A Dissertation on

TO STUDY THE PREVALANCE OF DIABETIC RETINOPATHY AND DRY EYE IN DIABETES MELLITUS AND COMPARING WITH DURATION AND UREA ,CREATININE LEVEL

Dissertation submitted in Partial fulfilment of the regulations required for the award of M.S. Degree in Ophthalmology

April 2015

THE TAMIL NADU Dr M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

DECLARATION

I hereby declare that this dissertation entitled“ **To study the prevalence of diabetic retinopathy and dry eye in diabetes mellitus patients and comparing with duration and urea, creatinine level**” is a bonafide and genuine research work carried out by me under the guidance of Dr. M.Hemanandini M.S.D.O, Head of the Department, Department of Ophthalmology, Coimbatore Medical College &Hospital, Coimbatore.

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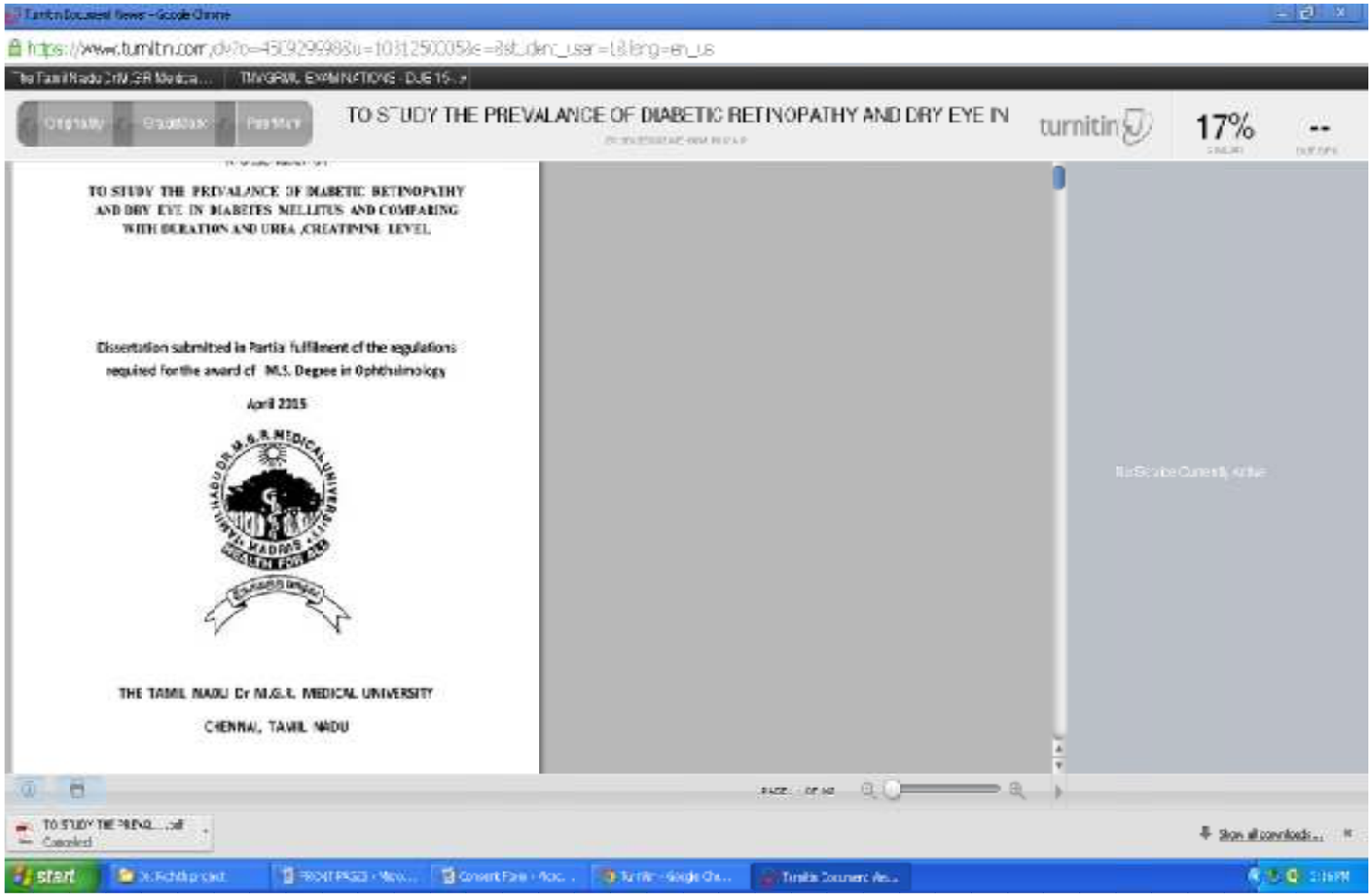
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A Dissertation on

TO STUDY THE PREVALANCE OF DIABETIC RETINOPATHY AND MOA TAE IN DIABETIC MELLITUS AND COMPARING WITH DURATION AND UREA CREATININE LEVEL

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April 2012

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

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Date:

Place: **Dr P MOHANAPRIYA**

ABBREVIATIONS

DM	:	Diabetes mellitus
MODY	:	Maturity Onset Diabetes mellitus in Young individuals
GTT	:	Glucose Tolerance Test
SCH	:	Sub Conjunctival Haemorrhages
DR	:	Diabetic Retinopathy
NPDR	:	Non Proliferative Diabetic Retinopathy
PDR	:	Proliferative Diabetic Retinopathy
VEGF	:	Vasclar Endothelial Growth Factor
LASIK	:	Laser In Situ Keratomileusis
BMI	:	Body Mass Index
UAER	:	Urine Albumin Excretion Rate
UKPDS	:	United Kingdom Prospective Diabetic Survey
ETDRS	:	Early Treatment Diabetic Retinopathy Study
TBUT	:	Tear Film Breakup Time
NFL	:	Nerve Fibre Layer
OPL	:	Outer Plexiform Layer
ILM	:	Internal Limiting Membrane
CSME	:	Clinically Significant Macular Edema
SPK	:	Superficial Punctate Keratopathy
DD	:	Disc Diameter

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INTRODUCTION

Diabetes mellitus is a group of metabolic disorder characterized by chronic hyperglycemia associated with disturbances of carbohydrate, protein and fat metabolism due to absolute or relative deficiency in insulin action and or secretion . Diabetes mellitus causes long term damage , dysfunction and failure of organs expecially the eyes, kidneys, heart, nerves and blood vessels.

TYPES OF DIABETES MELLITUS

Type 1

Beta cell destruction leading on to insulin deficiency

1. Auto immune
2. Idiopathic

Type 2

1. Predominantly insulin resistance
2. Predominantly insulin secretory defects

Other special types of diabetes melittus

1. Genetic defects of beta cell dysfunction,e.g.MODY 1 TO 6
2. Genetic defects in insulin action,.e.g.Type A insulin resistance
3. Diseases of exocrine pancreas, e.g Fibro Calculus pancreatopathy
4. Endocrinopathies,e.g. acromegaly, cushings
5. Drugs or chemical induced e.g glucocorticoids

6. Infections, e.g., congenital rubella
7. Other genetic syndromes
8. Gestational diabetes mellitus

DIAGNOSTIC CRITERIA OF DIABETES MELITTUS

Diabetes is diagnosed if the fasting value is ≥ 126 mg or 2 hour plasma glucose is ≥ 200 mg

Impaired GTT is present when the two – hour value is in the range of 140 – 199 mg/dl

Impaired glucose tolerance is present when the fasting level is ≥ 100 and ≤ 125 and the 2 hour value is ≤ 140 mg /dl.

AETIOPATHOGENESIS OF TYPE 1 DM

1. Genetic factors
2. Immunological factors
3. Viruses / toxins
4. Dietary factors.

AETIOPPATHOGENESIS OF TYPE 2 DM

1. Impaired pancreatic insulin secretion
2. Impaired peripheral action of insulin
3. Impaired resistance as a primary defect
4. Insulin secretory defect as a primary event

COMPLICATIONS OF DIABETES MELITTUS

ACUTE COMPLICATIONS –Diabetic ketoacidosis

CHRONIC COMPLICATIONS- Microvascular and macrovascular

- Vasculopathy
- Retinopathy
- Neuropathy
- Nephropathy

OPHTHALMIC MANIFESTATIONS OF DIABETES MELITTUS

- Anterior segment manifestations
- Posterior segment manifestations

ANTERIOR SEGMENT MANIFESTATIONS

- Lid and adnexa- hordeolum externum, hordeolum internum and chalazion
- Conjunctiva –recurrent SCH and dry eye or conjuctival xerosis
- Cornea – recurrent corneal erosions
- Anterior chamber and the angle – primary open angle glaucoma, neovascular glaucoma
- Iris -rubeosis iridis
- Pupil- smaller pupil
- Lens –posterior sub capsular cataract and nuclear cataract

POSTERIOR SEGMENT MANIFESTATIONS

- Vitreous –asteroid hyalosis , vitreous haemorrhage
- Retina –diabetic retinopathy.

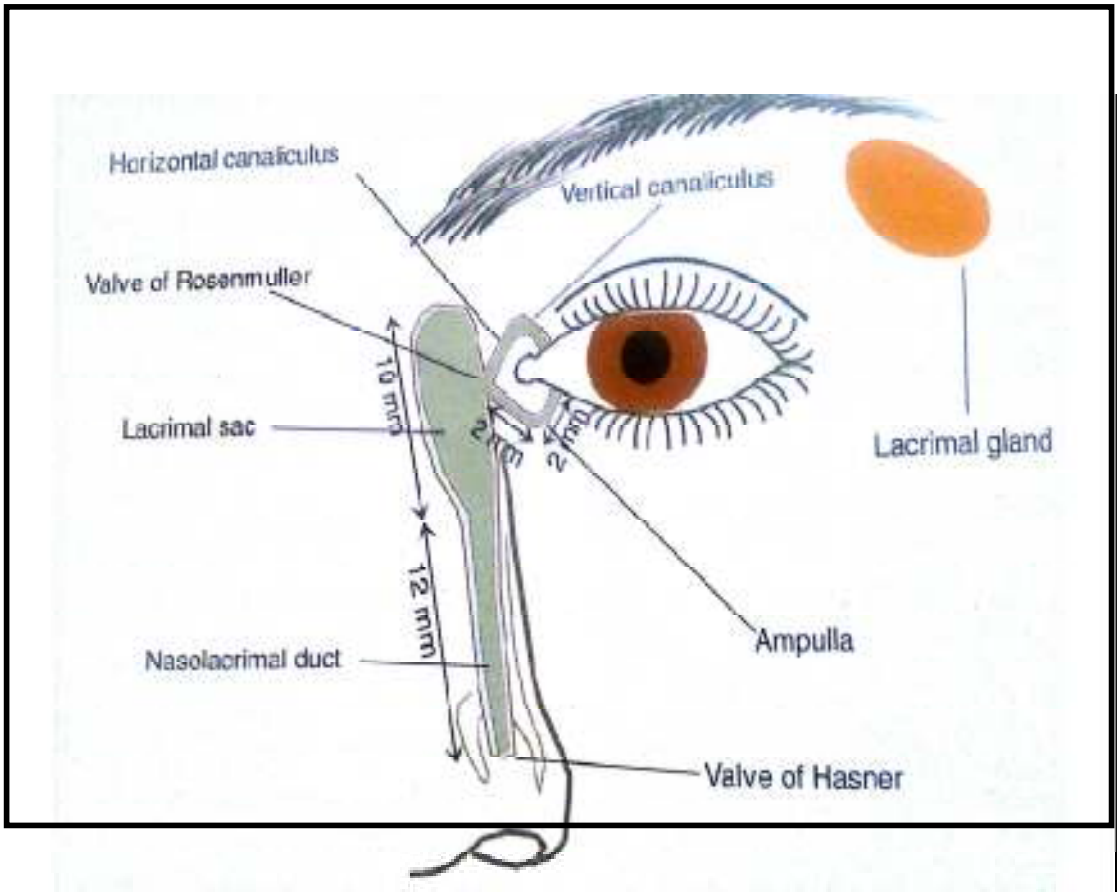


Figure 1: Lacrimal apparatus

LACRIMAL APPARATUS

- ANATOMY
- PHYSIOLOGY
- DRY EYE
- EVALUATION

ANATOMY OF LACRIMA L APPARATUS

- Lacrimal gland
- Upper and lower puncta
- Canaliculi
- Lacrimal sac
- Naso lacrimal duct

LACRIMAL GLAND ANATOMY

The lacrimal gland situated in the lacrimal gland fossa, formed by the orbital plate of frontal bone. The gland is divided into two parts. Orbital and palpebral parts by the aponeurosis of the LPS. About 10 to 12 ductules pass from the orbital part to the palpebral part vertically and opened into the superolateral fornix.

UPPER AND LOWER PUNCTA

They are located 6 and 6.5 mm lateral to the medial canthus respectively. They are visible only on slight eversion of the eyelids.

CANALICULI

Canaliculi have vertical and horizontal parts. The proximal limb is about 2 mm and is vertically oriented. Canaliculi then turn medially and run horizontally for about 8 mm before opening into the common canaliculus and empty into the lacrimal sac. Valve of the Rosenmuller which prevents reflux entry of tears into the canaliculi.

LACRIMAL SAC

It is about 12 to 15 mm long and lies in the lacrimal fossa bounded by the posterior and anterior lacrimal crest. The lacrimal sac is bounded superiorly by the medial palpebral ligament. Lacrimal and frontal bone of maxilla separate the lacrimal apparatus from the middle meatus

THE NASOLACRIMAL DUCT

It is about 18 mm long with intraosseous and intrameatal parts. It passes inferiorly, posteriorly and laterally to open into the inferior meatus of the nose. Mucosal fold covering the inferior meatus is called valve of Hasner.

PHYSIOLOGY OF LACRIMAL SYSTEM

TEAR SECRETION

The tear film composed of three layers.

1. Mucin layer
2. Aqueous layer
3. Lipid layer

Aqueous layer of the tear film is secreted by the lacrimal glands and the accessory lacrimal glands. The mucin layer is contributed by the goblet cells.. The lipid layer is secreted by the meibomian glands.

TEAR FILM FUNCTIONS

1. Tear film form an almost perfectly smooth optical surface on cornea by filling in and smoothening out small surface irregularities in the corneal epithelium.

2. It serves to keep the surface of the cornea and conjunctiva moist.

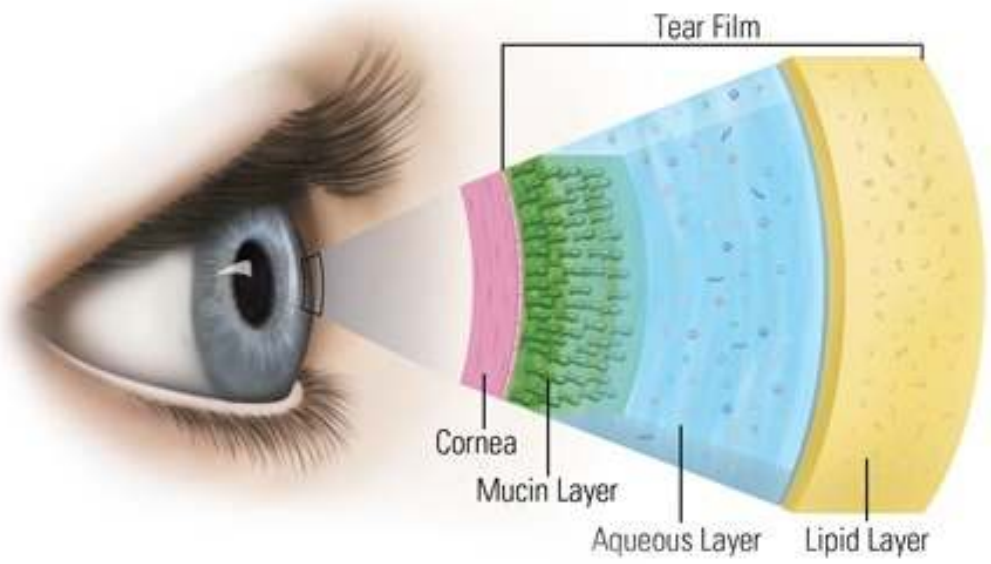
3. It serves as a lubricant for the preocular surface and lids, thereby decreasing the frictional forces that are generated during the constant blinking movements of the eye lids.

4. It transfers the oxygen from the ambient air to the cornea.

5. It prevents infection due to the presence of antibacterial substance such as lysosyme, betalysin, lactoferrin, immunoglobulins and other proteins.

6. It washes away debris and noxious irritants.

7. It provides pathway for white blood cells in case of injury.



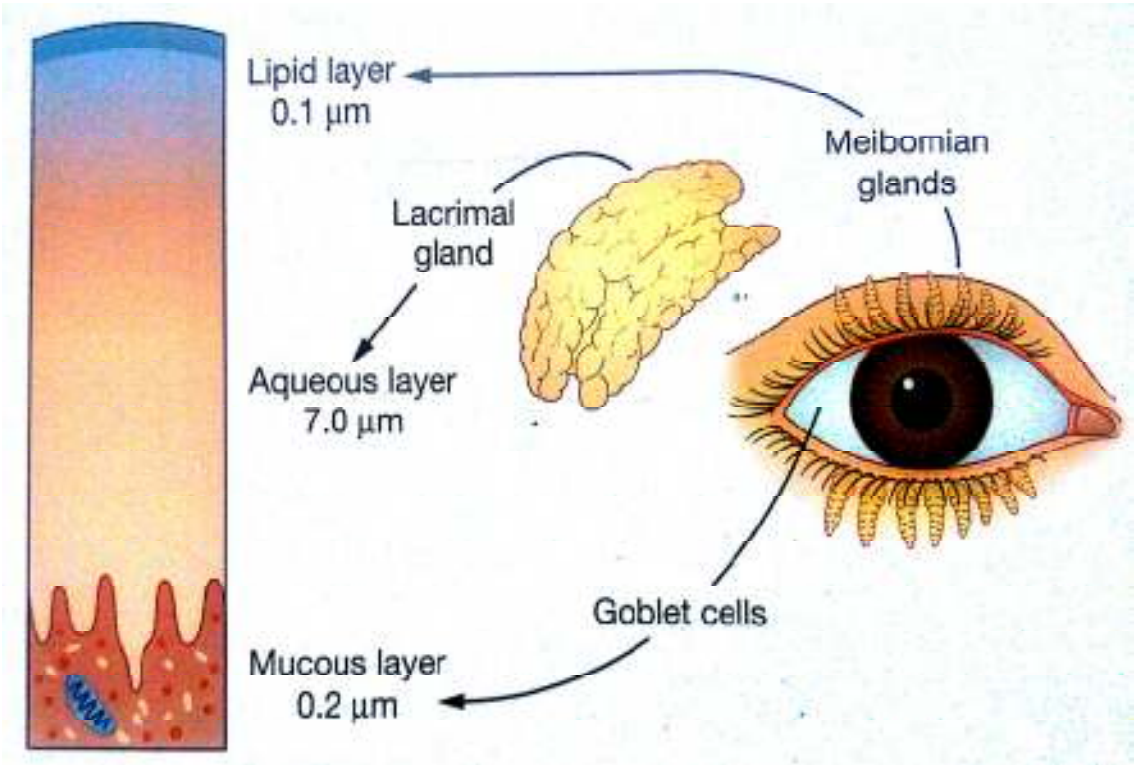


Figure2:Layers of tear film

STRUCTURE OF TEAR FILM

LIPID LAYER

This outermost superficial oily layer derived from the secretions of meibomian, zeiss, and moll glands. This layer consists of lipids having low polarity, such as wax and cholesterol esters. High polarity lipids present in less amounts. The lipid layer thickness is about 0.1 μm and depends on palpebral fissure width, it increases when lids are partially closed. The lipid layer prevents the overflow of tears and retards their evaporation.

AQUEOUS LAYER

The aqueous layer secreted by the main lacrimal glands and the accessory glands of Krause and Wolfring. This layer of tear film contributes to main thickness. Aqueous layer compromised more than 95% of tear film. Thickness of aqueous layer of precorneal tear film is about 10 μm. This layer constitutes inorganic salts, glucose ,urea, enzymes, proteins and glycoproteins. Lysosyme, lactoferrin, tear specific prealbumin and secretory immunoglobulin – A are the main constituents of protein fraction. It seems to provide atmospheric oxygen to the epithelium, washes away the debris and noxious irritants and contain antibacterial substances.

MUCOUS LAYER

The deepest layer is the mucous coat. It plays vital role in the stability of the tear film. The thickness layer is 0.02 -0.05 μm. This mucus layer is secreted by the goblet cells of conjunctiva, crypts of henle and the manz glands. Clear corneal epithelium is hydrophobic surface. Mucin produced by goblet cells, mixed and spread by action of lids ,gets adsorbed on the cell membrane of the epithelial cells and anchored by their microvilli forming a new hydrophilic surface. It plays important role in tear film stability.

The mucin lubricates the ocular and palpebral surfaces. It also provides a slippery coating over foreign bodies, thus protecting the cornea against abrasion.

TEAR FILM DYNAMICS

1. Secretion of tears
2. Formation of tear film
3. Retention and redistribution of tear film
4. Displacement phenonmenon
5. Evaporation from the tear film
6. Drying and break up of tear film
7. Dynamic events during blinking
8. Elimination of tears

SECRETION OF TEARS

Tears are secreted by main lacrimal gland and accessory glands. Reflex tear secretion occurs in respond to sensations from the cornea and conjunctiva probably produced by evaporation and breakup of tear film. Hyper secretions occurs due to irritative sensations from the cornea and conjunctiva. Afferent pathway for this secretion is formed by the fifth nerve and efferent by parasympathetic supply of lacrimal gland

The normal rate of tear production is about 1.2µl/min, tear volume in the eye at any time about 7 µl and turn over rate is 5 to 7 minutes

FORMATION OF PREOCULAR TEAR FILM

Sequence of events in the formation of precorneal tear film

1. Lids surfacing the cornea with a thin layer of mucus
2. On this new surface the aqueous component of tears now spread spontaneously
3. As the aqueous layer spreads , superficial lipid layer spreads over aqueous layer, probably contributing to its stability and retarding evaporation. So the outermost layer is superficial lipid layer.

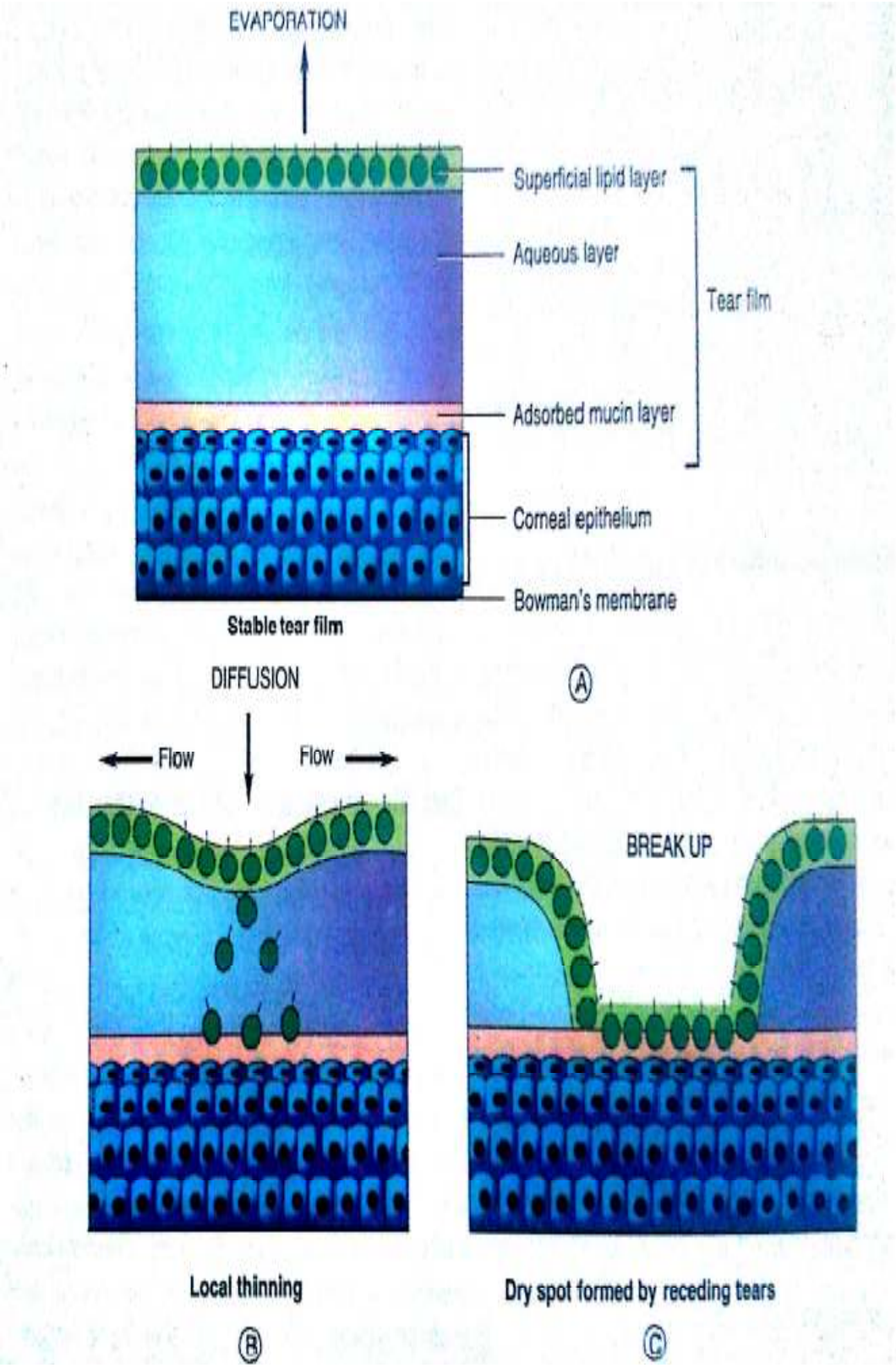


Figure 3:Mechanism of tear film breakup

RETENTION AND REDISTRIBUTION OF TEAR FILM

Outermost layer of the corneal epithelium, along with mucopolysaccharides play an important role in retaining fluid layer on the corneal surface.

DISPLACEMENT PHENONMENON

It is due to thin mono molecular layer on the surface of cornea.

EVAPORATION FROM THE TEAR FILM

Wax esters and cholesterol esters in the lipid layer retard the evaporation of the water. Superficial lipid layer is important under conditions of low humidity and turbulent air flow.

STABILITY, DRYING AND RUPTURE OF THE TEAR FILM

The tear film can function properly only if the tear film covers entire corneal surface and it is re-established quickly and completely after a blink

Mechanism of tear film break up

1. Tear film thins uniformly by evaporation
2. Tear film is thinned out to some critical thickness, lipid molecules are attracted by the mucin layer. So the mucin becomes hydrophobic and the tear film ruptures
3. Blink repair the rupture by removing lipid from the mucin layer and a restoring a thick aqueous layer

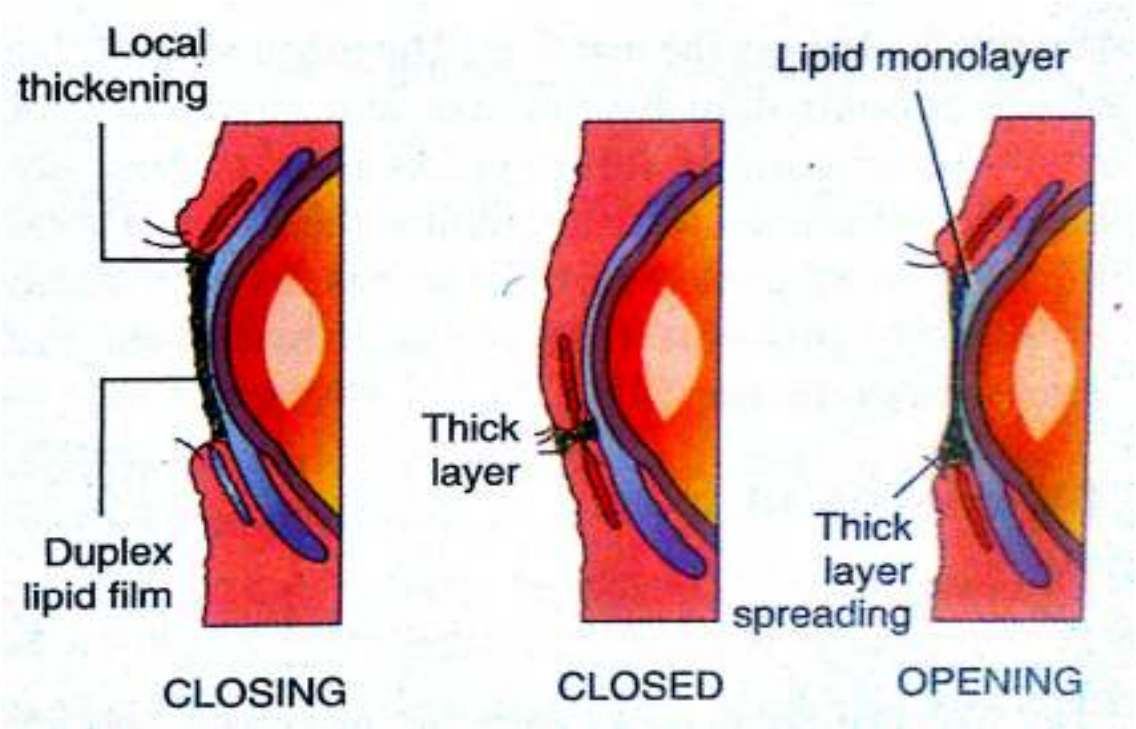


Figure 4 : Dynamic events during blinking

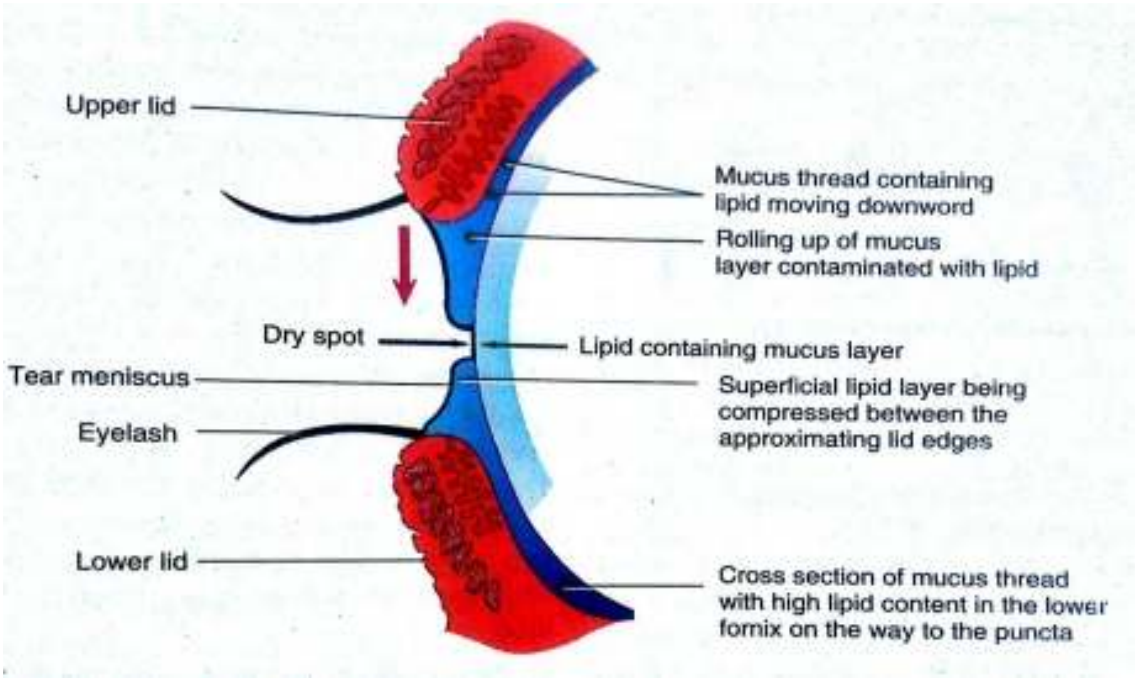


Figure 5 : Mechanism of removal of lipid contaminated mucus

DYNAMIC EVENTS DURING BLINKING

Complex of events take place during blinking. When upper lid moves down. The superficial layer is compressed. Lipid contaminated with mucus dragged into the lower fornix. When eyes open ,lipid spread in the form of mono layer followed by spreading of excess lipid. The spreading liquid drags aqueous with it, which results in thickening.

ELIMINATION OF TEARS

By active lacrimal pump mechanism. The muscle involved is preseptal portion of orbicularis oculi, which arise from the the lacrimal fascia and posterior lacrimal crest. It operates with blinking movements.

Events occuring during eye closure

1. Contraction of pretarsal fibres of orbicularis oculi compresses the ampulla and shortenes canaliculi. This movement propels tear fluid towards the lacrimal sac.
2. Contraction of pre tarsal fibres of orbicularis pulls lacrimal fascia and lateral wall of lacrimal sac laterally. So tear from canaliculi draws into lacrimal sac.
3. Increased tension on the lacrimal fascia closes the inferior portion of lacrimal sac, so preventing aspiration air from the nose.

Events occuring during eye open

1. Relaxation of pre tarsal fibres of orbicularis oculi allows the canaliculi to expand and reopen. This draws the fluid from the lacrimal lake.
2. Relaxation of Horner muscle allows lacrimal sac to collapse. This resulting in opening of naso lacrimal duct.

Drainage of lacrimal fluid into nasal cavity

1. Gravity helps in downward flow
2. Negative pressure in the naso lacrimal duct draws the tear into the nose.
3. Hasner valve opens until pressure in the nose is less than that of the naso lacrimal duct. When intra nasal pressure raises hasner valve closes.

TEAR FILM ABNORMALITIES

Dry eye is described as a state of abnormal tear film caused by alteration in its composition and conditions affecting their stability.

CLASSIFICATION OF TEAR FILM ABNORMALITY

1. Aqueous deficiency e.g kerato conjunctivitis sicca, congental alacrima, paralytic hypo secretion, idiopathic and systemic diseases causing decreased tear secretion

2. Mucin deficiency occur in hypovitaminosis A, ocular pemphigoid, SJ syndrome, drug induced, and chemical burns
3. Lipid abnormality occur in chronic blepharitis
4. Lid function abnormality seen in exposure keratitis, symblepharon, lagophthalmos and pterygium
5. Epithelial dysfunction seen in anaesthetic cornea, corneal epithelial abnormality of any cause

KERATO CONJUCTIVITIS SICCA- ANY EYE WITH SOME DEGREE OF DRYNESS

CLASSIFICATION OF KERATO CONJUCTIVITIS SICCA

1. Aqueous layer deficiency
    . sjogrens syndrome
    . non sjogrens
2. Evaporative
    . meibomian gland disease
    . exposure
    . defective blinking
    . contact lens associated
    . environmental factors

CAUSES OF NON SJOGRENS KERATO CONJUCTIVITIS SICCA

1. Primary - age related hypo secretion
2. Lacrimal gland destruction -tumours and inflammation
3. Absence or reduction of lacrimal gland tissue - surgical removal or rarely congenital
4. Conjunctival scarring and obstruction of lacrimal gland ductules - chemical burns, cicatricial pemphigoid, stevens Johnson syndrome , long standing trachoma
5. Neurological lesions with sensory or motor reflex loss
6. Vitamin A deficiency.

CAUSES OF EVAPORATIVE KERATO CONJUCTIVITIS SICCA

1. Meibomian gland dysfunction
    - . posterior blepharitis
    - . rosacea
    - . atopic keratoconjunctivitis
    - . congenital meibomian gland absence
2. Lagophthalmos
    - . severe proptosis
    - . facial nerve palsy
    - . eye lid scarring
    - . following blepharoplasty

3. Miscellaneous

    . cantact lens wear

    . environmental factors such as air conditioning

CLINICAL MANIFESTATIONS OF DRY EYE

OCULAR SYMPTOMS

1. Feeling of dryness
2. Grittiness
3. Burning
4. Stringy discharge
5. Transient blurring of vision
6. Redness

OCULAR SIGNS

1. Posterior blepharitis
2. Redness and mild keratinization of conjunctiva
3. Lipid contaminated mucin can accumulate as particles in tear film
4. Marginal tear meniscus is normally about 1 mm, it is less or absent in dry eye
5. Froth in tear film
6. Punctuate corneal erosions
7. Mucus filaments and mucous plaques

8. Peripheral superficial corneal vascularisation, epithelial break down, melting, perforation, bacterial keratitis are due to dry eye complications

DRY EYE DIAGNOSIS BY

- ❖ Tear production - schirmer test
- ❖ stability of the tear film - TBUT

TEAR FILM BREAKUP TIME

Tear film breakup time is abnormal in aqueous deficiency and meibomian gland disorders

- ❖ 2% fluorescein or an impregnated fluorescein strip soaked with saline is instilled into conjuctival lower fornix
- ❖ Patient is asked to blink
- ❖ Tear film is examined with slit lamp examination by using cobalt blue filter, formation of dry spots or lines on fluorescein areas indicates dry eye
- ❖ Break up time is an interval between the last blink and the appearance of the dry spot. TBUT of less than 10 seconds is considered as abnormal.
- ❖ Appearance of dry spot in same location may indicate local surface corneal abnormality rather than tear film instability.

Figure 6 :Fluorescein strips

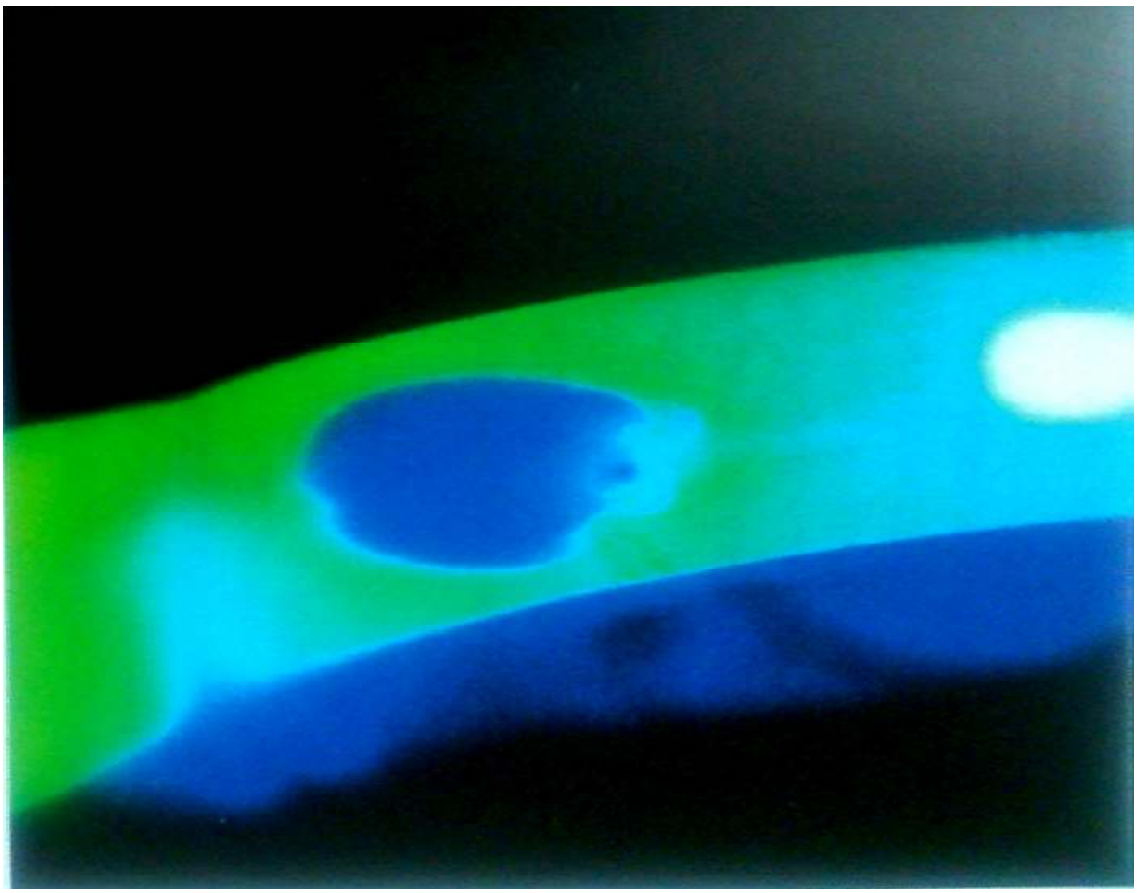


Figure 7: Tear film breakup time

SCHIRMER TEST

Assessing aqueous tear production

Measuring the amout of wetting of a special no 41 Whatman filter paper, it is 5 mm wide and 35 mm long

Schirmer test 1- with out an local anaesthetic. It measures both basal and reflex secretion

Schirmer test 2 - with an local anaesthetic. It measures only basal secretion

The filter paper is folded 5mm from one end and placed in the lower fornix at the junction of the middle one third and outer one third of the lower lid.

After 5 minutes the Schirmer strip is removed and the amout of wetting from the fold measured.

Less than 10 mm of wetting after 5 minutes with out anaesthesia and less than 6 mm with anaesthesia is abnormal.

Figure 8 : Schirmer strips

Figure 9 :Schirmer test

OTHER TESTS FOR DRY EYE

1. Ocular surface staining with rose Bengal and fluorescein
2. Fluorescein clearance test
3. Lactoferrin assay
4. Phenol red thread test
5. Tear meniscometry
6. Tear film osmolarity
7. Impression cytology

DIABETIC RETINOPATHY

The classification of diabetic retinopathy is based on clinical features. it is divided into

1. NON PROLIFERATIVE DIABETIC RETINOPATHY
2. PROLIFERATIVE DIABETIC RETINOPATHY

NPDR refers to presence of intra retinal vascular changes with out the presence of extra retinal fibrovascular tissue. It is further subdivided into mild ,moderate and severe. NPDR is also referred as background diabetic retinopathy. Proliferative diabetic retinopathy refers to presence of extraretinal fibrovascular proliferative tissue. It is further subdivided into early, highrisk or advanced.

PATHOGENESIS OF DIABETIC RETINOPATHY

Cause of diabetic retinopathy is exactly not known. It is believed that exposure to hyperglycemia over an extended period results in biochemical and physiological changes that ultimately cause endothelial damage. Specific changes in retinal capillaries include loss of pericytes and thickening of the basement membrane resulting in occlusion of capillaries and nonperfusion of retina and decompensation in retinal barrier function .This leads to retinal leakage and retinal edema.

The prevalence of DR increases with the duration of DM and patient age. The risk of diabetes increases after puberty.

The following hematological and biochemical abnormality have been correlated with the prevalence and severity of retinopathy.

- Increased platelet adhesiveness
- Increased erythrocyte aggregation
- Abnormal level of serum lipids
- Defective fibrinolysis
- Abnormal level of growth hormone
- Increased level of VEGF
- Abnormalities in serum and whole blood viscosity
- Local and systemic inflammation

RISK FACTORS FOR DIABETIC RETINOPATHY

1. Duration of diabetes
2. Poor glycemic control
3. Pregnancy - rapid progression of DR
4. Hypertension
5. Nephropathy
6. Hyperlipidemia
7. Cataract surgery
8. Obesity
9. Anaemia

CLASSIFICATION OF DIABETIC RETINOPATHY BY ETDRS CLASSICATION

1. Non proliferative Diabetic Retinopathy
2. Proliferative Diabetic Retinopathy

NPDR CLASSIFIED INTO

1. No NPDR
2. Very Mild NPDR
3. Moderate NPDR
4. Severe NPDR
5. Very severe NPDR

PROLIFERATIVE DIABETIC RETINOPATHY [PDR]

1. Mild - moderate PDR
2. High risk PDR
3. Advanced diabetic eye disease

DEFNITIONS

Very mild NPDR - micro aneurysms only

Mild NPDR - any or all of : microaneurysms, retinal haemorrhages, exudates, cotton wool spots, upto the level of moderate NPDR. No intra retinal micro angiopathy or significant beading.

Moderate NPDR - severe retinal haemorrhages in 1 or 3 quadrants, mild intra retinal microvascular abnormality, significant venous beading can be present in no more than 1 quadrant, cotton wool spots commonly present.

Severe NPDR	-	The 4-2-1 rule; one or more of Severe haemorrhages in all 4 quadrant Significant venous beading in two or more quadrants Moderate intra retinal micro vascular changes in 1 or more quadrants
Very severe	-	2 or more of the severe NPDR criteria PDR
Mild to moderate PDR	-	NVD or NVE, but extent insufficient to meet high risk criteria

High risk PDR - new vessels on the disc about 1/3 of disc area, any NVD with vitreous or pre retinal haemorrhage, NVE greater than ½ disc area with vitreous and pre retinal haemorrhage.

Advanced diabetic eye disease includes

- ❖ Haemorrhage may be preretinal, intra retinal or both
- ❖ Tractional retinal detachment

- Tractional retinoschisis
- Rubeosis iridis

MICRO ANEURYSMS

Are localised saccular outpouching of the capillary wall, often caused by pericyte loss.

They are round intra retinal lesions of 30 - 120 μ in size and are located in inner nuclear layer of the retina

Signs of microaneurysms - tiny red dots , they are earliest sign of diabetic retinopathy. They may be indistinguishable from dot haemorrhages.

RETINAL HAEMORRHAGES

- Retinal nerve fibre layer haemorrhages
- Intra retinal haemorrhages
- Deeper dark round haemorrhages

Retinal nerve fibre layer haemorrhages - flame shaped, occurring at the nerve fibre layer, they follow the architecture of the nerve fibre layer. They arise from superficial precapillary arterioles.

Intra retinal haemorrhage - they are from venous ends of capillaries. Located in middle layer of the retina resulting in dot and blot haemorrhages.

Deeper retinal haemorrhages - haemorrhagic retinal infarcts located in middle layer of retina. They are significant marker of progression to retinal neovascularisation.

EXUDATES

They are hard exudates.

Exudates are chronic localized edema and appear at the junction of the normal and swollen retina.

They are composed of lipoproteins and lipid laden macrpophages and are located in the main outer plexiform layer of the retina.

Signs - yellow waxy lesions with distinct margins, arranged in rings or clumps in posterior pole, surrounding local aneurysms. They get absorbed over a period of time.

COTTON WOOL SPOTS

They are due to ischemic infarction of the NFL layer of retina. Due to interruption of axoplasmic stasis with in the axons

Signs - small ,whitish superficial lesions ,obscuring underlying blood vessels.

INTRA RETINAL MICROVASCULAR ABNORMALITY

They are arteriolar - venular shunts that run from the retinal arterioles to venules.

Signs - fine, red irregular, intra retinal lines that run from arterioles to venules without crossing major blood vessels

CAUSES OF DIMNISHED VISION IN DIABETIC RETINOPATHY

1. Macular edema
2. Macular ischemia
3. Vitreous haemorrhage
4. Tractional retinal detachment
5. Optic atrophy due to direct papillopathy or following extensive panretinal photocoagulation

CSME DEFINED AS

1. Thickening of retina at or with in 500 μ of the centre of the macula
2. Hard exudates at or with in 500 μ of the centre of the macula with adjacent retinal thickening
3. Retinal thickening of 1 DD or larger, any part of which is within 1 DD of the centre of the macula.

PROLIFERATIVE DIABETIC RETINOPATHY

One quarter of the retina must be non perfused before proliferative retinopathy develops.

NVD - neovascularisation on or within 1 disc DD of the optic nerve head

NVE- neovascularisation further away from the disc

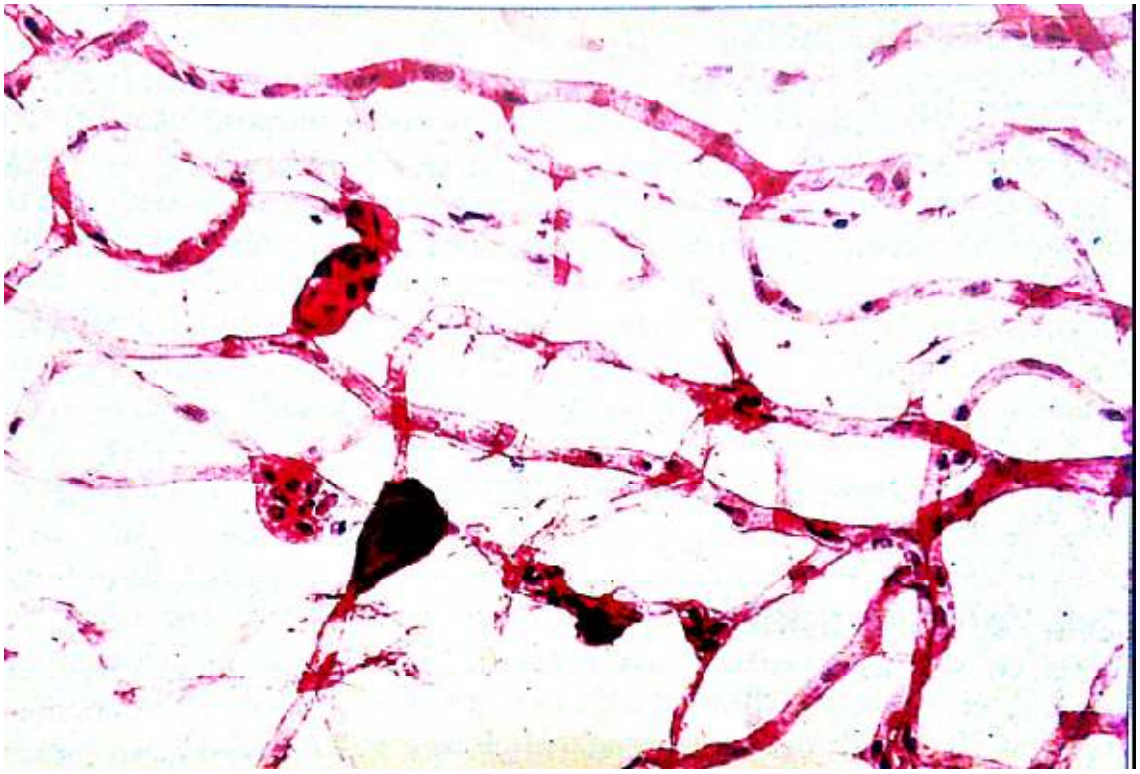


Figure 10 :Micro aneurysms, pericyte dropout, and acellular capillaries

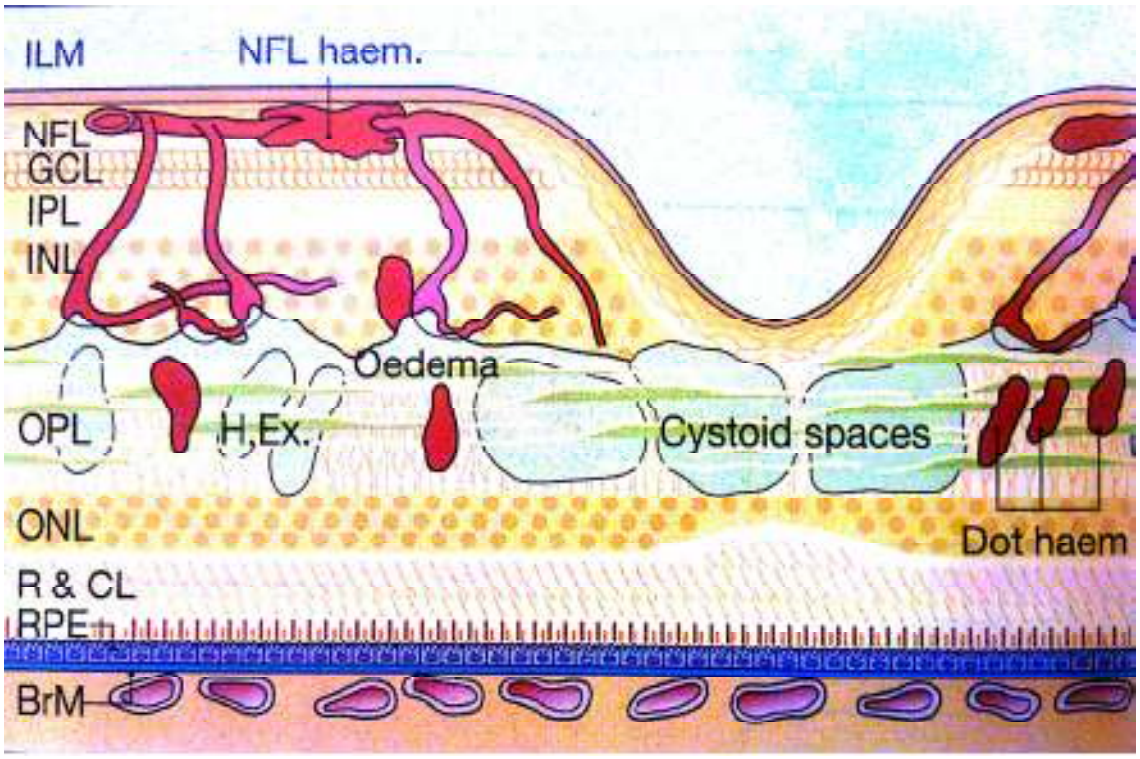


Figure 11 :Locations of lesions in diabetic retinopathy

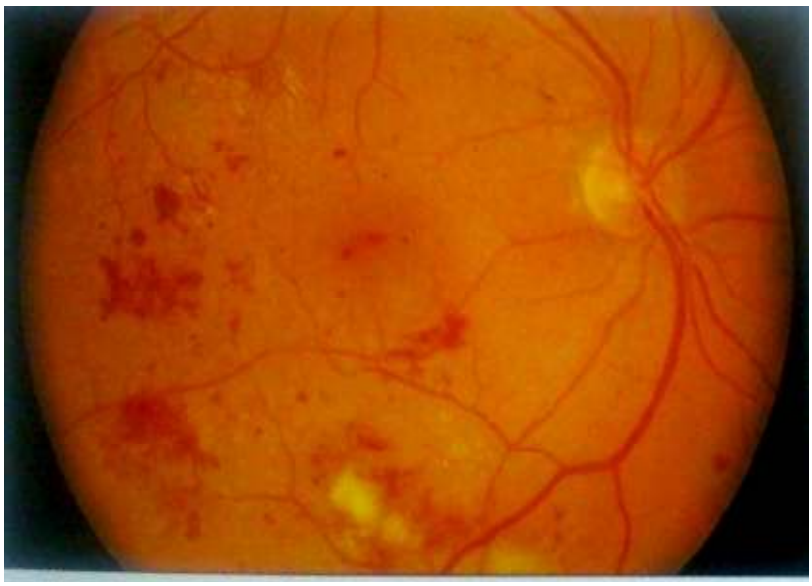


Figure 12 :NPDR with blot haemorrhages, splinter haemorrhages and cotton wool spots

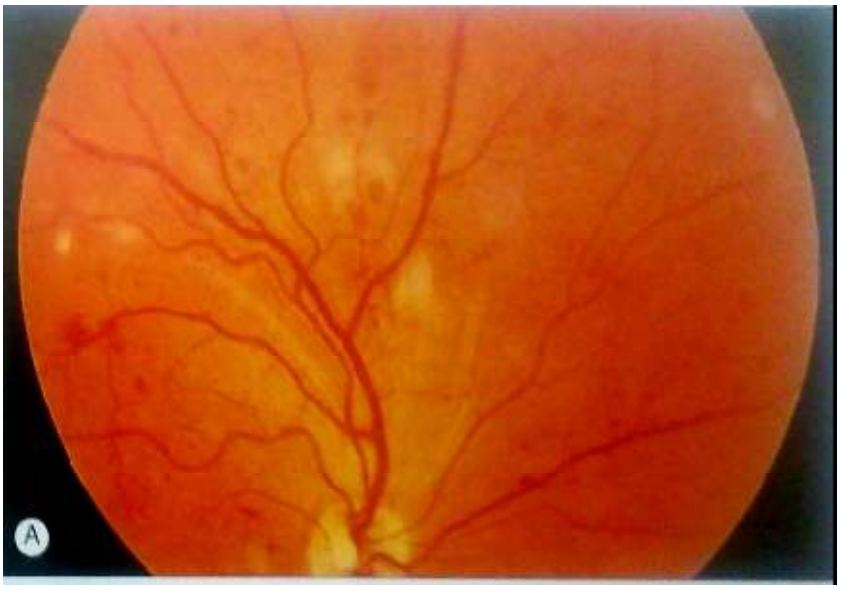


Figure 13 :Severe NPDR with IRMA and venous beading

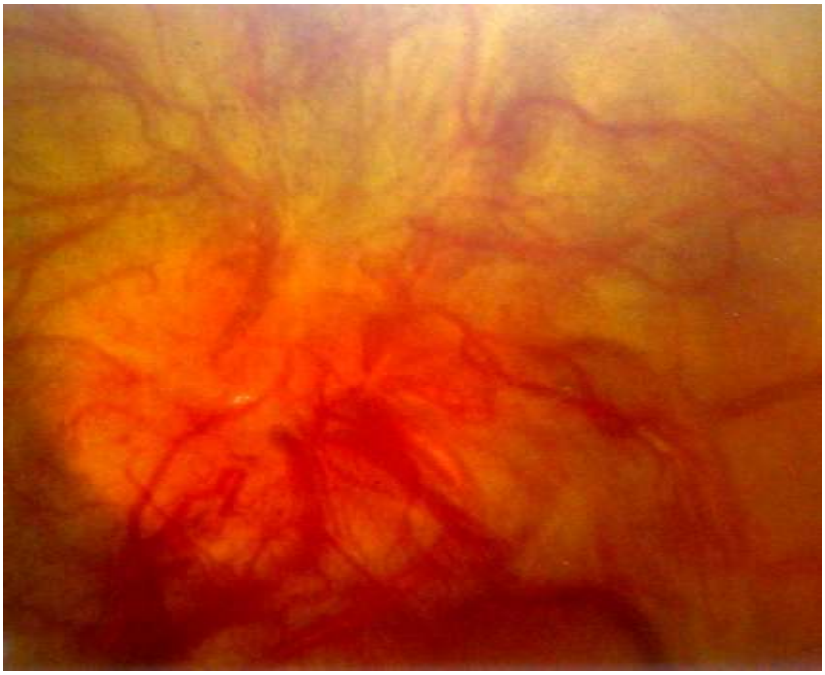


Figure 14 :Neovascularization of the disc

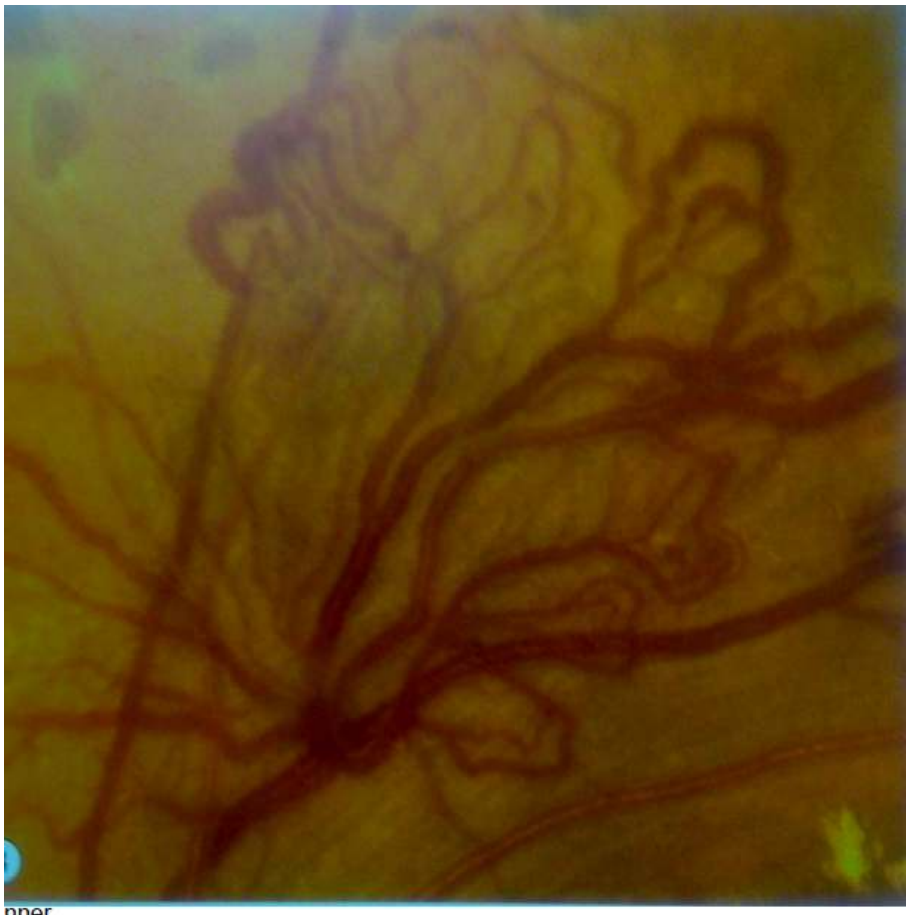


Figure 15 :Neovascularization elsewhere

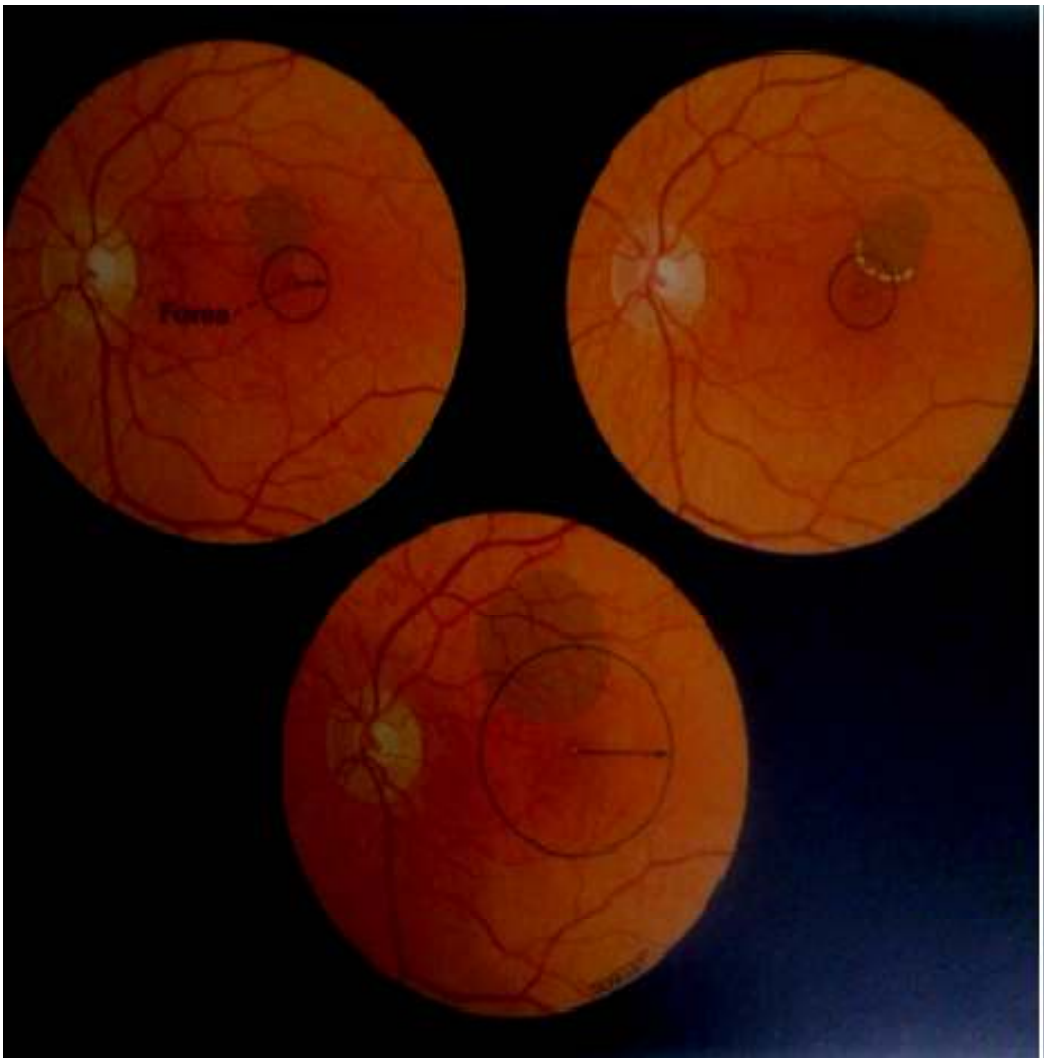


Figure 16 :Clinically significant macular edema

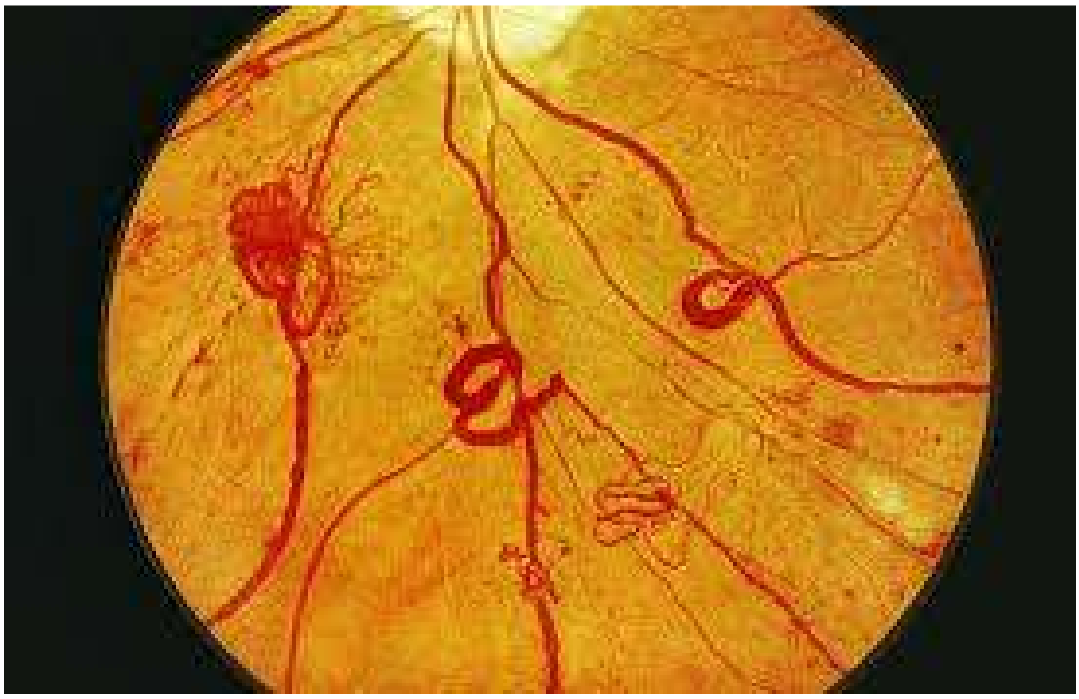


Figure 17 :DR with venous looping

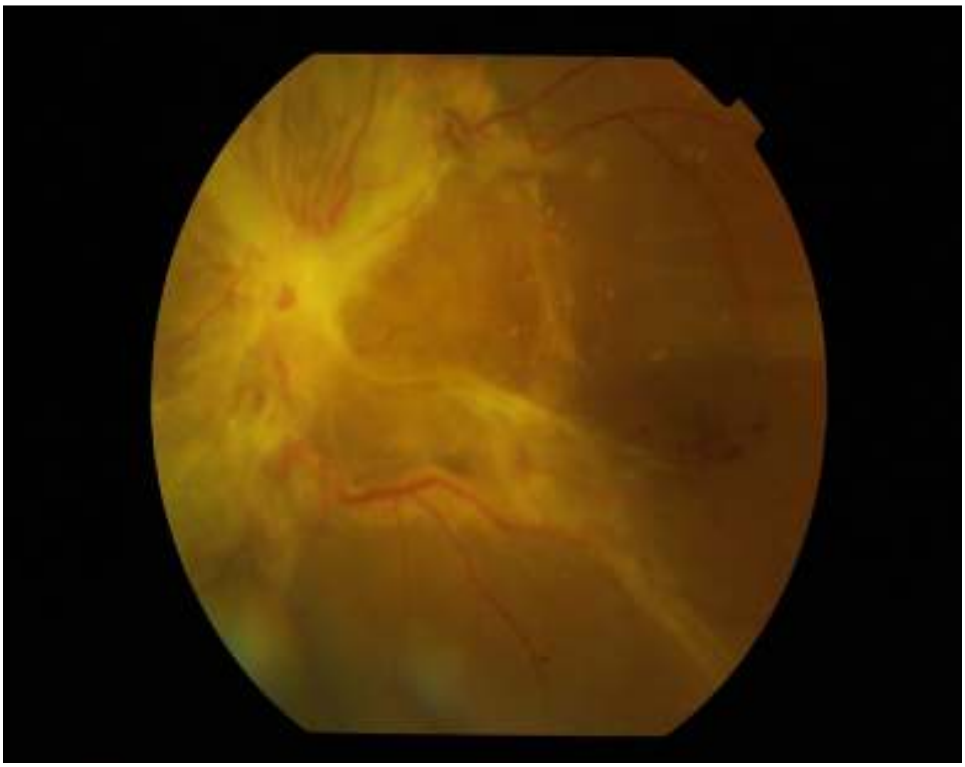


Figure 18 : Proliferative diabetic retinopathy

REVIEW OF LITERATURE

Dry eye was first described by French dermatologist Gougerot. He described dry eye was due to atrophy of the salivary and conjunctival gland in middle aged women. The Swedish ophthalmologist Henrik Sjogren in 1933 established the term kerato conjunctivitis sicca. He described a disease characterised by auto immune damage to lacrimal gland tissue, decreased tear secretion and ocular surface disease[1].

Maintenance of normal tear film depends on normal ocular surface. Normal ocular surface means normal epithelial surface and normal mucous surface[2]. Mishima was the first to recognise the contribution of the aqueous humour to the aqueous component of the tear film.

The lacrimal gland is innervated by both parasympathetic and sympathetic nerves. Parasympathetic fibres travelling along the lacrimal nerve stimulate lacrimal gland secretion[3]. Beta 1 adrenergic agonists also stimulates lacrimal gand secretion. So parasympathetic blockers and beta 1 adrenergic blockers can diminish lacrimal gland secretion. Lacrimal secretory system has two parts.

1. Basic secretion by goblet cells and glands of Krause and wolfring
2. Main lacrimal gland contributes to reflex secretion

Normal blink rate averages once every 5 seconds. Blinking is important for spreading the freshly secreted tears. Decreased corneal sensation in one eye does not diminish the blink rate. Diminished corneal sensation in both eye diminish the blink rate. So decrease in corneal sensation resulting in decreased tear secretion[4]. Meibomian gland dysfunction occur in meibomitis. This result in increased tear evaporation[5].

Diabetes is the leading cause of blindness in twenty to seventy four year age group[6]. Dry eye is the most common problem encountered in patients with diabetes mellitus. Due to dry eye patients develop corneal complication which include SPK, trophic ulceration and corneal epithelial defect[7]. Dry eye has many causes. Old age is one of the cause for dry eye[8]. The exact mechanism for cause of dry eye is not known[9]. Autonomic dysfunction plays a role in causing dry eye. Aldose reductase enzyme which converts glucose to sorbitol also plays a role. Aldose reductase inhibitors improve tear dynamics.[10]

Jin at al found that diabetes mellitus type 2 patients prone to develop dysfunction of tear film. So dry eye evaluation should be routinely done in all diabetic patients[11].

Diabetes is associated with ocular conditions such as diabetic retinopathy , changes in refraction, cataract, nerve dysfunction, macular edema and glaucoma. Dry eye is the most common complication[12]. More than half of the patients have dry eye and nearly 53% of the diabetes patients may experience dry eye symptoms[13] .

Cousen et al studied production of tears and sensation of cornea in diabetes. most common dry symptoms encountered in diabetes are foreign body sensation and burning. In addition to dry eye the following are encountered in diabetes[14,15]. Tear film instability, metaplasia of conjunctiva, decreased goblet cells, decreased sensation of cornea and decreased tear film lipid layer[14]. There is a close association between the family history and dry eye symptoms[15].

Dry eye workshop report classify dry eye into two groups, aqueous deficient and evaporative[16]. Beherens at al, classify dry eye as no lid margin disease, lid margin disease and distribution problems[17]. some studies suggests that deficiency of aqueous is the major cause of dry eye. Hyperglycemia impairs inflammatory process and invasion of lids by bacteria leading on to blepharitis in diabetes patients[18]. This is cause for evaporative dry eye. Lid diseases are highly prevalent in diabetes. Aqueous deficient dry eye responds well to control of blood sugar. In contrast evaporative dry eye patients show slow response to blood sugar control.

Both lid treatment and control of diabetes both are needed in evaporative dry eye

CAUSAL FACTORS FOR DIABETIC DRY EYE

Most frequently cited associations

1. Autonomic neuropathy
2. Due to hyperglycemia
3. Insulin deficiency
4. Inflammation

Autonomic neuropathy and hyperglycemia

Autonomic neuropathy is the key result of hyperglycemia and diabetes[19]. Nerve signal pathways are damaged in autonomic neuropathy, resulting in pain, numbness and also gangrene. Microvascular abnormalities and high blood sugar damages the corneal nerves and block the nerve feedback mechanism that controls secretion of tears. So lacrimal gland does not secrete tears[19]. Neurotrophic like condition occur on the cornea due to significant nerve damage.

Benetis – Del -Castillo et al studied relation between innervation of cornea with confocal microscopy and corneal sensations using noncontact esthesiometry in dry eye patients[20]. Dry eye patients have decreased corneal sensitivity due to change in corneal nerve architecture and decrease in volume and density of nerve architecture..

Insulin deficiency

Insulin exerts important effects on the glands throughout the body. Insulin mediates lot of functions, such as influx of nutrients , storage of energy, expression of genes and synthesis of proteins. Metabolism of lacrimal gland and cornea, growth, proliferation and growth of epithelial cell are influenced by insulin

Inflammation

It is another frequent manifestation of diabetes. So hyperglycemia alters inflammatory reactions and reduces tear secretion.

Jin et al described type 2 diabetes mellitus patients develop dysfunction of tear film. Dry eye can lead to vision deficit, perforation and scarring of the cornea and bacterial infection. Diagnosis of dry eye made early and treated will protect the cornea from above complications[21].

Smith JA et al told dry eye signs and symptoms in women with premature ovarian failure[21]. With no anaesthetic application placing

Whatman no 41 millimetre filter paper strip measuring 5× 30 mm placed in lower fornix and it is kept for a period of 5 minutes, values below 10 mm are pathological.

Lemp ma et al describes factors changing TBUT in normal individuals. Tear film break up time used to measure tear film stability. Less than 10 seconds considered to be abnormal[22].

Dogru stated that diabetes mellitus patients with autonomic neuropathy and uncontrolled diabetes patients exhibit pathological schirmer and TBUT test[23]. Schirmer test is an useful screening test for diagnosing lacrimal hyposecretion. but not adequate for tear production thresthold. Its sensitivity is between 10% and 30%. In diabetic patients schirmer test exhibit lower than normal values[24]. 55% of patients with diabetes had dry eyes symptoms signs[25]. The beaver dam eye study suggested that twenty percent of diabetes mellitus patients aged between forty three and eighty six years diagnosed to have dry eyes[26].

Yu et al , patients with PDR had a significant decrease in tear film function than patients with non proliferative DR[27]. So he concluded dry eye assessment should be done in all diabetic patients.

Boyed BF describes etiological factors for dry eye[28]

1. Increasing age decreases lacrimal gland secretion
2. Hormonal changes in women, menopausal women have reduced amout of androgens produced by ovaries.
3. Auto immune diseases
4. Pharmacological agents - anti depressants, anxiolytics, anti histamines, anti cholinergics, anti hypertensives, anti psychotics, anti parkinsonians, diuretics
5. Vitamin a deficiency - vitamin A is important for mucin production Mucin is important for the corneal surface wetting and its deficiency lead into instability of tear film.
6. Defective corneal sensation due to various etiologies, contact lens users and following LASIK
7. Deficient efferent innervations due to facial nerve palsy
8. Corneal defects such as corneal epitheliopathy, or corneal dystrophy and eyelid disorders such as ectropion and lagophthalmos causes failure of tears to spread over to ocular surface.

Kaiserman and associates have told that good control of blood sugar is important for the control and prevention of dry eye syndrome among diabetic patients[29].

Moss and associates told that females had high incidence of dry eyes compared to males[29]

Nepp and associates stated that severity of dry eye correlates with diabetic retinopathy severity[30]. In diabetes goblet cell density reduced in patients with neuropathy[31]. Decrease in mucin layer results from involvement of conjunctival surface[32].

Schultz et al reported forty seven to sixty four percentage of diabetic patients having corneal abnormalities such as epithelial defects, delayed healing of epithelium, corneal ulcer and kerato conjunctivitis sicca[33]. These manifestation are responsible for clinical corneal manifestation of diabetes

DM is an international health problem with prevalence ranging from 2 % to 11. 7 % in studied population around the world[34].

The microvascular triad of retinopathy, nephropathy and neuropathy is unique to diabetes. Patients with diabetes will have one or more of these as overt or subclinical manifestations during the course of their disease.

Yun et al stated that endothelial dysfunction as an important independent predictors of increased DR prevalence in patients with type 2 diabetes mellitus. Pathogenesis of DR involves inflammation and endothelial dysfunction[35].

Tomic et al revealed the relationship between inflammatory markers and anthropometric parameters and their relation with DR in patients with type 2 DM[36]. High levels of inflammatory markers found in diabetes patients.[37,38]

Diabetic patients with macular edema and PDR had increased levels of VEGF and cytokines in their vitreous than those without macular edema and PDR[39].

Endothelial abnormalities may result in increased vascular permeability, changes in blood flow, oxidative stress, angiogenesis and DR[40,41]. Adherence of leucocytes to the retinal endothelium results in vascular occlusion and most important factor in pathogenesis of DR[42]. Independant risk factors for diabetic retinopathy are longer the duration of diabetes, lean BMI, high systolic blood pressure and insulin resistance[43].

Increased duration influence the occurrence of DR and its severity was due to prolonged exposure to hyperglycemia. Duration of diabetes is independant risk factor. Ten percent of the newly diagnosed diabetes shows DR, suggesting that these patients not diagnosed earlier.

Pradeep et al observed male gender to be associated with the presence of any DR. But not its severity[43,44].

BMI

The inverse relation between body mass index and DR was noticed in Indian based population studies[45,46]. This was due to the catabolic effect of the lack of insulin over a longer duration of hyperglycemia. High BMI was observed in subjects with diabetes. But in asian population patients with diabetes mellitus are lean and low BMI[47,48,49]. Asian with type 2 DM show decreased insulin secretion , but increased insulin sensitivity, seen in asian diabetic patients.

Increase in systolic blood pressure by 10 mmHg showed a linear trend around 1 - 1.2 times the risk of influencing and severity of diabetic retinopathy[45,46,47].

Cataract surgery

Cataract surgery is risk factor for DR[44].

Microalbuminuria

Microalbuminuria is risk factor for the DR in patients with type 2 diabetes mellitus. The urine albumin excretion rate was assessed via 24 hour urine collection and measured by immunoturbidimetric assay. Normoalbuminuria was defined as UAER < 20 μg/ minute in 2 out of 3 consecutive test taken within 2 - 3 months.

Microalbuminuria is a marker of endothelial dysfunction may influence on alteration in the vasculature of retina and kidneys. Microalbuminuria is an independant risk factor for the incidence of DR in patients with type 1 DM[50,51,52,53]. In patients with type 2 DM microalbuminuria is independently associated with DR. Studies reported that DR might develop in patients with type 2 DM patients without microalbuminuria[54,55,56].

Possible risk factor for diabetic retinopathy

1. Number of years since diagnosis of DM[57,58]
2. HbA1c levels
3. Coexistence of hypertension
4. Age
5. Gender

Duration

Important predictor of DR and its severity[57,58,59].

Hypertension

It is an important risk factor for the onset and progression of disease and it is an independant risk factor for DR[60,61]. The UK Prospective Diabetes Study demonstrated that blood pressure control is associated with a reduction in DR prevalence.

HbA1c levels

High HbA1c levels are closely associated with severe DR. Decrease in HbA1c concentrations by 1 % leads to an estimated reduction of 30 % in the risk of microvascular complications[62,63].

Epidemiological studies in diabetic retinopathy

1. Wisconisin Epidemiologicaly Study on Diabetic Retinopathy
2. Diabetic Retinopathy Study
3. Diabetes Control and Complications Trial
4. Diabetic Retinopathy and Vitrectomy Study
5. UK Prospective Diabetic survey

These studies established the various risk factors and provided guidelines for the management of diabetic retinopathy.

The VISION 2020 protocol projects diabetic retinopathy and the glaucoma are the emerging causes of blindness in developing countries.

The Diabetes Control and Complications Trial revealed that good control of metabolic status will reduce the risk of diabetic retinopathy and delays the onset of retinopathy in patients who do not have retinal changes at the time of presentation

United Kingdom Prospective Diabetic Study

Study confirmed that good glycemic control in type 2 non insulin dependant diabetes mellitus is also beneficial and delays the onset of retinopathy

Wisconsin Epidemiological Study of Diabetic Retinopathy

The study reported that increased prevalence of diabetic retinopathy in both type 1 and type 2 DM directly associated wth duration of DM

AIM OF THE STUDY

1. To correlate the dry eye and diabetic retinopathy with duration of diabetes and urea, creatinine level.
2. Evaluation of risk factors attributed to dry eye and diabetic retinopathy in diabetes mellitus patients
3. To study the prevalence of dry eye and diabetic retinopathy in diabetes mellitus patients

MATERIALS AND METHODS

STUDY DESIGN

It is a hospital based cross sectional study

STUDY PERIOD

This study was done between the periods from August 2013 to July 2014

STUDY POPULATION

The study was done on all diabetic patients attending ophthalmology outpatient clinic

Before commencing the study Ethics committee approval was obtained from the Coimbatore medical college and government hospital. Diabetic patients attending outpatient clinic were screened for dry eye and diabetic retinopathy after attaining consent.

Protocol

Informed oral consent obtained

Detailed history taking

Name , age, sex, presenting symptoms, duration and associated systemic diseases were noted.

Regarding diabetes

Type of diabetes, duration, fasting and post prandial blood sugar, urea , creatinine level were noted.

Inclusion criteria

All patients of either sex in all age groups diagnosed to had diabetes mellitus of any duration.

Exclusion criteria

- ❖ Known case of ocular surface disorder
- ❖ Patients who are using contact lenses
- ❖ History of ocular surgeries in past
- ❖ Patients on local or systemic medications which cause dry eye.

QUESTIONNAIRE IN DRY EYE PATIENTS

1. History of discomfort, pain and duration
2. History of foreign body sensation
3. History of itching
4. History of foreign body sensation
5. History of blurred vision
6. History of redness of eyes
7. History of photophobia
8. History of difficulty in opening the eyes and history of watering of eyes
9. History of frequent blinking

Ophthalmic examination

1. Both visual acuity and best corrected visual acuity assessed using snellen chart
2. Detailed anterior segment examination using slit lamp, condition of lid, meibomian gland and corneal surface were noted
3. Subjects are asked to answer the questionnaire
4. Tear film evaluation done by schirmer test and tear film break up time
5. Intra ocular tension measured by applanation tonometer
6. Detailed fundus examination under full mydriasis attained by 1 % tropicamide and 5 % phenylephrine with both direct and indirect ophalmoscope.
7. Diabetic retinopathy graded according to ETDRS criteria
8. All findings were noted and the final ophthalmic status noted
9. At the end of the study period the data were given to an investigator for statistical analysis

STATISTICAL ANALYSIS

Table-1

Total no patients included in the study : 150

Sex Distribution

Gender	No of patients	Percent
Female	100	66.7
Male	50	33.3
Total	150	100.0

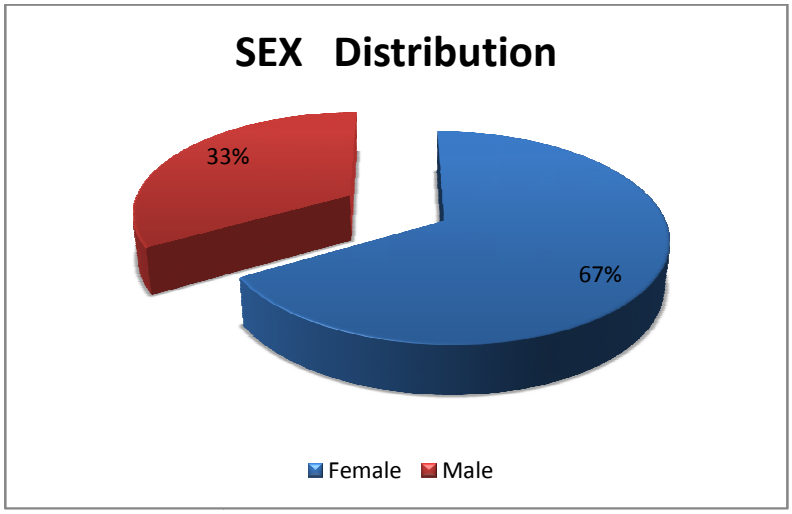


Table 1 shows among 150 patients,100 patients are females and 50 patients are males

Table-2

DURATION RANGE

Duration	Frequency	Percent
Less than 1 yr	6	4.0
1 to 5 yrs	92	61.3
6 to 10 yrs	38	25.3
11 to 20 yrs	14	9.3

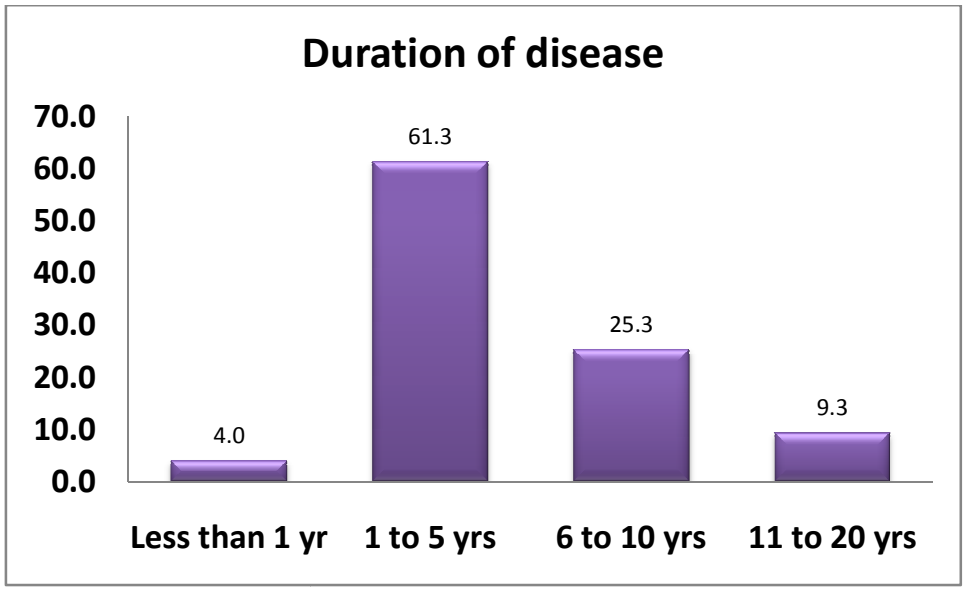


Table 2 shows, less than 1 year duration -4 %

1 to 5 years duration - 61.3 %

6 to 10 years duration – 25.3%

11 to 20 years duration -9.3%

Table -3

PREVALENCE OF DRY EYE SYMPTOMS

DE Symptoms	No of patients	Percent
Absent	97	64.7
Present	53	35.3
Total	150	100.0

DE- Dry Eye

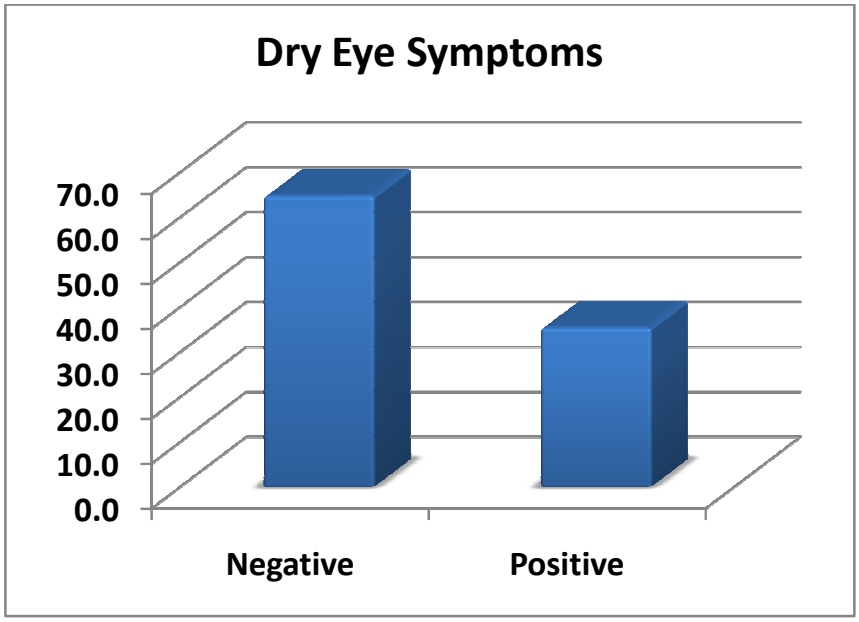
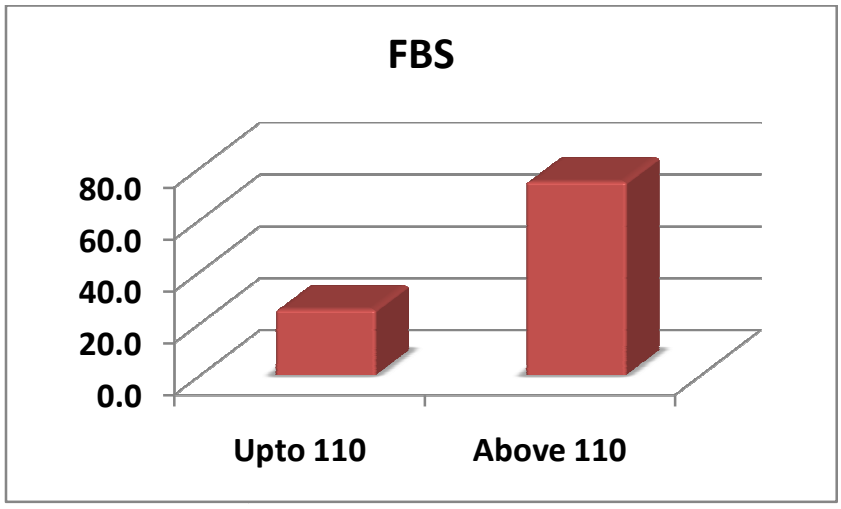


Table 3:shows 64.7% had dry eye symptoms. 35.3% had no dry eye symptoms

TABLE - 4

FASTING BLOOD SUGAR RANGE

FBS Range	Frequency	Percent
FBS < 110	38	25.3
FBS >110	112	74.7
Total	150	100.0

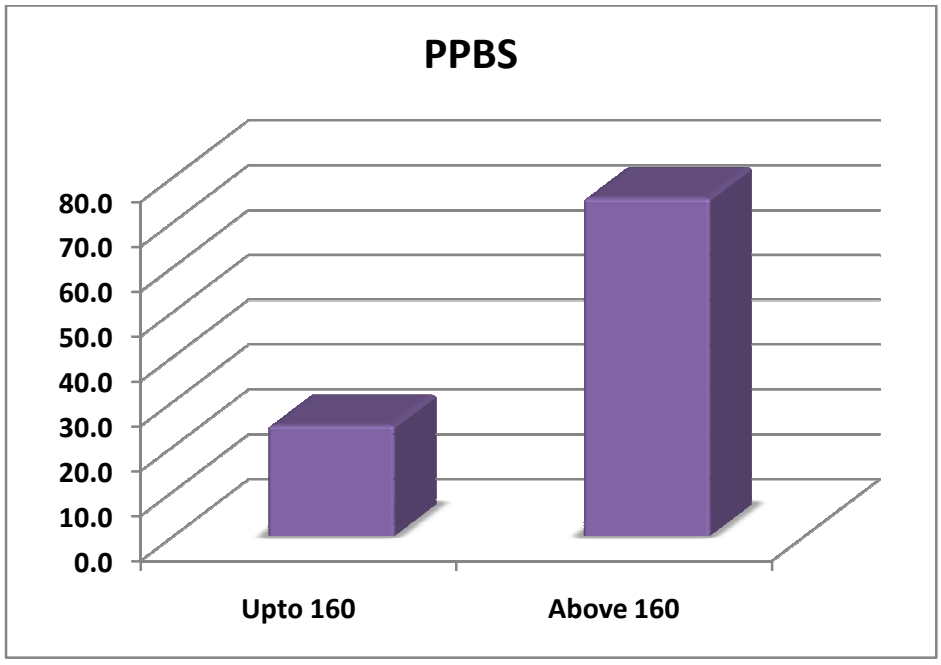


Tabe 4 shows 25.3% of patients had FBS less than 110mg/dl. 74.7% had FBS more than 110mg/dl

Table – 5

POST PRANDIAL BLOOD SUGAR RANGE

PPBS Range	Frequency	Percent
Upto 160	37	24.7
Above 160	113	75.3
Total	150	100.0

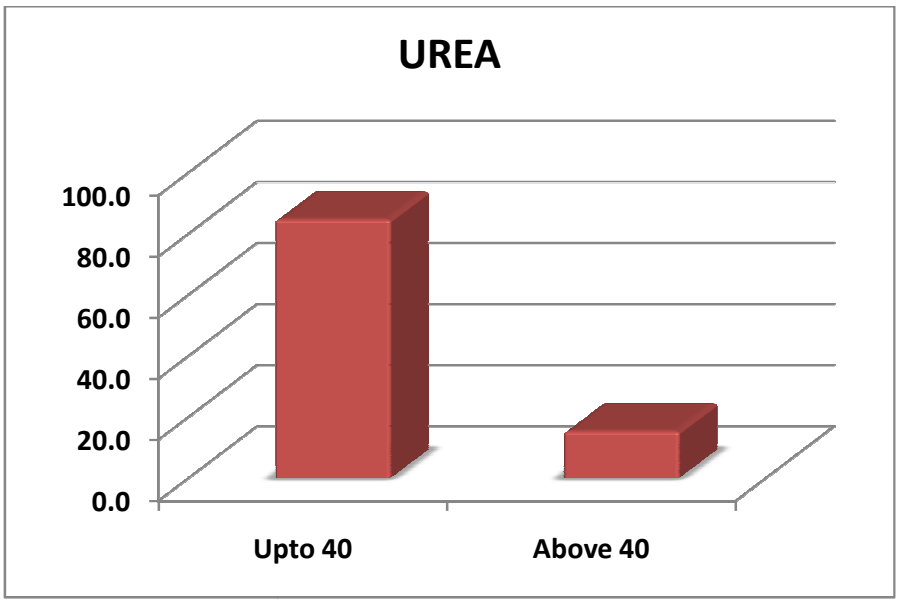


This table shows 37 patients had PPBS less than 160 mg/dl. 133 patients more than 160 mg/dl

Table -6

UREA RANGE

Urea Range	Frequency	Percent
Upto 40	127	84.7
More than 40	23	15.3
Total	150	100.0

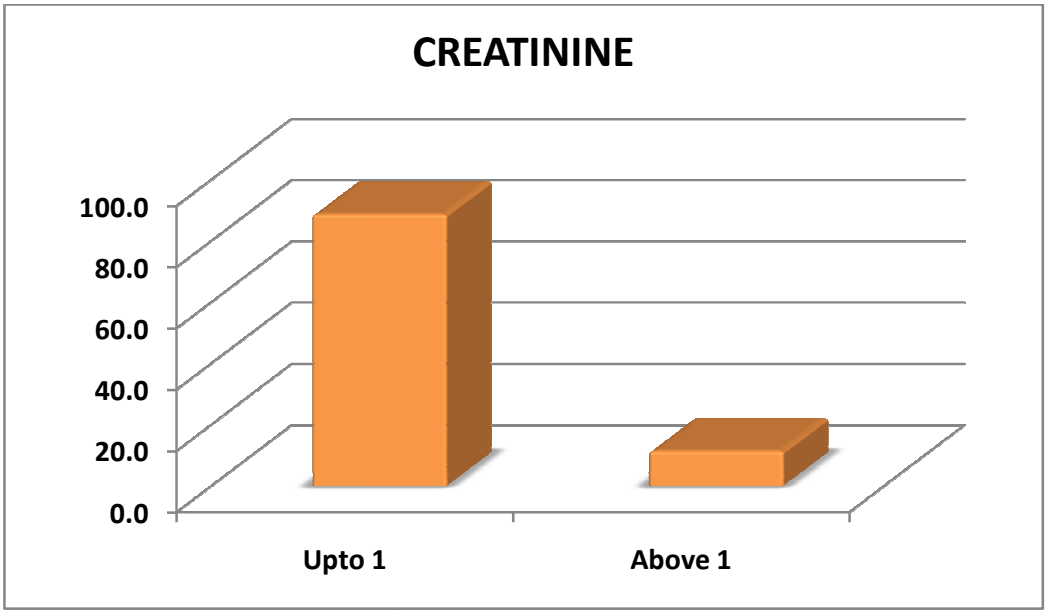


127 patients had urea level less than 40 mg /dl. 23 patients had urea level more than 40 mg/dl

Table -7

Creatinine Range	Frequency	Percent
Upto 1	133	88.7
More than 1	17	11.3
Total	150	100.0

Creatinine Range

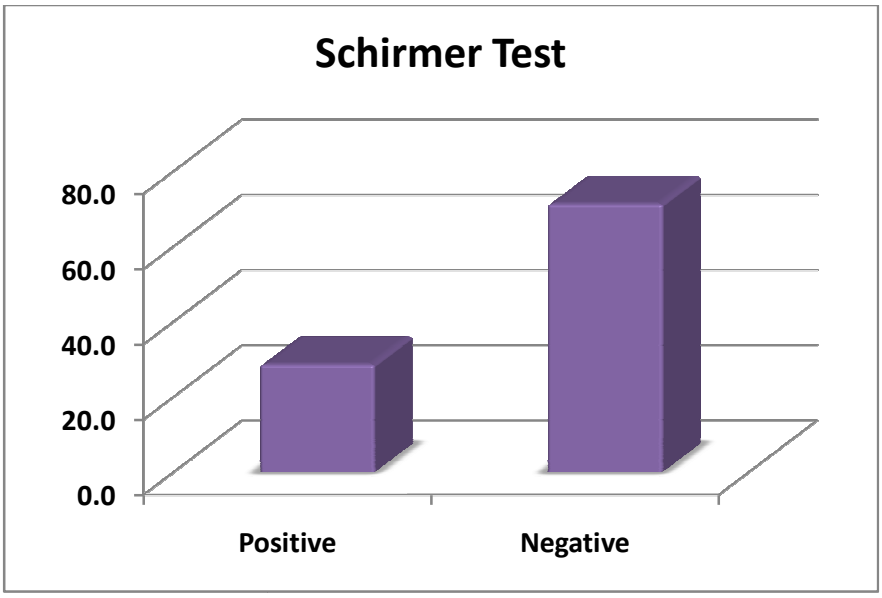


133 patients had Creatinine level less than 1 mg/dl. 17 patients had Creatinine more than 1 mg/dl

Table -8

SCHIRMER TEST

Schirmer	Frequency	Percent
Positive	43	28.7
Negative	107	71.3
Total	150	100.0

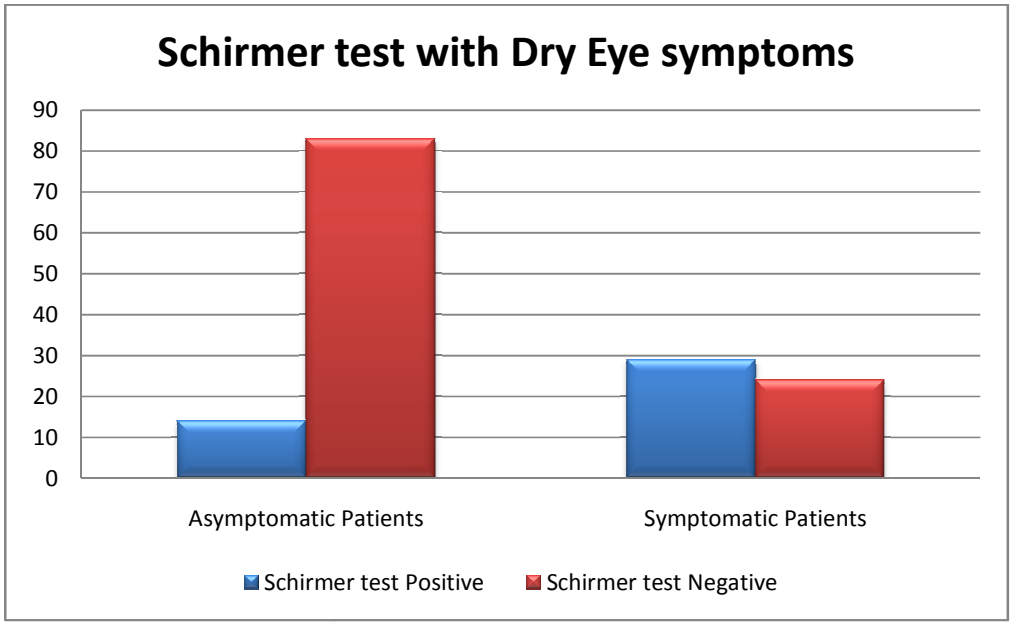


Among 150 patients, 43 patients were Schirmer test positive. 157 patients were negative for Schirmer test

Table -9

SCHIRMER TEST

	Asymptomatic patients	**Symptomatic patients**
Schirmer test Positive	14	29
Schirmer test Negative	83	24

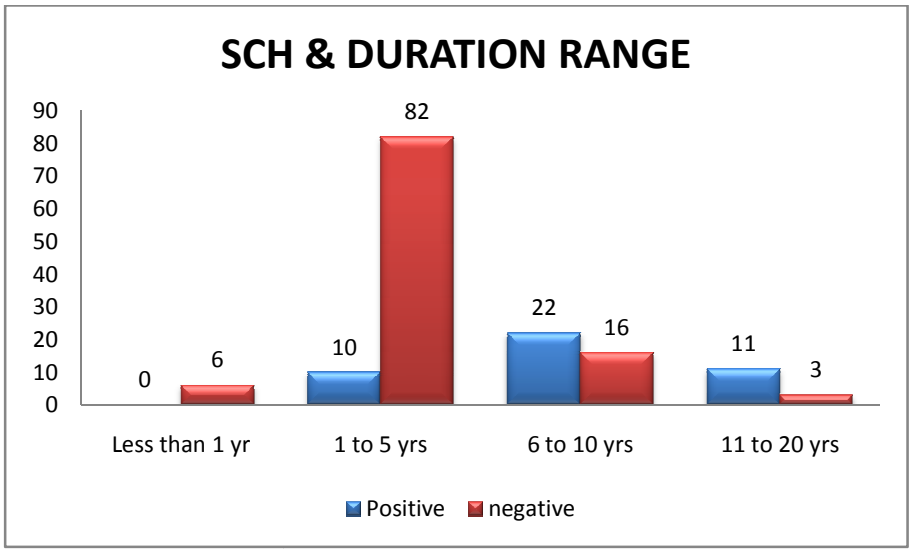


Among 53 symptom positive patients, 29 of them Schirmer test positive and 24 of them Schirmer test negative.

Table -10

SCHIRMER TEST AND DURATION OF DIABETES

Schirmer test	Less than 1 yr	1 to 5 yrs	6 to 10 yrs	11 to 20 yrs
Positive	0	10	22	11
negative	6	82	16	3

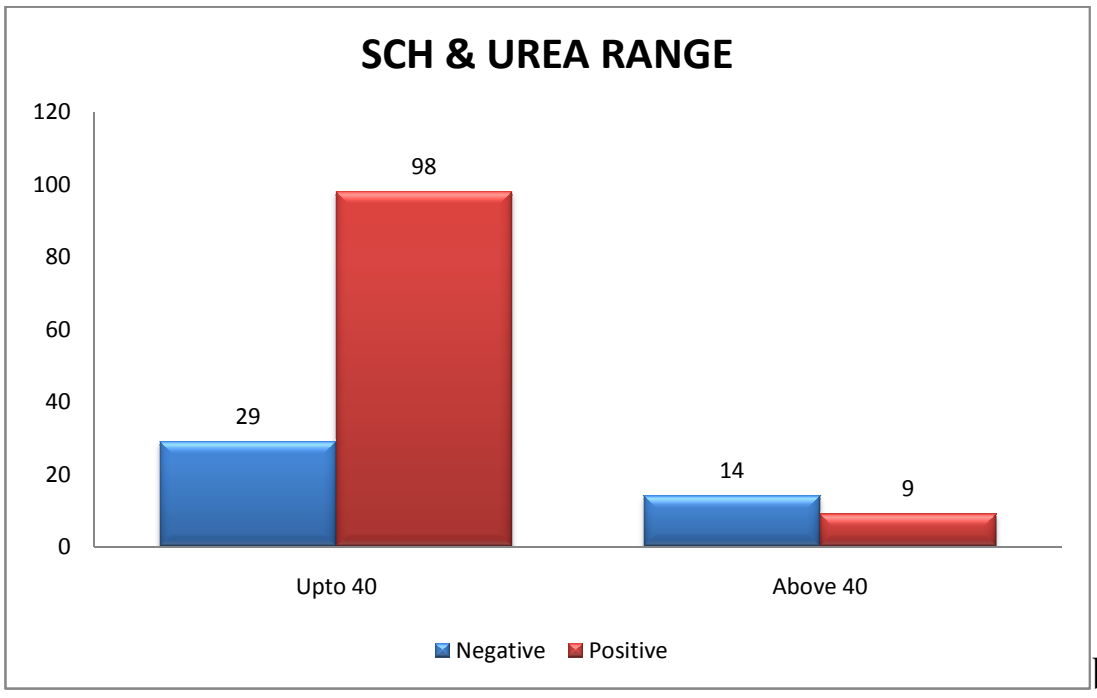


P value of above comparison was significant. Among 43 schirmer test positive patients 10 patients had 1 to 5 years of diabetes. 22 of them were 6 to 10 years of diabetes. 11 of them were 10 to 20 years of diabetes.

Table -11

UREA LEVEL AND SCHIRMER TEST

Schirmer test	Upto 40	Above 40
Positive	29	14
Negative	98	9



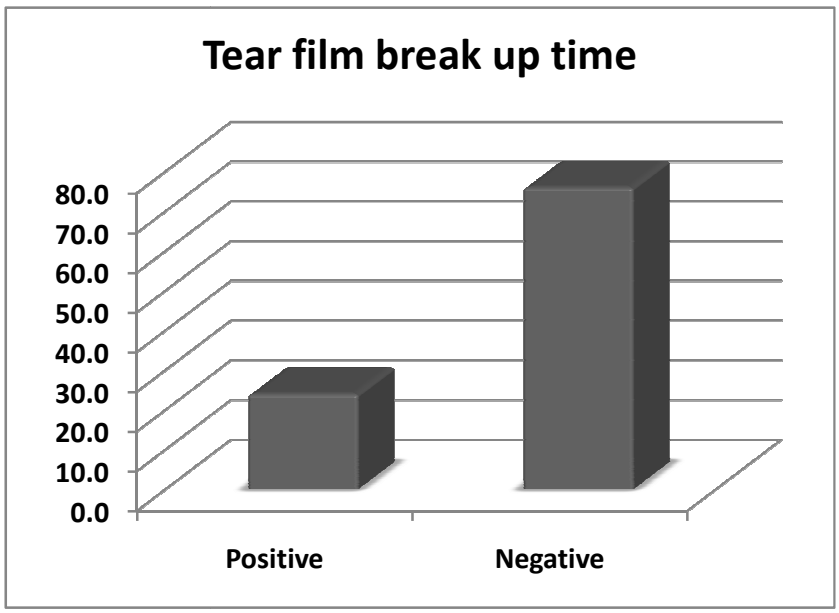
b

Among23 patients Schirmer test positive,14 patients had urea level more than 40 mg/dl.

Table -12

TEAR FILM BREAKUP TIME

TBUT	Frequency	Percent
Positive	36	24.0
Negative	114	76.0
Total	150	100.0

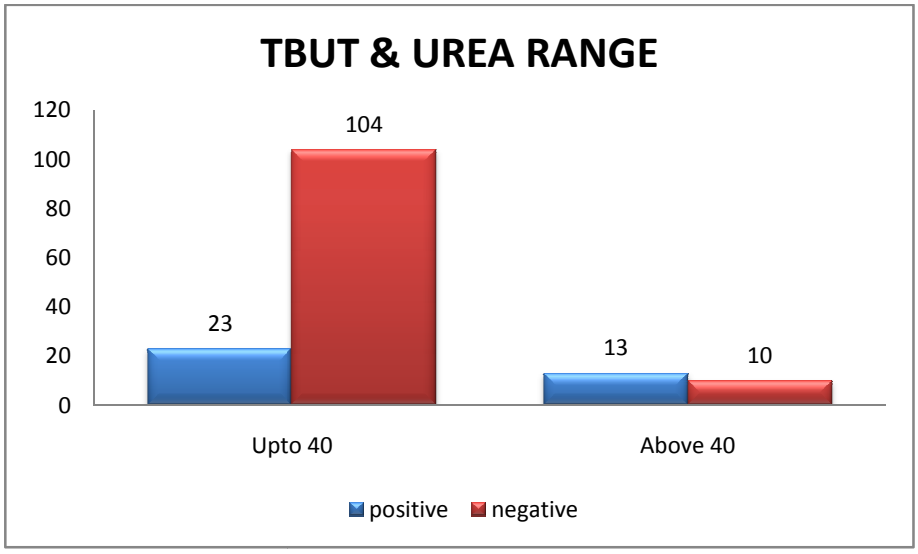


The p value of the above chart is < 0.01. Among 53 dry eye symptom positive patients,only 29 of them are Schirmer test positive

Table -13

UREA RANGE AND TBUT

TBUT	UREA Upto 40	UREA Above 40
positive	23	13
negative	104	10

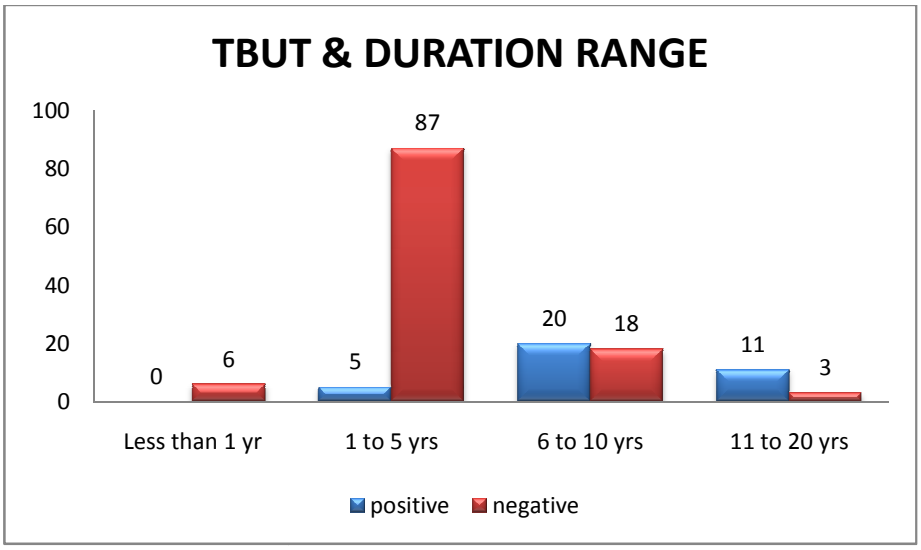


P value of the above correlation was significant. 36 of them TBUT test positive. Among 36 patients 23 of them urea level less than 40 mg/dl and 13 of them had urea level more than 40 mg/dl.

Table -14

TBUT AND DURATION OF DIABETES

Duration	Less than 1 yr	1 to 5 yrs	6 to 10 yrs	11 to 20 yrs
positive	0	5	20	11
negative	6	87	18	3

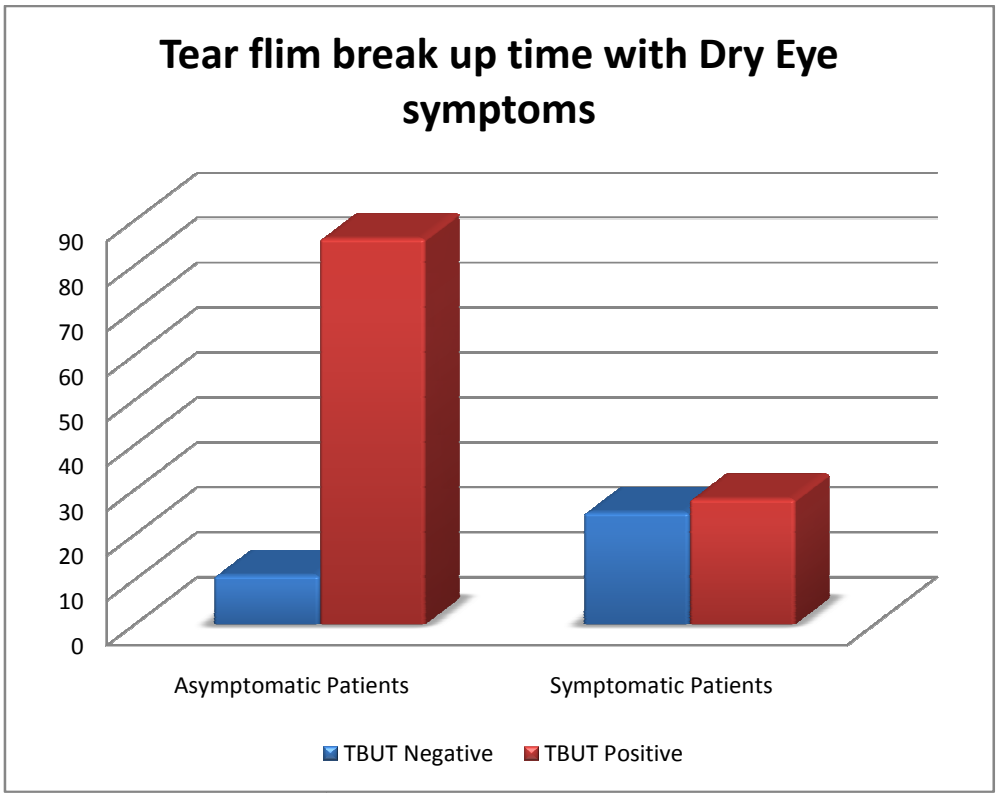


P value of the test was significant. Among 36 TBUT test positive patients, 5 of them were in 1 to 5 years duration of diabetes,20 of the were in 6 to 10 years duration and 11 of them were in 11 to 20 years duration of diabetes

Table -15

TBUT WITH DRY EYE SYMPTOMS

TBUT	Asymptomatic patients	Symptomatic patients
positive	11	25
negative	86	28

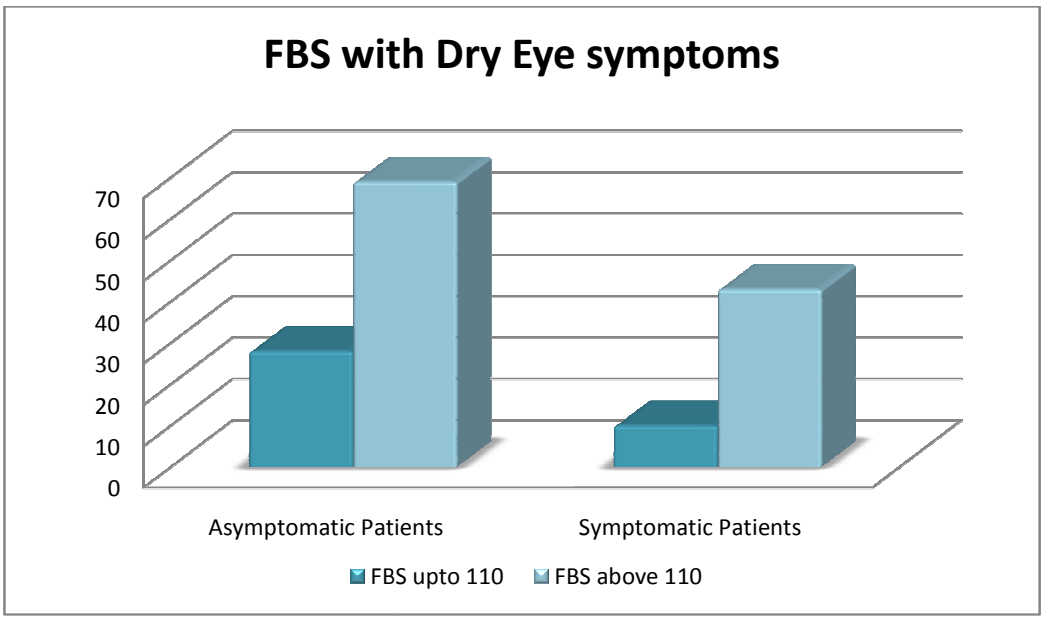


The p value of the above chart is < 0.01. Only25 of them were TBUT positive in symptomatic patients. 28 of them Schirmer test negative.

Table -16

FBS RANGE AND DRY EYE SYMPTOMS

FBS	Asymptomatic patients	Symptomatic patients
Upto 110	28	10
Above 110	69	43



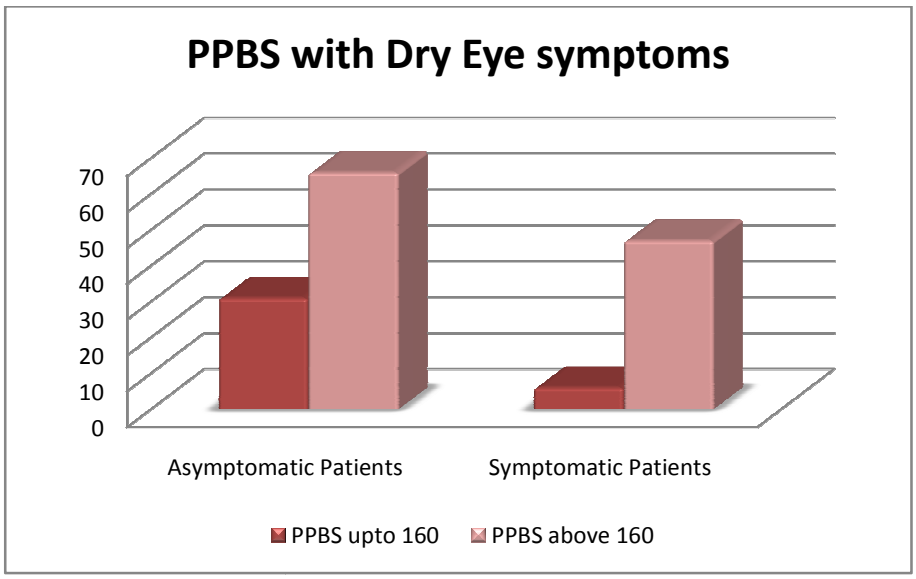
The p value of the above chart is 0.178, not significant.

In symptomatic patients only 10 of them had FBS less than 110 mg/dl. 43 of them had FBS more than 110 mg/dl

Table -17

PPBS AND DRY EYE

PPBS	Asymptomatic patients	Symptomatic patients
Upto 160	31	6
Above 160	66	47



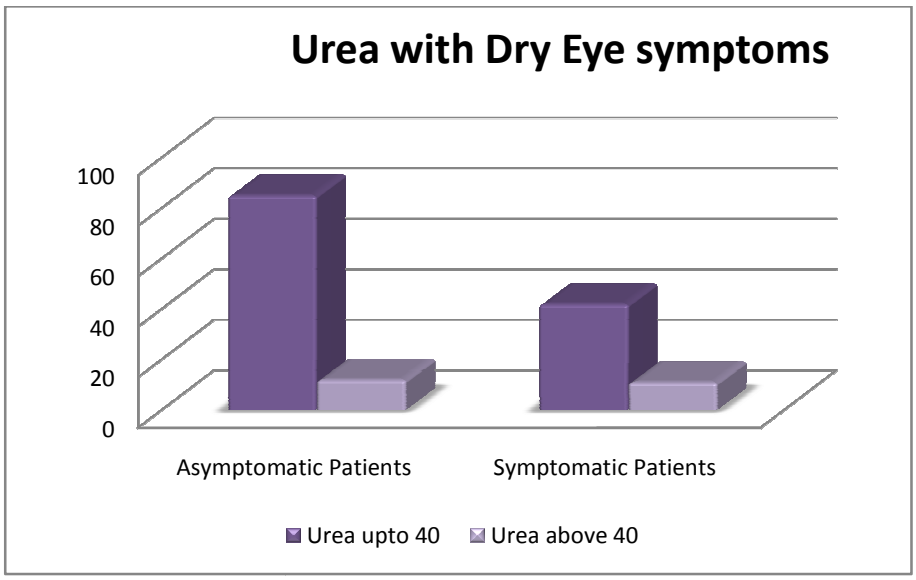
The p value of the above chart is < 0.05.

47 patients had PPBS more than 160 mg/dl and also had dry eye ssymptoms. Only 6 symptomatic patients had PPBS less than 160 mg/dl

Table -18

UREA RANGE AND DRY EYE

Urea	Asymptomatic patients	Symptomatic patients
Upto 40	85	42
Above 40	12	11

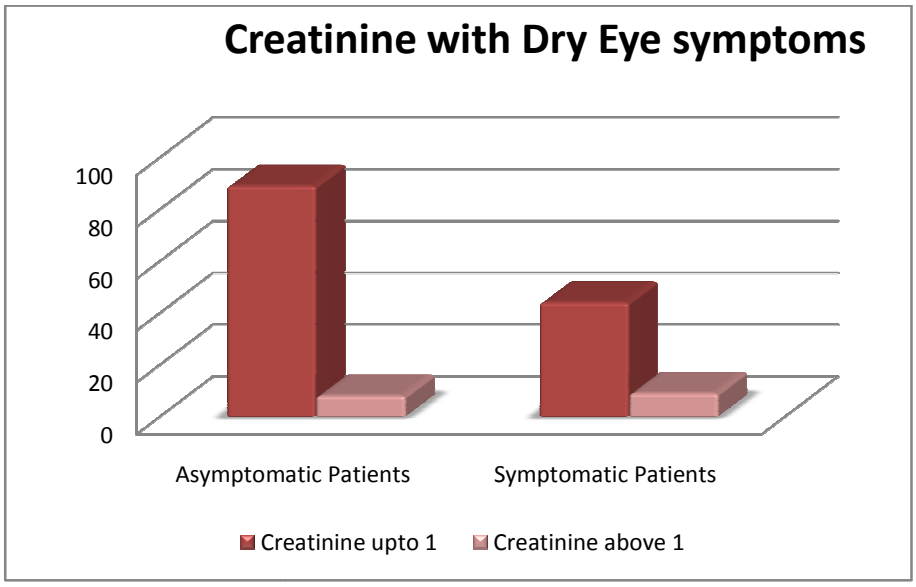


The p value of the above chart is 0.173, not significant. Among 53 dry eye symptom positive patients 42 of them had urea level less than 40 mg/dl

Table -19

CREATININE RANGE AND DRY EYE

Creatinine	A symptomatic patients	Symptomatic patients
Upto 1	89	44
Above 1	8	9



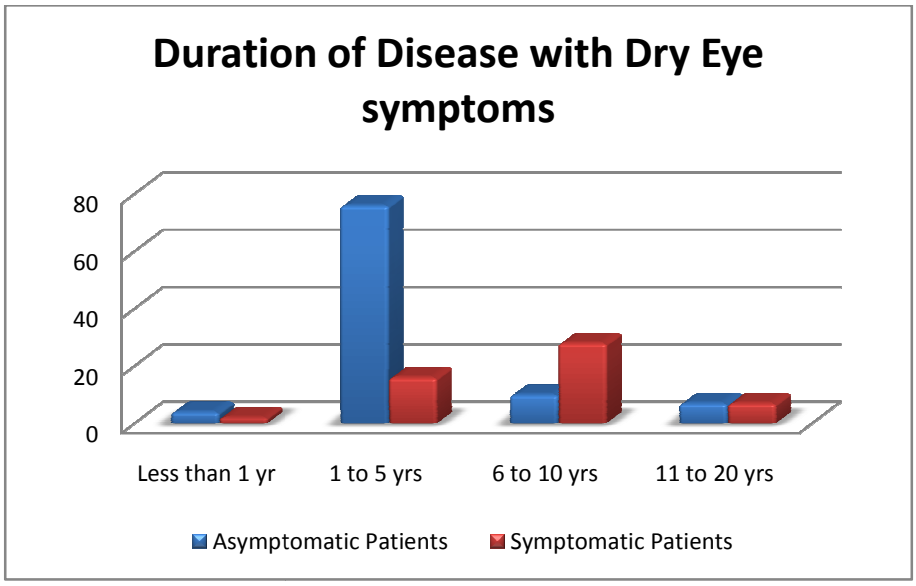
The p value of the above chart is 0.106, not significant.

Among 53 dry eye symptomatic patients, 44 of them had normal Creatinine level, only 9 of them had more than 1mg/dl of creatinine

Table -20

DURATION RANGE AND DRY EYE

Duration	Asymptomatic patients	Symptomatic patients
Less than 1 yr	4	2
1 to 5 yrs	76	16
6 to 10 yrs	10	28
11 to 20 yrs	7	7

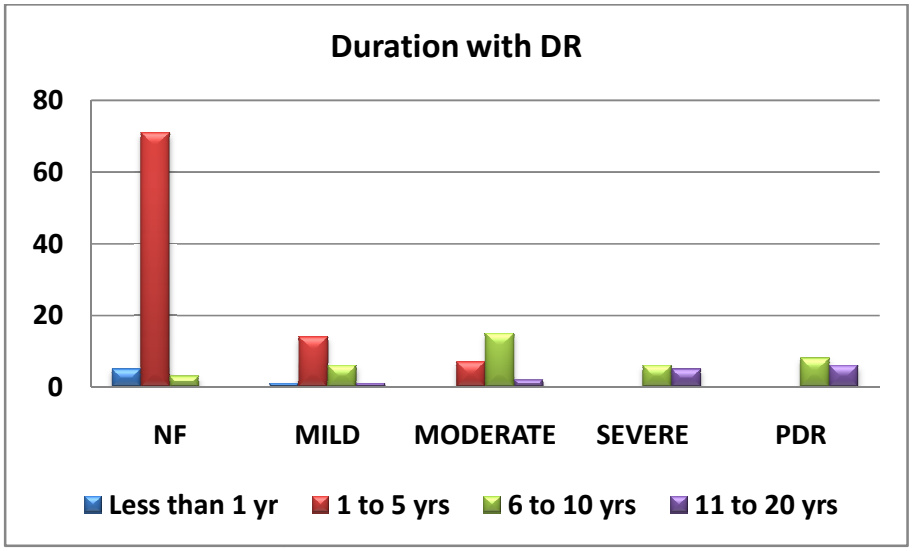


The p value of the above chart is < 0.01. Highly significant.Among 53 dry eye symptom positive patients.2 of them in less than 1 year duration.16 of them in 1 to 5 years duration.28 of them in 5 to 10 years duration,7 of them in 10 to 20 years duration.

Table -21

DURATION RANGE AND DR

Duration	NF	MILD NPDR	MODERATE NPDR	SEVERE NPDR	PDR
Less than 1 yr	5	1	0	0	0
1 to 5 yrs	71	14	7	0	0
6 to 10 yrs	3	6	15	6	8
11 to 20 yrs	0	1	2	5	6

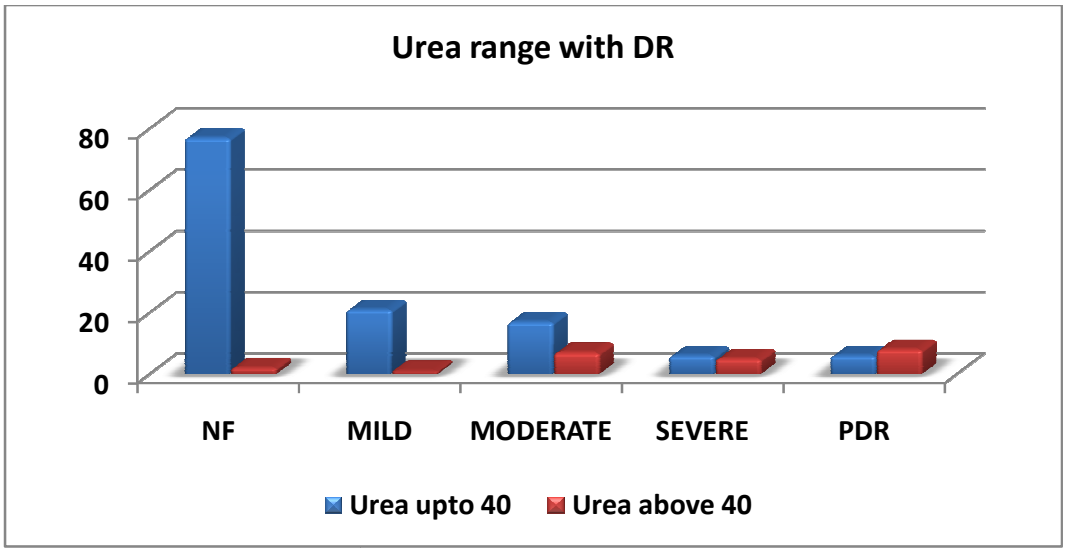


The p value of the above chart is significant.92 patients were in 1 to 5 years duration. Among 92 patients,71 of them had normal fundus, 14 of them mild NPDR and 7 of them had moderate NPDR.

Table -22

UREA RANGE AND DIABETIC RETINOPATHY

Urea	NF	MILD NPDR	MODERATE NPDR	SEVERE NPDR	PDR
Upto 40	77	21	17	6	6
Above 40	2	1	7	5	8

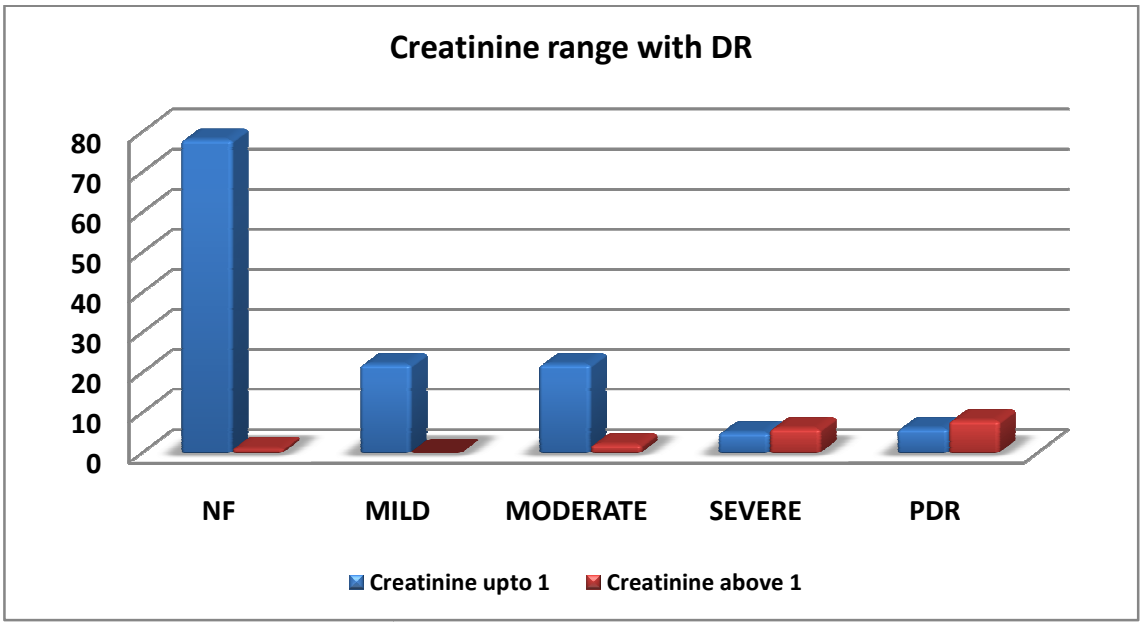


The p value of the above chart is <0.01, significant. 18 patients had urea more than 40 mg/dl. Among 18 patients 8 patients had PDR.

Table -23

CREATININE RANGE AND DR

Creatinine	NF	MILD NPDR	MODERATE NPDR	SEVERE NPDR	PDR
Upto 1	78	22	22	5	6
Above 1	1	0	2	6	8

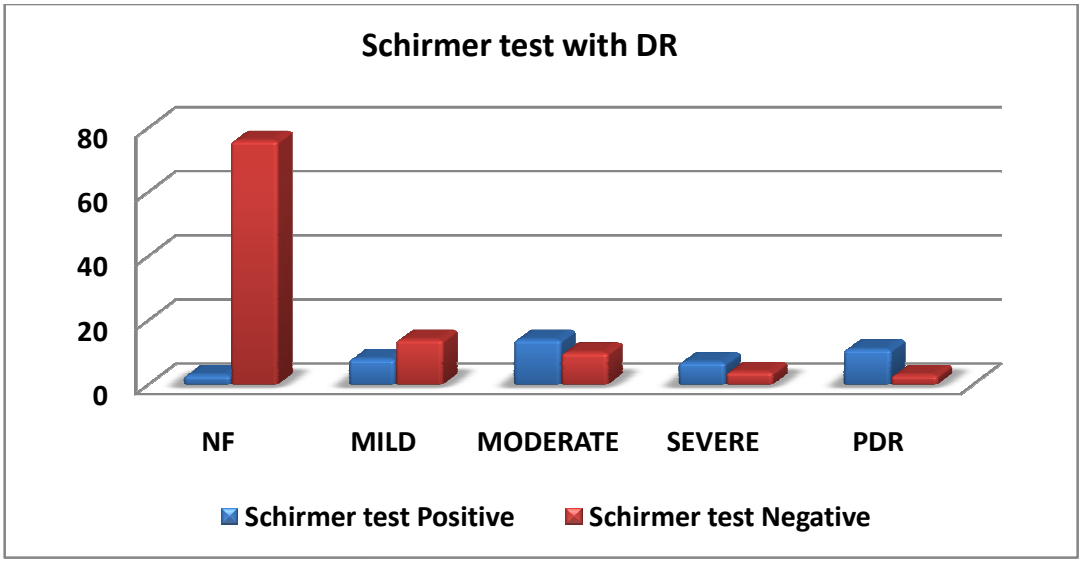


The p value of the above chart is <0.01, significant.17 patients had Creatinine level > 1 mg/dl. Among 17 patients 8 patients had PDR

Table -24

SCHIRMERS AND DR

Schirmer	NF	MILD	MODERATE	SEVERE	PDR
Positive	3	8	14	7	11
Negative	76	14	10	4	3

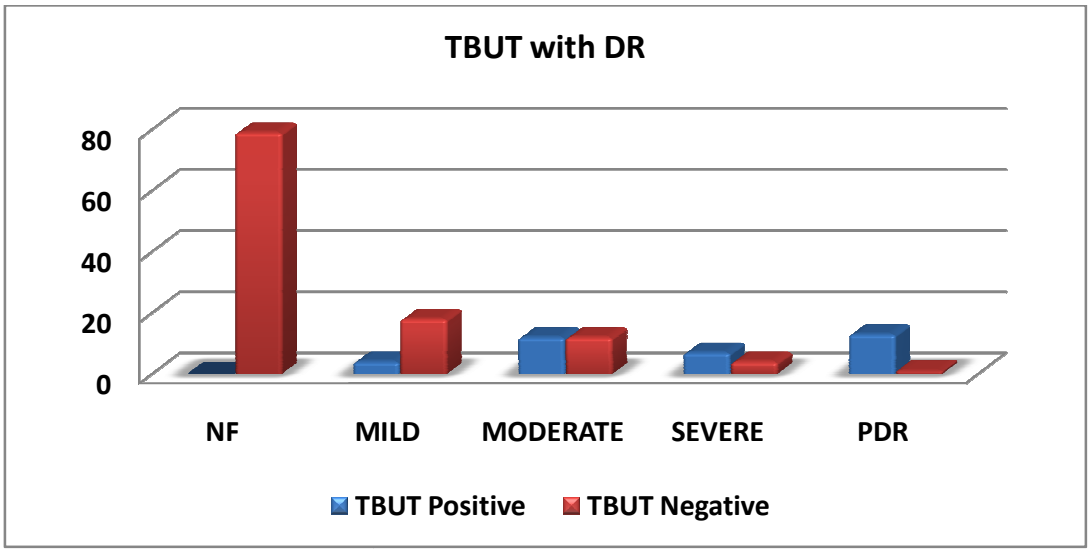


The p value of the above chart is <0.01, significant.43 patients are Schirmer test positive. Among 43 patients 3 of them NF, 8 of them mild NPDR, 14 of them moderate NPDR, 7 of them severe NPDR, 11 of them had PDR.

Table -25

TBUT AND DR

TBUT	NF	MILD	MODERATE	SEVERE	PDR
Positive	0	4	12	7	13
Negative	79	18	12	4	1

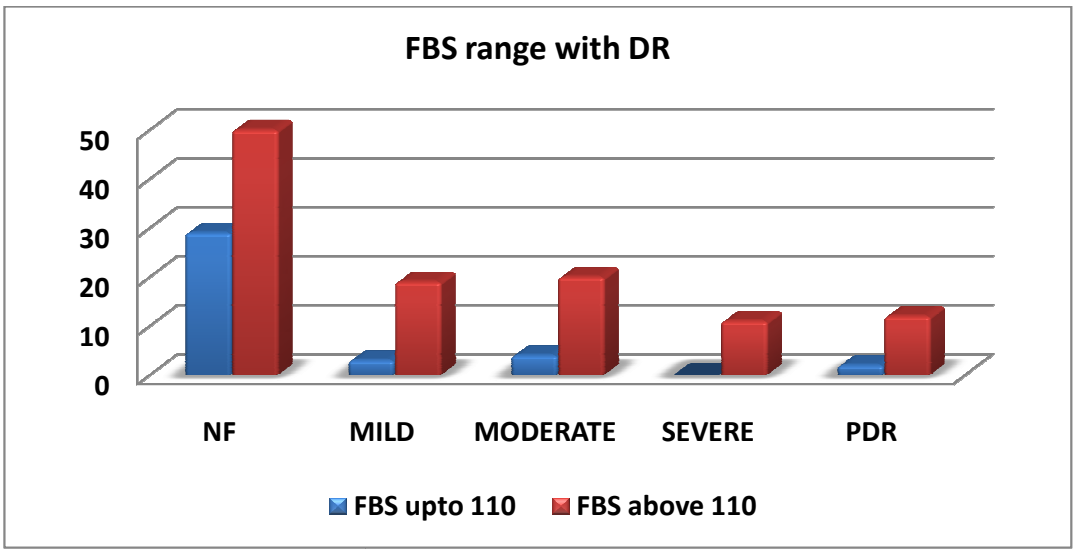


The p value of the above chart is <0.01, significant.36 patients were TBUT positive. Among 36 patients 4 patients had mild NPDR, 12 patients had moderate NPDR, 7 patients had severe NPDR and 13 of them had PDR.

Table -26

FBS RANGE WITH DR

FBS	NF	MILD NPDR	MODERATE NPDR	SEVERE NPDR	PDR
Upto 110	29	3	4	0	2
Above 110	50	19	20	11	12

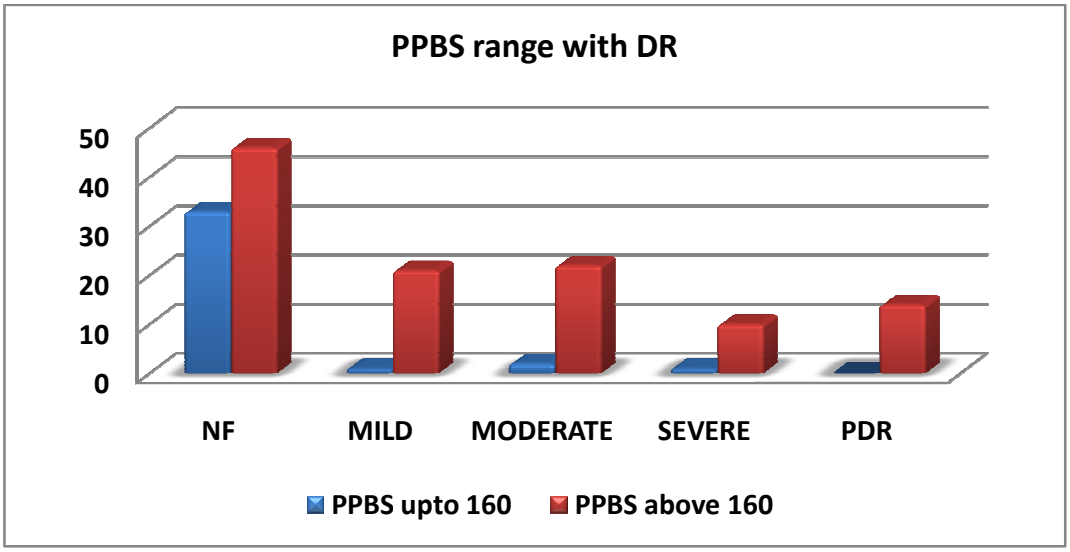


The p value of the above chart is <0.01, significant.112 patients had FBS more than 110 mg/dl. Among 112 patients 50 of them had normal fundus , 19 of them had mild NPDR, 20 of them had moderate NPDR, 11 of them had severe NPDR and 12 0f them had PDR

Table -27

PPBS RANGE WITH DR

PPBS	NF	MILD	MODERATE	SEVERE	PDR
upto 160	33	1	2	1	0
above 160	46	21	22	10	14

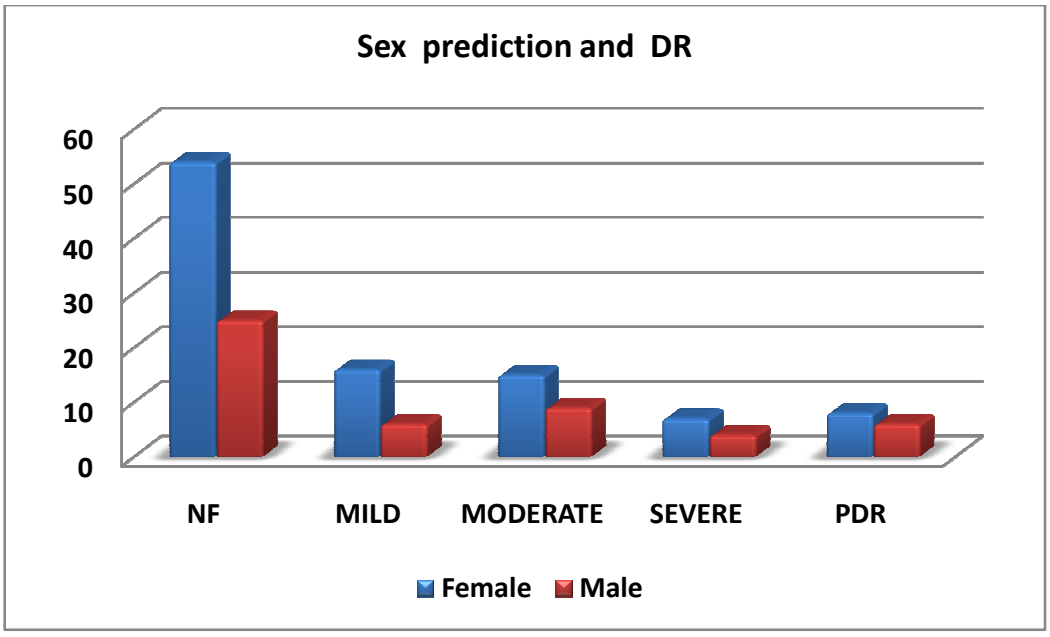


The p value of the above chart is <0.01, significant.113 patients had PPBS more than 160 mg/dl.46 of them had normal fundus, 21 of them had mild NPDR, 22 of them moderate NPDR, 10 of them had severe NPDR and 14 of them had PDR.

Table -28

SEX PREDICTION AND DR

	Female	Male
NF	54	25
MILD NPDR	16	6
MODERATE NPDR	15	9
SEVERE NPDR	7	4
PDR	8	6

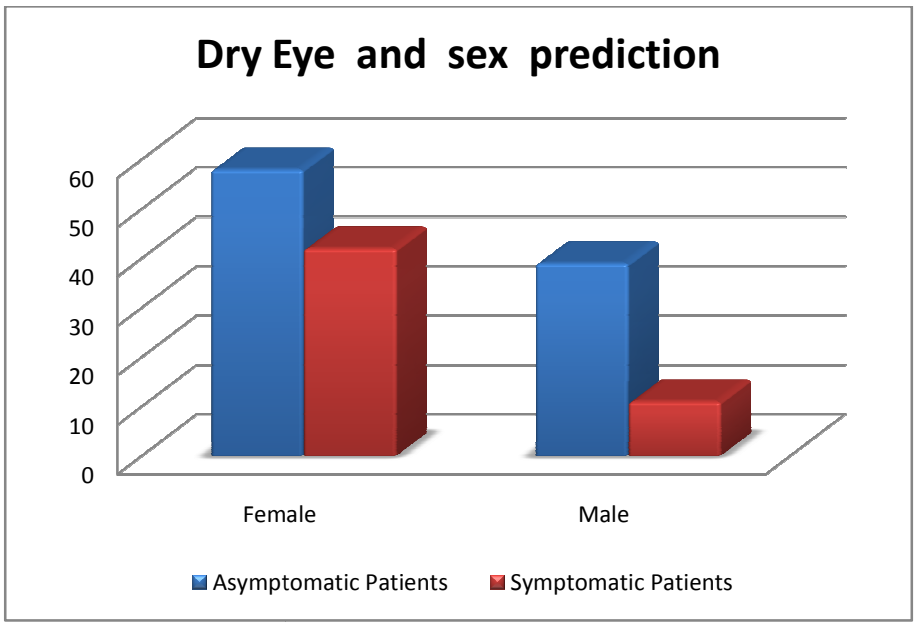


The p value of the above chart is not significant. Among 50 male patients 25 of them had normal fundus

Table -29

DRY EYE AND SEX PREDICTION

DE SYMPTOMS	Female	Male
Negative	58	39
Positive	42	11



The p value of the above chart is <0.01, significant.Among 53 symptomatic patients 42 of them female

DISCUSSION

In the present study 150 patients with type 2 diabetes mellitus were examined for dry eye and diabetic retinopathy manifestations and above manifestations are correlated with age, duration, control of diabetes and urea, creatinine level.

SEX DISTRIBUTION

Among 150 patients ,100 patients were females and 50 were males.

DURATION OF DIABETES MELLITUS IN OUR STUDY

- less than 1 year - 4%
- 1 to 5 years -61.3 %
- 5 to 10 years -25.3%
- 10 to 20 years -9.3%

GLYCEMIC CONTROL IN OUR STUDY

Fasting blood sugar

Less than 110 mg/dl -25. 3 %

More than 110 mg/dl-74.3 %

Post prandial blood sugar

Less than 160 mg/dl -24.7 %

More than 160 mg/dl – 75.3%

The study showed that 75% of them were not in good gycemic control

UREA AND CREATININE LEVEL IN OUR STUDY

Urea level

- Less than 40 mg/dl – 84. 7%
- .more than 40 mg/dl- 15.3%

Creatinine level

- Less than 1 mg /dl- 88.7 %
- More than 1 mg / dl- 11.3%

PREVALENCE OF DRY EYE SYMPTOMS IN OUR STUDY

- Symptomatic patients – 53 patients (35.3%)
- Asymptomatic patients – 147 patients (64.6%.)

Binder A et al study showed that 55 % of patients with diabetes had dry eye signs and symptoms at least at some of the time.

SCHIRMER TEST AMONG SYMPTOMATIC PATIENTS IN OUR STUDY

- Schirmer test positive – 28.6%
- Schirmer test negative – 71.3 %

Among 53 symptomatic patients 29 of them Schirmer test positive and 24 of them Schirmer negative. 14 of them shows Schirmer test positive in asymptomatic patient. Schirmer test is useful screening test for diagnosing

hyposecretion. But not for an adequate determination of tear production. Its sensitivity is between 10 % and 30 %.

TEARFILM BREAKUP TIME

- TBUT positive - 36 patients
- TBUT negative – 114 patients

Among symptom positive patients (53) TBUT is positive in 25 patients. The p value of above comparison is significant. Mannavit published prevalence of dry eye in diabetic patients utilising either schirmers test or TBUT.

SYMPTOMATIC PATIENTS AND SEX CORRELATION

42 females are dry eye symptom positive and only 11 males are symptom positive. P value is significant. Prevalence of dry eye is higher among advancing age, female sex, poor glycemic control, patients with diabetic retinopathy and high urea, creatinine level.

Moss and associates also reported a high incidence of dry eye among females 16 .7 % compared to males 11.4 %. Nepp and associates have showed that severity of keratoconjunctivitis sicca correlates with severity of diabetic retinopathy. Kaiserman and associates have reported that good blood sugar regulation is important for prevention of dry eye among diabetic patients. Goebbel had reported that Schirmer test reading is significantly reduced in diabetes. Kaiserman et al had also reported that

prevalence of dry eye increases with age . Age and sex plays an important role in dry eye. Good glycemic control is important for prevention and control of dry eye syndrome. Chronically elevated blood sugar lead to autonomic neuropathy affecting the lacrimal gland. It also affects the quality of tears by increasing amount of glucose in tears and disrupting normal chemical composition of tears which contributes to dry eye. Seifert et al told close monitoring of diabetic patients and good blood sugar regulation is important for the prevention of dry eye syndrome and retinopathy. Goebel study stated that Schirmer test and TBUT was significantly lower in diabetics.

CORRELATION OF SCHIRMER TEST AND TBUT WITH DIABETIC RETINOPATHY

In our study higher grades of diabetic retinopathy is associated with higher incidence of dry eye symptoms and the P value of the test is significant.

DURATION AND DIABETIC RETINOPATHY

Among 150 type 2 diabetes mellitus patients

- normal fundus -79 patients
- mild NPDR – 22 patients
- moderate NPDR- 24 patients

- severe NPDR – 11 patients
- PDR -14 patients

Correlating the duration of diabetes with diabetic retinopathy In 11 to 20 years duration of diabetes – all of them had diabetic retinopathy of varying severity. P value of the above correlation is significant.

CORRELATING UREA LEVEL WITH DR

Among 150 patients,

- Urea level > 40 mg/dl -23 patients
- Urea level < 40 mg/dl- 127 patients

23 patients had urea level more than 40 mg/dl. In 23 patients only 2 of them had normal fundus. 1 patient had mild NPDR. 7 of them had moderate NPDR. 5 of them had severe NPDR. 8 of them had PDR.P value if the above test is significant.

CORRELATING CREATININE LEVEL WITH DR

Creatinine< 1 mg/dl – 17 patients

Creatinine.> 1 mg/dl – 133 patients

17 of them had more than 1mg/dl . only one patient had normal fundus. Remaining patients with elevated creatinine level had some degree of DR.P value of the above correlation is significant.

GLYCEMIC CONTROL AND DR

38 patients among 150 patients had fasting blood sugar less than 110mg/dl. Remaining patients(112) had more than 110 mg/dl. 50patients had normal fundus in more than 110 FBS group. 72 of them had some degrees of diabetic retinopathy, Microvascular complication ,develops in nearly all patients with type 1 diabetes and in more than 77 % of those with type 2 diabetes who survives for over 20 years with the disease. Vision threatening retinopathy is rare in type 1 diabetes in the first 3 to 5 years of diabetes or before puberty. After 2 decades nearly all patients with type 1 DM develop retinopathy. 21 % of type 2 diabetes patients have retinopathy at the time of first diagnosis of diabetes.

DR was graded according to ETDRS criteria. Segata et al study showed that prevalence of DR in late onset diabetic patients was lower than younger onset diabetes patients. Patients with DR had a significantly higher mean duration of diabetes than those patients without DR. Rema et al showed that lower grades of DR was more common in women and higher grades of DR was more common in men. Klein et al showed a significant association between the prevalence of DR and diabetes association.

DURATION OF DIABETES

It is probably the strongest predictor for the development and progression of retinopathy.

GLYCEMIC CONTROL

UKPDS demonstrated that improved blood glucose control reduced the risk of developing retinopathy, nephropathy and possibly reducing neuropathy.

SEX

Pradeep et al observed male sex associated with the presence of any DR but not its severity. But in our study p value of gender correlation with diabetic retinopathy is not significant

SUMMARY

STUDY RESULTS

1 61.3% of diabetic patients were in 1 to 5 years duration.

2 35.3% of diabetic patients had dry eye symptoms.

3 Among symptomatic patients , 28.7% Schirmer test positive and 24 % of them TBUT positive.

4 Dry eye symptoms more prevalent in female gender.

5 The prevalence of dry eye increases in the duration of the disease had poor glycemic control (74 %).

6 Urea and Creatinine level is high in 15.3% and 11.3 % respectively.

7 The prevalence of diabetic retinopathy was 47 %.

8 The prevalence of diabetic retinopathy also increases with longer duration of disease , poor glycemic control and high urea and Creatinine level.

Diabetes mellitus is one of the leading causes of blindness in twenty to seventy four year old individuals. Cataract and retinopathy are known complications. Dry eyes are more prevalent in diabetic individuals. The ocular surface disease in diabetic individuals confirmed by Schirmer test and tear film breakup time. Schirmer test is gold standard in diagnosing dry eye. Diabetic patients had increased prevalence of persistant epithelial defect,corneal ulceration and dry eye.

Early detection of dry eye and diabetic retinopathy prevents vision threatening complications like corneal opacity, diabetic macular edema, tractional retinal detachment and vitreous haemorrhage.

CONCLUSION

Dry eye syndrome is a definite entity , that occurs in diabetic patients, more so, when the duration of diabetes is longer, poor glycemic control, high urea, Creatinine level associated with DR, irrespective of severity.

Hence it is mandatory to look for ocular surface abnormality in diabetic patients , as it can interfere with vision further.

Schirmer test and TBUT test are easy to perform and easily available. They can be rountinely done in all diabetic patients, in order to diagnose dry eye in early stage, so that prompt treatment can be initiated.

As dry eye symptoms and DR have a correlation, it is important to rule out DR in symptomatic dry eye patients and vice versa. Since poor glycemic control and elevated urea, Creatinine levels have been associated with increased incidence of dry eye, it is important to look for the same, even in asymptomatic patients with altered biochemical parameters. Apart from ophthalmoscopy, Schirmer test and TBUT should be compulsorily done in all diabetic patients.

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PROFORMA

Name of the patient

Age

Sex

Op no

Diabetes history

Age of Onset

Duration

Treatment history

Biochemical parameter - FBS, PPBS, Urea, Creatinine

Chief complaints

History of chief complaints

Dry eyequestionarre

Past history

History of hypertension/ rheumatoid arthritis/ systemic illness

History of past ocular surgeries

History of contact lens wear

Family history

General examination

Pulse, blood pressure

Ocular examination

BCVA

Lids

Conjunctiva

Cornea

Anterior chamber

Pupil

Lens

Extra ocular movements

Above examination findings confirmed with slit lamp examination

Intra ocular tension by applanation tonometer

Fundus examination

Pupil dilated with 1% tropicamide and 5% phenylephrine

After full dilatation of pupil , fundus examination was done with direct ophthalmoscopy and the above findings were confirmed with indirect ophthalmoscopy and 90 D examination.

Key to Master Chart

Sno – Serial Number

OPno – Out Patient Number

DM – Diabetes Mellitus

Duration Y & M – Duration Years and Months

FBS – Fasting Blood Sugar

PPBS – Post Prandial Blood Sugar

NF – Normal Fundus

Mi-NPDR – Mild Non Proliferative Diabetic Retinopathy

Mo-NPDR - Moderate Non Proliferative Diabetic Retinopathy

S-NPDR – Severe Non Proliferative Diabetic Retinopathy

PDR - Proliferative Diabetic Retinopathy

DE-SP – Dry Eye Symptoms Present

DE-SA – Dry Eye Symptoms Absent

SCH – SCHIRMER Test

TBUT- Tear Film Break Up Time

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் கண் மருத்துவத் துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவர் மேற்கொள்ளும் **"நீரழிவு நோயினால் கண்ணீர் சுரப்பு குறைவதையும், கண்ணின் நரம்பு விழித்திரையான ரெட்டினா பாதிப்பு அடைவதையும், நீரழிவு நோயின் காலவரையறை மற்றும் யூரியா கிரியேட்டினின் அளவோடு ஒப்பிடுதல்"** குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :

CONSENT FORM

Here by I volunteer and to participate in this study "TO STUDY THE PREVALANCE OF DIABETIC RETINOPATHY AND DRY EYE IN DIABETES MELLITUS AND COMPARING WITH DURATION AND UREA CREATININE LEVEL“ was fully explained about the nature of this study by the doctor, knowing which I Mr/Mrs_____ fully consent to volunteer in this study.

Date:

Place: Signature of the volunteer

Signature of Witness

Sno	Name	Age	Sex	Opno	DM Type	Duration Y & M		FBS	PPBS	Urea	Creatinine	NF	Mi-NPDR	Mo-NPDR	S-NPDR	PDR	DE-SP	DE-SA	SCH>10mm	SCH<10mm	TBUT>10S	TBUT<10S
1	Vijayalakshmi	51	F	5686	2	3	0	120	161	28	0.8	+	-	-	-	-	-	+	+	-	+	-
2	Jyothi	42	F	11711	2	3	0	121	237	24	0.6	-	+	-	-	-	-	+	+	-	+	-
3	Shobana	60	F	11704	2	8	0	112	160	38	0.7	-	-	-	+	-	+	-	-	+	-	+
4	Panner Selvam	60	M	11717	2	7	0	118	172	26	0.4	-	-	+	-	-	+	-	-	+	+	-
5	Thavamani	62	M	12082	2	15	0	103	183	52	2	-	-	-	-	+	-	+	-	+	-	+
6	Jagathambal	46	F	101401	2	0	2	129	289	25	0.6	-	+	-	-	-	-	+	+	-	+	-
7	Palaniammal	70	F	10751	2	7	0	137	253	29	0.6	-	-	+	-	-	+	-	-	+	-	+
8	Krishnaveni	60	F	11981	2	5	0	90	139	26	0.6	-	+	-	-	-	+	-	-	+	+	-
9	Unslima	52	F	12189	2	10	0	217	324	38	0.6	-	-	-	-	+	-	+	-	+	-	+
10	Dowbath	35	F	10748	2	1	0	93	160	28	0.6	+	-	-	-	-	+	-	+	-	+	-
11	Kannammal	60	F	13074	2	4	0	98	153	25	0.7	-	-	+	-	-	-	+	-	+	-	+
12	Thilagavathi	55	F	10175	2	10	0	200	260	40	4.1	-	-	-	-	+	-	+	-	+	-	+
13	Nanjurabai	48	F	5314	2	2	0	105	134	21	0.6	+	-	-	-	-	+	-	+	-	+	-
14	Bakyalakshmi	48	F	10671	2	0	3	105	270	32	0.9	+	-	-	-	-	+	-	+	-	+	-
15	RoseMary	53	F	10557	2	6	6	225	273	21	0.7	-	+	-	-	-	+	-	-	+	+	-
16	Kannaki	45	F	10175	2	1	0	160	195	20	0.4	+	-	-	-	-	-	+	+	-	+	-
17	Krishnaveni	55	F	14068	2	6	0	100	187	24	0.4	-	-	+	-	-	+	-	-	+	+	-
18	IlliyaJyothi	39	F	14052	2	2	0	132	196	21	0.6	+	-	-	-	-	-	+	+	-	+	-
19	Karambal	68	F	13983	2	10	0	217	324	68	0.6	-	-	-	-	+	+	-	-	+	-	+
20	Amsal	58	F	13974	2	16	0	217	324	78	0.6	-	-	-	-	+	-	+	-	+	-	+
21	Krishnaveni	39	F	13827	2	0	10	210	280	29	0.9	+	-	-	-	-	-	+	+	-	+	-
22	Mthammal	46	F	13569	2	4	0	102	118	28	0.4	+	-	-	-	-	-	+	+	-	+	-
23	Sarramma	56	F	13514	2	6	0	102	180	34	0.8	-	-	+	-	-	+	-	+	-	-	+
24	Gandhimathi	52	F	13452	2	12	0	162	260	36	0.8	-	-	+	-	-	+	-	-	+	-	+
25	Raja	26	M	13242	2	3	0	110	140	26	0.7	+	-	-	-	-	-	+	+	-	+	-
26	Sagunthala	49	F	13164	2	10	0	160	190	38	0.7	-	-	-	+	-	-	+	-	+	-	+
27	Lakshmanan	67	M	12965	2	5	0	160	172	24	0.6	-	-	+	-	-	+	-	+	-	+	-
28	Malliya	75	M	12926	2	3	0	110	162	28	0.6	+	-	-	-	-	-	+	+	-	+	-
29	Mahabi	60	M	12476	2	7	0	110	170	26	0.7	-	+	-	-	-	-	+	+	-	+	-
30	Mangalam	70	F	12576	2	10	0	177	283	42	0.8	-	-	+	-	-	+	-	-	+	-	+
31	Ram	40	M	11366	2	2	0	140	160	26	0.7	+	-	-	-	-	-	+	+	-	+	-
32	Pandiyammal	55	F	10866	2	10	0	172	210	24	0.8	-	+	-	-	-	+	-	+	-	+	-
33	Subbulakshmi	38	F	10371	2	2	0	140	182	26	0.4	+	-	-	-	-	-	+	+	-	+	-
34	Manniyammal	40	F	30272	2	2	0	110	140	28	0.6	+	-	-	-	-	-	+	+	-	+	-
35	kamala	62	F	10740	2	1	0	140	180	31	0.8	+	-	-	-	-	+	-	+	-	+	-
36	Jyothi	42	F	10721	2	2	0	116	172	28	0.6	+	-	-	-	-	-	+	+	-	+	-
37	Shobana	52	F	10717	2	12	0	112	162	46	1.7	-	-	-	+	-	+	-	-	+	-	+
38	Slochana	67	F	10712	2	4	0	160	210	42	0.8	-	+	-	-	-	+	-	-	+	-	+
39	Devi	50	F	10586	2	2	0	171	221	36	0.4	-	+	-	-	-	+	-	-	+	+	-
40	Suleka	51	F	10550	2	10	0	161	192	48	0.6	+	-	-	-	-	-	+	+	-	+	-
41	Arul Mozhi	46	F	10477	2	2	1	140	160	28	0.7	+	-	-	-	-	-	+	+	-	+	-
42	Sajina	40	F	10469	2	2	0	160	180	26	0.7	+	-	-	-	-	-	+	+	-	+	-
43	Padhama	49	F	10349	2	2	0	160	180	26	0.4	+	-	-	-	-	-	+	+	-	+	-
44	Dhanalakshmi	55	F	10286	2	1	0	120	160	18	0.7	+	-	-	-	-	-	+	+	-	+	-
45	Dandush	55	F	10229	2	3	0	160	182	24	0.6	+	-	-	-	-	-	+	+	-	+	-
46	Sabeema	62	F	10216	2	10	0	210	282	40	0.7	-	-	-	-	+	+	-	+	-	-	+
47	Senbagam	45	F	10150	2	3	0	161	182	24	0.6	+	-	-	-	-	-	-	+	-		-

48	Jyothi	44	F	9998	2	2	0	140	162	6	0.7	+	-	-	-	-	-	+	-	+	+	-
49	Dhanam	42	F	8999	2	1	0	120	160	28	0.8	+	-	-	-	-	-	+	-	+	+	-
50	Sarathambal	60	F	9833	2	9	0	160	410	43	0.9	-	-	+	-	-	+	-	-	+	-	+
51	Pappathi	57	F	9871	2	4	0	120	160	24	0.8	+	-	-	-	-	-	+	+	-	+	-
52	Bakyam	56	F	9800	2	3	0	200	242	31	0.7	+	-	-	-	-	-	+	+	-	+	-
53	Lakshmi	60	F	9793	2	12	0	200	260	32	0.7	-	+	-	-	-	+	-	-	+	-	+
54	Pappu	58	F	9722	2	2	0	110	160	31	0.8	+	-	-	-	-	-	+	+	-	+	-
55	Rajeshwari	52	F	9766	2	5	0	110	140	26	0.8	+	-	-	-	-	-	+	+	-	+	-
56	Sharadha	58	F	9542	2	2	0	110	140	28	0.6	+	-	-	-	-	-	+	+	-	+	-
57	Manjula	60	f	9701	2	10	0	200	260	38	0.4	-	-	+	-	-	+	-	+	-	-	+
58	Arjunan	64	M	9670	2	20	0	300	342	48	2	-	-	-	+	-	-	+	-	+	-	+
59	Ramathal	58	F	9490	2	3	0	143	180	24	0.6	-	+	-	-	-	-	+	+	-	+	-
60	Sabeer	44	M	8306	2	2	0	120	146	28	0.7	+	-	-	-	-	-	+	+	-	+	-
61	Ramachandran	65	M	8615	2	10	0	240	292	141	7.6	-	-	+	-	-	-	+	+	-	+	-
62	Veeran	60	M	8399	2	1	6	120	188	28	0.6	-	-	+	-	-	-	+	+	-	+	-
63	Hari	61	M	8354	2	3	0	140	180	27	0.8	+	-	-	-	-	-	+	+	-	+	-
64	Aruchamy	56	M	8463	2	2	0	120	160	28	0.7	+	-	-	-	-	-	+	+	-	+	-
65	Palani	70	M	8358	2	3	0	112	168	24	0.8	+	-	-	-	-	-	+	+	-	+	-
66	Arathal	60	F	8072	2	10	0	182	242	27	0.6	-	-	+	-	-	+	-	-	+	-	+
67	Manjula	47	F	7520	2	2	0	160	212	34	0.7	+	-	-	-	-	-	+	+	-	+	-
68	filomina	57	f	6445	2	4	0	140	180	27	0.6	+	-	-	-	-	-	+	+	-	+	-
69	Vijayalakshmi	51	F	5688	2	4	0	150	200	32	0.7	-	+	-	-	-	+	-	-	+	-	+
70	Kathiya	62	F	6421	2	8	0	200	210	36	0.7	-	-	+	-	-	+	-	-	+	+	-
71	Helarani	35	F	7517	2	2	0	141	182	24	0.6	+	-	-	-	-	-	+	+	-	+	-
72	Manjula	47	F	7520	2	5	0	110	172	28	0.7	+	-	-	-	-	-	+	+	-	+	-
73	Arathal	65	F	7924	2	10	0	200	210	42	2	-	-	-	+	-	+	-	-	+	+	-
74	Mani	36	F	8352	2	4	0	172	194	28	0.7	+	-	-	-	-	-	+	+	-	+	-
75	Vargeez	62	M	8430	2	5	0	110	170	27	0.6	-	+	-	-	-	-	+	+	-	+	-
76	Hari	61	M	8354	2	4	0	112	182	24	0.7	+	-	-	-	-	-	+	+	-	+	-
77	Aarathan	56	M	8463	2	2	0	102	171	23	0.4	+	-	-	-	-	-	+	+	-	+	-
78	ValarmaTHI	48	f	8190	2	5	0	140	192	24	0.6	+	-	-	-	-	-	+	+	-	+	-
79	Rajeshwari	52	f	8113	2	3	0	112	172	26	0.8	+	-	-	-	-	+	-	+	-	+	-
80	Sasikumar	38	M	12712	2	2	0	200	240	22	0.4	+	-	-	-	-	-	+	+	-	+	-
81	Sudha	35	F	10141	2	1	0	93	160	28	0.6	+	-	-	-	-	-	+	+	-	+	-
82	Renuka	58	F	10013	2	6	0	225	273	40	0.7	-	+	-	-	-	+	-	-	+	+	-
83	Saraswathi	56	F	7565	2	10	0	140	172	28	0.6	-	-	-	+	-	+	-	-	+	-	+
84	Gomathi	52	F	8121	2	2	0	112	260	36	0.8	-	-	+	-	-	+	-	-	+	-	+
85	Valarmathi	58	F	8190	2	5	0	161	474	37	0.9	-	-	+	-	-	+	-	+	-	+	-
86	Rani	26	F	8424	2	3	0	117	164	28	0.8	+	-	-	-	-	-	+	+	-	+	-
87	Valliammal	55	F	5343	2	20	0	202	284	40	2.1	-	-	-	-	+	+	-	-	+	+	-
88	Sameena	36	F	7002	2	2	0	105	134	26	0.7	+	-	-	-	-	-	+	+	-	+	-
89	Ruckumani	58	F	8141	2	15	0	217	324	38	2.1	-	-	-	+	-	+	-	+	-	-	+
90	Krishnaveni	39	F	8421	2	0	8	112	142	26	0.9	+	-	-	-	-	-	+	+	-	+	-
91	Valliammal	48	F	6869	2	2	0	141	164	29	0.8	-	+	-	-	-	-	+	+	-	+	-
92	Chellammal	56	F	4072	2	4	0	120	162	22	0.6	+	-	-	-	-	-	+	+	-	+	-
93	Kalliammal	54	F	6894	2	1	0	110	140	26	0.7	+	-	-	-	-	-	+	+	-	+	-
94	Govindaraj	65	M	8517	2	8	0	212	312	46	1.2	-	-	+	-	-	+	-	-	+	+	-
95	Maruthammal	65	F	51031	2	5	0	250	230	38	0.7	-	-	+	-	-	-	+	-	+	-	+
96	Indrani	55	F	53442	2	2	0	230	208	36	0.8	+	-	-	-	-	+	-	+	-	+	-
97	Jothimani	58	F	53611	2	4	0	161	212	40	0.9	-	+	-	-	-	+	-	+	-	+	-
98	Rajammal	50	F	10856	2	3	0	100	125	26	0.7	+	-	-	-	-	-	+	+	-	+	-
99	Krishnaveni	58	F	11981	2	10	0	90	139	28	0.7	-	-	+	-	-	+	-	-	+	+	-
100	Devika	51	F	7004	2	4	0	131	164	24	0.7	+	-	-	-	-	-	+	-	-	+	-
101	Radhika	40	F	5686	2	2	0	110	162	26	0.4	+	-	-	-	-	-	+	+	-	+	-
102	Bakyam	62	F	11342	2	10	0	158	289	46	2	-	-	-	-	+	+	-	-	+	-	+
103	Jothimani	42	F	7028	2	3	0	121	237	28	0.7	+	-	-	-	-	-	+	+	-	+	-
104	Kannaki	50	F	7021	2	4	0	111	162	24	0.8	+	-	-	-	-	-	+	+	-	+	-
105	Kamala	60	F	10769	2	2	0	170	200	24	0.7	+	-	-	-	-	+	-	+	-	+	-
106	Thangam	63	F	9689	2	10	0	172	194	22	0.7	-	-	-	-	+	+	-	+	-	-	+
107	Valli	60	F	9846	2	5	0	116	172	28	0.7	-	+	-	-	-	+	-	+	-	+	-
108	Vasanthi	40	F	98884	2	1	0	132	164	26	0.8	+	-	-	-	-	-	+	+	-	+	-
109	Pachiammal	64	F	9959	2	8	0	212	244	34	0.6	-	+	-	-	-	+	-	-	+	+	-
110	Kamala	45	F	9427	2	3	0	121	237	27	0.8	+	-	-	-	-	-	+	+	-	+	-

100	Devika	51	F	7004	2	4	0	131	164	24	0.7	+	-	-	-	-	-	+	-	-	+	-
101	Radhika	40	F	5686	2	2	0	110	162	26	0.4	+	-	-	-	-	-	+	+	-	+	-
102	Bakyam	62	F	11342	2	10	0	158	289	46	2	-	-	-	-	+	+	-	-	+	-	+
103	Jothimani	42	F	7028	2	3	0	121	237	28	0.7	+	-	-	-	-	-	+	+	-	+	-
104	Kannaki	50	F	7021	2	4	0	111	162	24	0.8	+	-	-	-	-	-	+	+	-	+	-
105	Kamala	60	F	10769	2	2	0	170	200	24	0.7	+	-	-	-	-	+	-	+	-	+	-
106	Thangam	63	F	9689	2	10	0	172	194	22	0.7	-	-	-	-	+	+	-	+	-	-	+
107	Valli	60	F	9846	2	5	0	116	172	28	0.7	-	+	-	-	-	+	-	+	-	+	-
108	Vasanthi	40	F	98884	2	1	0	132	164	26	0.8	+	-	-	-	-	-	+	+	-	+	-
109	Pachiammal	64	F	9959	2	8	0	212	244	34	0.6	-	+	-	-	-	+	-	-	+	+	-
110	Kamala	45	F	9427	2	3	0	121	237	27	0.8	+	-	-	-	-	-	+	+	-	+	-
111	Devainai	58	F	13137	2	10	0	160	190	39	0.6	-	-	-	+	-	+	-	-	+	-	+
112	Bakkiappan	63	M	13283	2	3	0	110	140	27	0.8	+	-	-	-	-	+	-	+	-	+	-
113	Muthulakshmi	52	f	13642	2	0	4	102	180	27	0.8	+	-	-	-	-	+	-	+	-	+	-
114	Sarala	50	F	13409	2	12	0	112	260	36	0.8	-	-	+	-	-	-	+	-	+	-	+
115	Gunaseelan	55	M	8199	2	15	0	162	194	47	1.2	-	-	-	+	-	-	+	+	-	+	-
116	Chinnasamy	60	M	8262	2	4	0	171	182	32	0.7	-	+	-	-	-	-	+	+	-	+	-
117	Eswaran	56	M	8303	2	7	0	102	161	29	0.8	+	-	-	-	-	-	+	+	-	+	-
118	Govindaraj	65	M	8517	2	6	0	300	342	92	6.2	-	-	-	+	-	-	+	+	-	+	-
119	Antony	60	M	8592	2	8	0	123	182	40	0.9	-	-	+	-	-	+	-	+	-	-	+
120	Murugan	45	M	8930	2	8	0	141	184	29	0.7	+	-	-	-	-	+	-	+	-	+	-
121	Duraisamy	54	M	8961	2	0	6	120	162	25	0.8	+	-	-	-	-	-	+	+	-	+	-
122	Sraman	55	M	8942	2	2	0	111	152	32	0.7	+	-	-	-	-	-	+	+	-	+	-
123	Banu	55	F	1045	2	3	0	90	120	24	0.8	+	-	-	-	-	-	+	+	-	+	-
124	Nagarathinam	59	F	11452	2	3	0	99	120	28	0.7	+	-	-	-	-	-	+	+	-	+	-
125	Stephen	60	M	8368	2	7	0	120	170	31	0.7	-	+	-	-	-	+	-	+	-	-	+
126	Shanmugam	75	M	8900	2	5	0	171	192	38	0.9	-	+	-	-	-	-	+	+	-	+	-
127	Dhanraj	45	M	9355	2	2	0	110	140	28	0.7	+	-	-	-	-	-	+	+	-	+	-
128	Grish	50	M	9488	2	4	0	90	120	25	0.8	+	-	-	-	-	-	+	+	-	+	-
129	Mariammal	46	F	12919	2	2	0	120	162	29	0.7	-	+	-	-	-	-	+	+	-	+	-
130	Rangasamy	69	M	12812	2	15	0	103	183	45	1.2	-	-	-	-	+	+	-	-	+	-	+
131	Moorthy	60	M	10912	2	13	0	212	260	38	0.7	-	-	-	+	-	+	-	+	-	+	-
132	Dhanraj	49	M	11711	2	10	0	160	190	94	7.1	-	-	-	-	+	+	-	+	-	-	+
133	Kalayni	46	F	8321	2	2	0	110	142	28	0.7	+	-	-	-	-	-	+	+	-	+	-
134	Rangammal	50	F	8428	2	3	0	141	162	30	0.8	+	-	-	-	-	-	+	+	-	+	-
135	Selvaraj	44	M	1342	2	12	0	112	260	36	0.8	-	-	-	-	+	-	+	-	+	-	+
136	Bakkiappan	63	M	13283	2	3	0	110	144	26	0.7	+	-	-	-	-	-	+	+	-	+	-
137	Abdull	45	M	13061	2	4	0	141	164	31	0.7	-	+	-	-	-	-	+	+	-	+	-
138	Marriammal	46	F	12098	2	2	0	110	140	28	0.6	+	-	-	-	-	-	+	+	-	+	-
139	Joseph	61	M	12546	2	2	0	177	283	42	0.8	-	-	+	-	-	-	+	+	-	+	-
140	Arunkumar	45	M	17563	2	1	0	212	240	41	1.2	+	-	-	-	-	-	+	+	-	+	-
141	Kumarasamy	63	M	7668	2	6	0	210	240	41	0.7	-	-	+	-	-	-	+	+	-	+	-
142	Basteen	39	M	10465	2	4	0	112	142	34	0.6	+	-	-	-	-	-	+	+	-	+	-
143	Balraj	48	M	10643	2	2	0	162	171	28	0.7	+	-	-	-	-	-	+	+	-	+	-
144	Velu	54	M	995	2	9	0	240	312	60	4.2	-	-	-	-	+	+	-	-	+	-	+
145	Nagaraj	66	M	13562	2	6	0	171	240	42	0.9	-	-	+	-	-	-	+	+	-	+	-
146	Venkatachalam	54	M	13871	2	3	0	112	140	28	0.6	+	-	-	-	-	-	+	+	-	+	-
147	Arumugam	40	M	7123	2	2	0	90	160	31	0.8	+	-	-	-	-	-	+	+	-	+	-
148	Manohar	54	M	10276	2	15	0	400	512	81	3	-	-	-	-	+	-	+	-	+	-	+
149	Karuppan	52	M	16975	2	2	0	100	140	26	0.8	+	-	-	-	-	-	+	+	-	+	-
150	Sasikumar	52	M	12712	2	1	0	142	192	27	0.5	+	-	-	-	-	-	+	+	-	+	-