDR	5	4	9	9	positive	positive	41.6	oral	mild	no

170	Shettappa Bandiwadda	50/M	36354	2yrs	6.8	110/156	6/24	6/18	Normal	Normal	Blepharitis	Xanthelesma	19	15	28	25	negative	negative	0	oral	no	yes
171	Prakash Rajaput	37/M	313289	2yrs	7.8	100/146	6/36	6/24	Normal	Normal	Normal	CC	13	17	20	21	negative	negative	0	oral	no	yes
172	Shankargouda Patil	56/M	313763	12yrs	7.6	103/235	CF- 2m	6/9	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	3	4	7	9	positive	positive	57.2	oral	moderate	yes
173	Gurupadappa Kattimani	61/M	313328	1.5yrs	6.3	118/179	6/12	6/12	Normal	Normal	Normal	Normal	15	16	25	23	negative	negative	0	oral	no	yes
174	Bhagirathi Patil	69/F	254120	8yrs	6	106/180	6/24	6/6	Normal	Normal	Stye+CC	PSC	4	5	8	9	positive	positive	23,9	oral	mild	yes
175	Raju Dalawaj	47/M	313290	11yrs	6.9	112/216	6/6	6/6	Mild NPDR	Mild NPDR+CSME	Mild NPDR	Mild NPDR+CSME	4	3	6	8	positive	positive	46.1	oral	moderate	no
176	Paravathi	60/F	28492	7yrs	10.7	200/366	6/6	HM	Mod NPDR	Glow absent	Pseudo P + Mod NPDR	Mature cataract	2	2	5	5	positive	positive	70.3	oral	severe	no
177	Ranganna Vaidya	48/M	313286	1.5yrs	6.4	108/168	6/6	6/6	Normal	Normal	Pterygium	Normal	24	21	27	32	negative	negative	0	oral	no	no
178	Neelkanth Amalikant	66/M	304791	26yrs	7.4	209/253	6/12	6/12	Mod NPDR	Mod NPDR+CSME	PSC+NSII+Mod NPDR	PSC+NSII+Mod NPDR+CSME	2	1	3	4	positive	positive	73.4	oral	severe	yes
179	Siddappa Bagewadi	45/M	17582	New	8.4	190/296	6/6	6/6	Dry ARMD	Dry ARMD	Dry ARMD	Dry ARMD	21	22	36	40	negative	negative		oral	no	no
180	Bangarawwa walikar	80/F	18635	9yrs	6.3	187/260	CF- 3M	CF- 1m	Mild NPDR	Mild NPDR	Pterygium+NS III +Mild NPDR	NS III +Mild NPDR	4	3	6	9	positive	positive	54.8	oral	moderate	yes
181	Hanu Chavan	62/M	18898	4yrs	6.9	180/280	6/6	6/6	Normal	Normal	Xanthelesma	Xanthelesma	19	22	29	27	negative	negative	0	oral	no	no
182	Gangamma Terdal	50/F	223496	4yrs	6.8	140/240	6/24	6/24	Early ARMD	Early ARMD	Early ARMD	Early ARMD	14	12	20	19	positive	positive	6.3	oral	no	no
183	Ramappa Paramappanavar	54/M	223029	4yrs	7	90/175	6/6	6/6	Normal	Normal	Stye	normal	16	18	25	23	positive	positive	5.1	oral	no	no
184	Huvanna Adagi	72/M	234456	1.5yrs	6.6	128/178	6/9	6/9	Normal	Normal	Blepharitis	Normal	20	21	35	34	negative	negative	0	oral	no	no
185	Arjun Vander	58/M	10818	6 yrs	6.4	106/160	6/6	6/6	Normal	Normal	Normal	Normal	5	3	8	8	positive	positive	21.06	oral	mild	no
186	Pandappa Kamble	60/M	7859	9yrs	7.2	118/178	CF- 3M	CF- 3M	Mod NPDR	Mod NPDR	Cortical+NSII + Mod NPDR	Subluxated lens+Mod NPDR	4	4	5	5	positive	positive	54.3	oral	moderate	no
187	G K Biradar	72/M	81444	2yrs	7	102/156	6/6	6/6	Normal	Normal	Normal	Normal	26	22	25	27	negative	negative	0	oral	no	no
188	Vishal Bidari	50/M	43543	5.5yrs	7	114/166	6/6	6/6	Normal	Normal	Normal	Normal	5	5	9	9	positive	positive	22.8	oral	mild	no
189	Hanumant Bankalgi	61/M	37324	4.5Yrs	10.2	166/256	6/60	CF- 1M	Sev NPDR	Sev NPDR	Sev NPDR	CC+PSC + Sev NPDR	20	24	31	30	positive	positive	5.3	oral	no	yes
190	Ravindranath Hajeri	50/M	153274	8yrs	6.4	180/280	6/9	6/6	Normal	Normal	Normal	Normal	4	3	9	9	positive	positive	24.6	oral	mild	no
191	Shankar Hiremath	60/M	153356	6yrs	7.8	260/340	6/24	6/24	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	4	5	7	9	positive	positive	45.1	oral	moderate	yes
192	Rajendra K	51/M	10995	6.5yrs	6.4	96/146	6/6	6/6	Normal	Normal	Normal	Normal	5	5	10	10	positive	positive	22.8	oral	moild	no
193	Ashok Narayanappa	45/M	156767	6mon	6.4	130/200	6/6	6/6	BRAO	Normal	BRAO	Normal	21	23	33	34	negative	negative	0	oral	no	no
194	Shrishail	70/M	9406	11yrs	6.4	92/158	6/60	6/60	Mild NPDR	Mild NPDR	Mild NPDR	Mild NPDR	4	3	9	8	positive	positive	32.6	oral	mild	yes
195	Laxman Hosamani	57/M	36565	1 yr	7.4	145/287	6/60	6/60	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	29	27	39	43	negative	negative	0	oral	no	yes
196	S R Narale	80/M	35566	15yrs	7.4	134/190	6/18	6/24	Sev NPDR	Sev NPDR	Pseudo P +Sev NPDR	Pseudo P + Sev NPDR	1	3	6	6	positive	positive	70.39	oral	severe	yes
197	Arjun Bidari	55/M	34365	15yrs	7.2	140/240	6/6	6/6	Mod NPDR	Mod NPDR	Cruciform cataract+ Mod NPDR	Cruciform cataract+ Mod NPDR	2	3	5	7	positive	positive	69.04	oral	severe	no
198	Mallikarjun Mane	58/M	231211	2yrs	6.4	110/160	6/18	6/18	Normal	Normal	Normal	Normal	24	26	38	40	negative	negative	0	oral	no	yes
199	Chandappa	60/M	274670	5.5yrs	8.8	80/128	6/36	CF- 3M	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	4	5	9	8	positive	positive	25.07	INJ+ORAL	mild	yes
200	N R Jadhav	84/M	300387	25yrs	7	164/250	CF- 3M	6/24	Wet AMD + PDR	Dry ARMD + PDR	Wet ARMD+ PDR	Dry ARMD +PDR	2	1	4	3	positive	positive	78.04	oral	severe	yes
201	Tarabai Rathod	62/F	291778	1 yr	6.8	159/264	6/12	CF- 3m	Normal	Normal	Normal	NSII cataract	30	30	31	34	negative	negative	0	oral	no	yes
202	Shreedevi	28/F	291898	1mon	8.2	139/380	6/6	6/6	Normal	Normal	Normal	Normal	28	27	32	31	negative	negative	0	oral	no	no
203	Vishnu Gaudakar	50/M	26537	3yrs	6.9	116/138	6/6	6/6	Normal	Normal	Normal	Normal	21	25	34	29	negative	negative	0	oral	no	yes
204	Khajisab	65/M	25518	10yrs	7.4	148/261	6/60	CF- 3M	Mod NPDR	Mod NPDR	Mod NPDR	NSII cataract + Mod NPDR	3	4	9	9	positive	positive	30.5	oral	mild	yes
205	Mallawwa	59/M	29176	11yrs	7.2	113/215	6/18	6/9	Sev NPDR	Sev NPDR	PSC + Sev NPDR	PSC+ Sev NPDR	2	3	7	8	positive	positive	60.01	oral	moderate	no
206	Subhas	42/M	22125	3yrs	8	146/296	6/12	6/9	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	4	4	12	10	positive	positive	30.05	oral	mild	yes
207	Mallapa	66/M	23990	6yrs	6.8	118/168	6/24	CF3M	Mod NPDR	Mod NPDR	NS II + Mod NPDR	NS III+ Mod NPDR	5	5	8	9	positive	positive	29.04	oral	mild	yes

208	Gangamma R	50/F	24236	2yrs	8	170/270	6/6	6/6	Normal	Normal	Normal	Normal	22	22	36	31	negative	negative	0	oral	no	no
209	Rajendra	68/M	21339	3yrs	8.2	130/220	CF- 3M	CF- 2M	Normal	Normal	PSC	PSC	5	6	10	10	positive	positive	30.04	oral	mild	no
210	Mahadevi Srinath	50/F	2014315	4yrs	8.2	256/307	6/6	6/6	Mild NPDR	Mild NPDR	Mild NPDR	Mild NPDR	5	4	10	9	positive	positive	28.01	oral	mild	no
211	Mallamma	52/F	9856	2yrs	6.6	96/120	6/6	6/6	Normal	Normal	Normal	Normal	31	33	40	45	negative	negative	0	oral	no	no
212	Siddhamma	72/F	21044	3yrs	6.4	108/239	PL	cf- 1.5m	Normal	Normal	Mature cataract	Intemuscent Cataract	29	31	36	37	negative	negative	0	oral	no	no
213	Devendrappa siddappa	36/M	2014412	2yrs	7.6	250/310	6/36	6/36	Normal	Normal	Normal	CC	23	25	41	43	negative	negative	0	oral	no	no
214	Madevi	49/F	49955	9yrs	7	86/168	6/24	6/24	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	3	3	5	6	positive	positive	56.06	oral	moderate	yes
215	Shantidevi	70/F	50429	3yrs	6.4	156/260	6/12	6/12	Normal	Normal	Normal	Normal	19	24	36	34	negative	negative	0	oral	no	no
216	Yamanappa Toradgi	60/M	2014519	New	7.8	140/256	6/6	6/6	Normal	Normal	Normal	Normal	29	28	29	32	negative	negative	0	oral	no	no
217	Siddhawwa	40/F	1253	1 yr	6.6	94/176	6/12	6/12	Normal	Normal	Normal	Normal	30	32	45	41	negative	negative	0	oral	no	yes
218	Dnyaneshwari	35y/F	7568	3mon	9	94/114	6/6	6/6	Normal	Normal	Normal	Normal	27	29	39	43	negative	negative	0	oral	no	no
219	Kashibai Dashwant	55/F	529	4yrs	7.1	98/110	6/6	6/6	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	5	5	10	10	positive	positive	21.2	oral	mild	no
220	M H Utamal	52/M	13697	5.5yrs	6.8	136/206	6/24	6/24	Normal	Normal	Normal	Normal	5	5	10	9	positive	positive	30.09	oral	mild	yes
221	Basavraj	44/M	12218	9yrs	7.2	210/286	6/24	6/24	Mod NPDR	Mod NPDR	Mod NPDR	Mod NPDR	4	5	10	10	positive	positive	23.6	oral	mild	yes
222	Savithri	48/F	40386	3yrs	7.2	200/290	6/6	6/6	Normal	Normal	Normal	Normal	28	29	45	43	negative	negative	0	oral	no	no
223	Kaushibai Yankappa	66/F	1489	2yrs	6.4	170/229	6/6	6/6	Normal	Normal	Normal	Normal	30	31	34	35	negative	negative	0	oral	no	no
224	Shantabai	75/F	3137	2.5yrs	7	160/198	6/18	6/18	mod	mod	Pseudo P	Pseudo P	21	21	28	34	negative	negative	0	oral	no	yes
225	Chandappa	65/M	46719	5yrs	8.6	220/310	6/24	PL	Sev NPDR	Sev NPDR	Aphakia + Sev NPDR	Mature cataract+Sev NPDR	4	5	9	10	positive	positive	26.7	INJ+ORAL	mild	yes
226	Gundappa Bhimappa	61/M	1323	11 yrs	6.8	120/180	6/60	6/36	Mild NPDR	Mild NPDR	Pseudo P +mild NPDR	Pseudo P + mild NPDR	3	3	4	5	positive	positive	64.5	oral	moderate	yes
227	Parvathibai	60/F	36346	4yrs	6.2	116/176	6/12	6/6	Normal	Normal	Pseudo P	NSII cataract	4	4	10	10	positive	positive	30.4	oral	mild	yes
228	Gurunath Gani	75/M	331338	8yrs	6	126/176	6/36	6/36	Normal	Normal	Blepharitis + Pseudo P	Blepharitis + Pseudo P	4	4	8	9	positive	positive	31.5	oral	mild	yes
229	Shankar	62/M	331449	12 yrs	6.8	114/200	6/24	6/6	Mild NPDR	Mild NPDR	PSC+ Mild NPDR	PSC+ Mild NPDR	3	3	6	7	positive	positive	61.5	oral	moderate	yes
230	Mahadevi Hiremath	48/F	291813	2yrs	6	187/222	6/6	6/6	CRAO	Normal	CRAO	Normal	21	22	34	43	negative	negative	0	oral	no	no
231	Parvathi	65/F	291848	4yrs	8.4	190/320	6/24	6/36	Normal	Normal	CC	CC	5	5	9	9	positive	positive	29.9	oral	mild	no
232	Bagappa	85/M	21880	11yrs	6.9	126/196	6/36	CF- 3M	Sev NPDR	Sev NPDR	Sev NPDR	CC + Sev NPDR	4	3	5	6	positive	positive	36.8	oral	moderate	no
233	Shankar Halamma	50/M	205235	13 yrs	7.4	170/3110	6/6	6/6	Mod NPDr	Mod NPDR	Mod NPDR	Mod NPDR	3	2	8	8	positive	positive	41.9	oral	moderate	no
234	S K Kalli	58/M	18646	11yrs	9.3	180/230	6/9	6/9	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	4	4	7	7	positive	positive	47.8	oral	moderate	yes
235	Madevva S	45/F	207142	3yrs	6	100/146	6/24	6/18	Mild NPDR	Mild NPDR	Mild NPDR	Mild NPDR	4	5	8	9	positive	positive	30.8	oral	mild	yes
236	Ratnabai	60/F	207626	9mon	7.6	170/260	6/60	6/60	Normal	Normal	CC	3rd CN palsy + CC	15	16	38	37	negative	negative	0	oral	no	no
237	Nirmala Desai	48/F	229222	4mon	6.5	100/150	6/6	6/6	Normal	Normal	Normal	Normal	25	23	43	40	negative	negative	0	oral	no	no
238	Ramappa Patil	54/M	192208	14 yrs	6.6	87/185	6/9	6/9	PDR	PDR	PDR	PDR	3	2	2	4	positive	positive	70.4	oral	severe	yes
239	A D Bagli	61/M	192235	14yrs	8.6	313/419	6/36	6/18	PDR	PDR	PSC + PDR	Stye+CC+PDR	2	2	3	4	positive	positive	66.8	oral	severe	yes
240	A M Kunjal	60/M	176286	11yrs	7	148/260	6/12	6/12	Mod NPDR	Mod NPDR	CC+ Mod NPDR	CC+PSC+ Mod NPDR	2	3	6	5	positive	positive	50.7	oral	moderate	yes
241	S R Gurabatti	50/M	176717	1 yr	7.1	100/145	6/9	6/12	Normal	Normal	CC	CC	35	34	40	40	negative	negative	0	oral	no	no
242	Sonabee Rathod	70/F	28625	2yrs	8.6	116/170	CF- 3M	6/24	Normal	Normal	NS II	CC	19	23	25	28	negative	negative	0	oral	no	no
243	Rayappa Patted	47/M	238619	2yrs	8.2	100/136	6/6	6/6	Normal	Normal	Normal	Normal	22	25	39	35	negative	negative	0	oral	no	no
244	Subhas Jammalagi	42/M	22125	5mon	7.4	135/200	6/12	6/9	Normal	Normal	PSC	PSC	30	31	45	46	negative	negative	0	oral	no	yes
245	Sidappamamani	63/M	23975	New	7.6	230/374	6/18	6/9	Normal	Normal	Normal	Blepharitis	22	26	32	34	negative	negative	0	oral	no	yes
246	Mallappa	60/M	23990	бyrs	9.4	186/312	6/24	CF- 1M	Mod NPDR	Mod NPDR	NS III Cataract + Mod NPDR	NS III Cataract + Mod NPDR	4	4	8	8	positive	positive	24.5	oral	mild	no

247	Vijaykumar	48/M	22082	8yrs	9.3	168/222	6/60	6/60	Sev NPDR +CSME	Sev NPDR +CSME	Pseudo P+ Sev NPDR+CSME	Pseudo P+ Sev NPDR+CSME	3	3	8	6	positive	positive	34.2	oral	moderate	yes
248	Sayadam khatum	50/F	24468	12yrs	6.6	110/170	6/60	6/60	Mod NPDR	Mod NPDR	Stye +PSC+Mod NPDR	Stye+PSC+ Mod NPDR	4	4	7	8	positive	positive	45.9	oral	moderate	no
249	Gurudatt Jadhav	62/M	26874	18yrs	7.8	180/300	CF- 2m	6/24	PDR	PDR	NSII Cataract+ PDR	NSII Cataract+ PDR	2	2	5	7	positive	positive	68.7	INJ+ORAL	severe	no
250	Channamallayya Hiremath	40/M	291800	3yrs	6	98/148	6/6	6/6	Normal	Normal	Normal	Normal	30	32	38	40	negative	negative	5.8	oral	no	no
251	Allabaksha Bagali	51/M	238639	11yrs	6.4	118/168	6/6	6/6	Sev NPDR	Sev NPDR	Sev NPDR	Sev NPDR	3	3	6	5	positive	positive	40.6	oral	moderate	no