Dissertation on

"AN ANALYTIC STUDY TO EVALUATE SEVERITY OF DIABETIC RETINOPATHY AND INCIDENCE OF NEPHROPATHY AND NEUROPATHY IN PATIENTS WITH TYPE II DIABETES MELLITUS"

Submitted in partial fulfillment of requirements of

MASTER OF SURGERY DEGREE

BRANCH – III – (OPHTHALMOLOGY)

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MADURAI- 20

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Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI

2015

Madurai 20, 24.09.2014.

CERTIFICATE

This is to certify that this dissertation entitled **"AN ANALYTIC STUDY TO EVALUATE SEVERITY OF DIABETIC RETINOPATHY AND INCIDENCE OF NEPHROPATHY AND NEUROPATHY IN PATIENTS WITH TYPE II DIABETES MELLITUS"** is a bonafide record of research work done by **Dr. M. BAGAVATH SHALINI,** Post Graduate Resident in Department of Ophthalmology, Madurai Medical College, Madurai.

She has submitted this in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University, for the award of Master of Surgery Degree Branch III (ophthalmology), under our guidance and supervision during the academic years 2012-2015.

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This is to certify that this dissertation entitled**"AN ANALYTIC STUDY TO EVALUATE SEVERITY OF DIABETIC RETINOPATHY AND INCIDENCE OF NEPHROPATHY AND NEUROPATHY IN PATIENTS WITH TYPE II DIABETES MELLITUS"** is a bonafide record of research work done by **Dr. M.BAGAVATH SHALINI,** Post Graduate Resident in Department of Ophthalmology, Madurai Medical College, Madurai.

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The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 05.03.2014, Wednesday at 10.00 am to 12.00.noon at the Auditorium, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

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Name of P.G.	Course	Name of the Project	Remarks
Dr.M.Bagavath Shalini mb.shalini@yahoo.com	PG in MS (Ophthalmology) Madurai Medical College and Government Rajaji Hospital, Madurai	An analytic study to evaluate severity of diabetic retinopathy and incidence of neuropathy, nephropathy in patients with type II diabetes mellitus.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
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The above Applicant
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DECLARATION

I, **Dr. M. BAGAVATH SHALINI** hereby solemnly declare that, this dissertation titled **"AN ANALYTIC STUDY TO EVALUATE SEVERITY OF DIABETIC RETINOPATHY AND INCIDENCE OF NEPHROPATHY AND NEUROPATHY IN PATIENTS WITH TYPE II DIABETES MELLITUS"** was done by me.

I also declare that this bonafide work / a part of this work was not submitted by me / anyone else, for any award, for Degree / Diploma to any other University / Board either in India / abroad. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of Master of Surgery degree Branch -III (Ophthalmology) to be held in April 2015.

Place: Madurai Dr. M.BAGAVATH SHALINI

Date:

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PART ONE

INTRODUCTION

Diabetes mellitus, a metabolic disorder with disturbance in carbohydrate, protein and fat metabolism because of partial or complete deficiency of insulin secretion or action. According to International Diabetes Federation, 381 million people worldwide suffer due to diabetes. In India, 62 million suffer from diabetes and 1 million die every year due to diabetes.

Three clinical presentation of diabetes mellitus namely – Insulin dependent diabetes mellitus (IDDM), Non - Insulin dependent diabetes mellitus (NIDDM) and Gestational diabetes (GDM). They present either with normal glucose tolerance / impaired glucose tolerance or frank diabetes mellitus.

RISK FACTORS FOR TYPE 2 DIABETES:

- Ethnicity
- Advancing age
- Lack of physical activity
- Increased cholesterol intake in diet
- Obese with BMI > 30kg/sq.m

- Family history
- Drug / chemical causing diabetes
- Insulin resistance
- Beta cell failure
- smoking

GENOTYPE / PHENOTYPE CORRELATION:

Hyperglycemia being most important factor for complication in patients with diabetes mellitus. Diabetic retinopathy progression can be prevented with good control of glycemic level but in certain individual there is progression of retinopathy is rapid despite of good glycemic control.

There is great inter individual difference in the onset, rapidity of progression and intensity of complication in diabetic patients. Most important factors are mutation, environmental and familial factors.

Individuals with the interaction of gene-environment mutation will develop such a phenotype when the person enters such kind of greater risk environmental condition, which may cause specific alteration in the flow of blood as well as blood retinal barrier.

PROTECTIVE FACTORS FROM DR:

1. Myopia - 2.00D is protective due to reduced blood flow, retinal thinning, posterior vitreous detachment
2. Glaucoma – because of decreased blood flow / decreased metabolic activity due to reduced ganglionic cells which are viable
3. Posterior vitreous detachment
4. Retinitis pigmentosa

Diabetic patients during course of disease develop the following:

Microvascular Complications

1. Diabetic retinopathy in their eyes
2. Diabetic nephropathy in their kidneys
3. Diabetic neuropathy in their nerves

Macrovascular Complications

1. Coronary artery disease in heart
2. Stroke in brain
3. Foot ulcers in limbs

Glycaemic control is of utmost importance in the preventing death due to micro and macrovascular complications. Management of diabetes includes diet, exercise, oral hypoglycaemic drugs, and insulin therapy according to stage of diabetes along with genetic counselling.

ANATOMY OF RETINA

Retina is a thin delicate transparent innermost tunic of eyeball, extending from optic disc to ora serrata with surface area of about 266sq mm.

Ophthalmoscopically the retina is described as follows,

1. **Optic disc** :

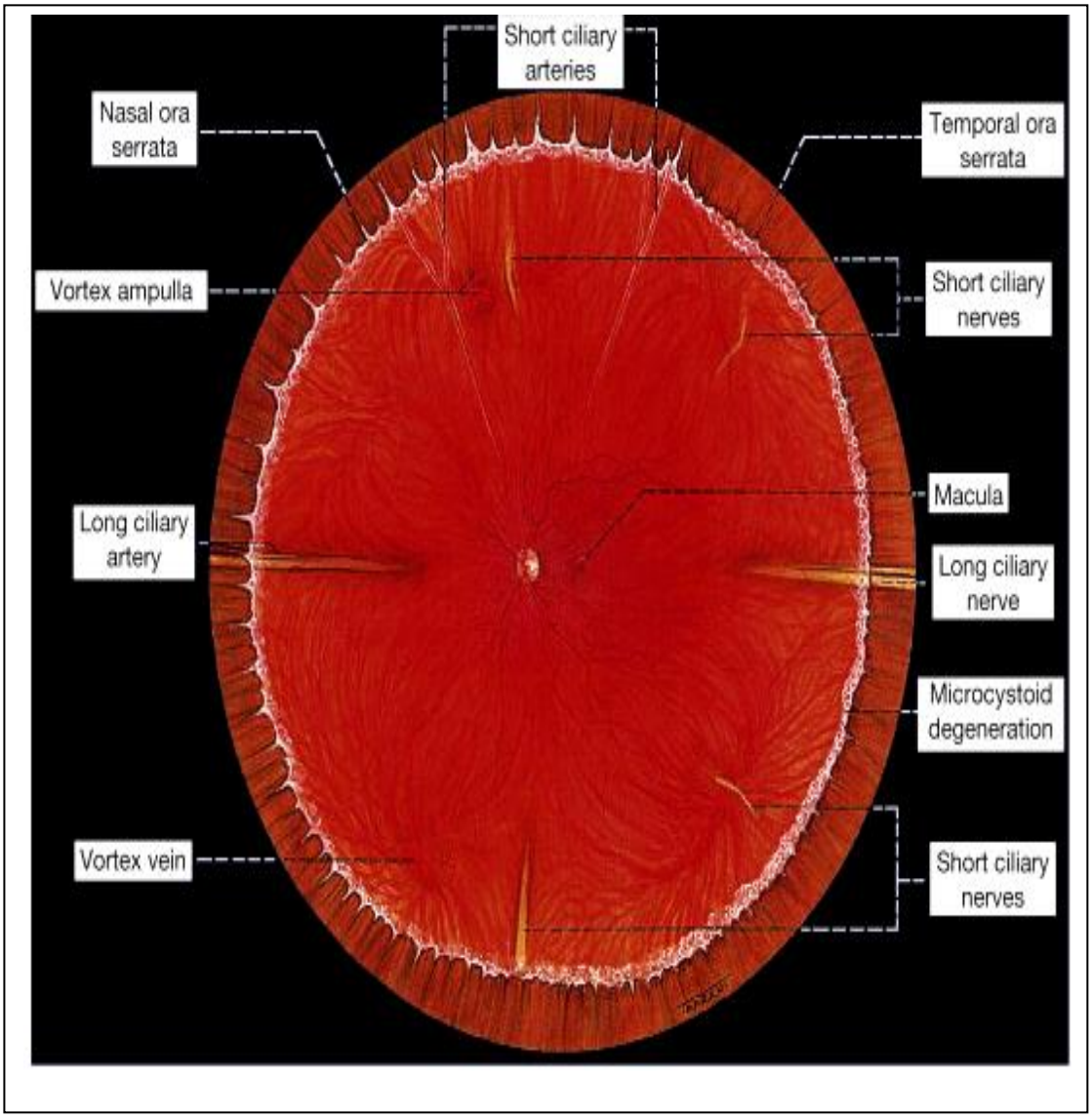
   Circular pale area in the posterior pole of 1.5 mm diameter which makes the exit of axons of ganglion cells from retina and continue through lamina cribrosa as optic nerve.

2. **Macula** :

   Temporal to disc in the posterior pole of eye is the macula. A dark area measuring about 5.5 mm in diameter. Central depressed area of macula is Fovea centralis corresponding to 5 degree of field of vision. Foveola forms floor of fovea. Umbo tiny depression visible ophthalmoscopically as foveal light reflex. Foveal avascular zone inside fovea. Yellow spot of macula is due xanthophyll's.

3. **Peripheral retina** :

Retina extending up to ora serrata where retina ends with serrated margins and ciliary body begins. 6-8mm away from equator and 25mm away from optic nerve.



Near periphery	Circumscribed area surrounding the centralis of about 1.5mm
Mid periphery	The 3mm wide zone surrounding the near periphery is involved most in diabetes.
Far periphery	Extend from optic disc, in horizontal meridian temporal side 9-10mm and nasally 16mm.

BLOOD SUPPLY:

1. Outer four layers – choriocapillaries
2. Inner six layers – central retinal artery
3. Outer plexiform layer – both choriocapillaries and central retinal artery

Macula - central retinal artery superior and inferior temporal branches, cilioretinal artery and choriocapillaries are absent in this layer.

BLOOD SUPPLY	*LAYER OF RETINA*	*CELL TYPE AND FUNCTION*
Chorio capillaries	Retinal pigment epithelium (RPE)	Outermost layer of retina consist of a single layer of hexagonal cells. Adjacent RPE cells are connected by tight junctions (zonulae occludens and zonulae adherens). The RPE is firmly attached to underlying Bruch's membrane and loosely adherent to the photoreceptors. Its functions are photoreceptor renewal, vitamin A recycling, mechanical support to photoreceptors, phagocytic action.

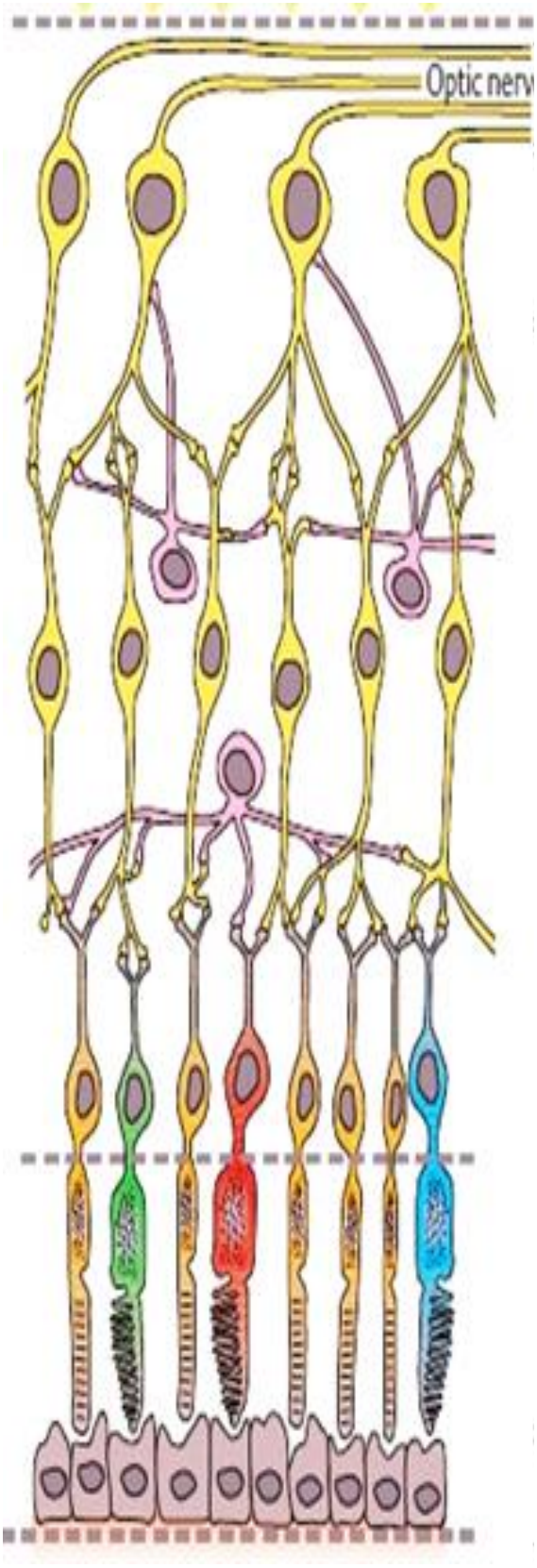
	Photoreceptor cells	Rods and cones, are special sensory cells that absorbs the photons of light, contain photo pigments. Rods are active in dull light, cones in bright light. Parts of rods and cones – 1. Outer segment contains visual pigment that converts light into neural signal 2. Cilium - connecting stalk 3. Inner segment contains metabolic apparatus 4. Outer fiber 5. Cell body 6. Inner fiber
	External limiting membrane	Junction between photoreceptors and Muller's cell, it is not a true basement membrane.
	Outer nuclear layer	Rods and cone nuclei forms this layer. Next to external limiting layer is single layer of outer nuclear layer.

Central retinal artery	Outer plexiform layer.	By synapses of spherules of rod and pedicles of cone with bipolar cell dendrites and horizontal cell processes.
	Inner Nuclear Layer	Consist of bipolar cells, Amacraine cells, horizontal cells, Muller's cell, and retinal capillaries.
	Inner Plexiform Layer	Synapses of bipolar cell axons and ganglion cell dendrites forms this layer. This layer is absent in Foveola.
	Ganglion Cell Layer	Single layer of ganglionic cells throughout retina except at macula it is multi layered and two layers temporal to disc.
	Nerve Fibre Layer	Unmyelinated ganglionic cell axons form this layer, they converge at optic disc and passes into lamina cribrosa and get

		myelin sheath posterior to lamina. Muller's cell processes interweave with ganglion cell axons.
	Internal Limiting Membrane	True basement membrane. Contains collagen fibrils type 1 and 4, proteoglycans, basement and plasma membrane

1. Central Retinal Artery – End artery enters optic nerve at 1cm behind the globe, have intima, internal elastic lamina, media and adventitia.
2. Retinal Arterioles – Contains smooth muscles within their walls
3. Capillaries – Consist of endothelial cells, basement membrane, and pericytes.
4. Venous system – Small venules are just larger than the capillaries, Larger venules have smooth muscles, Veins - contains less smooth muscles and elastic tissue.

MICROSCOPIC STRUCTURE:



Internal limiting membrane

Nerve fiber layer

Ganglion cell layer

Inner plexiform layer

Amacrine cells

Inner nuclear layer

Outer plexiform layer or Henle's layer Horizontal cells

Outer nuclear layer

Outer limiting membrane

Photoreceptor layer of rods and cones & Muller cells

Retinal pigment epithelium

Bruch's membrane

FUNDUS EXAMINATION:

1. Indirect ophthalmoscopy head mounted

   -gives steoscopc view

   - magnification depends on lens power and working distance

Eg. Lens,	magnification ,	degree field ofview
20	3	45
25	2.5	50
30	2	60
40	1.5	65

2. Slit lamp biomicroscopy with 90D and 78D lens
3. Goldmann 3- mirror examination
4. Direct ophthalmoscopy

RISK FACTORS IN DIABETIC RETINOPATHY

1. Diabetes duration – more than 5years
2. Poor glycaemic control HbA1c –

   Normal < 5.8%, Pre-diabetic 5.9 - 6.4%, Diabetic > 6.5%

3. Hyperlipidaemias - ↑ triglycerides and ↓ HDL
4. Hypertension – more than140 / 80 mm Hg
5. Pregnancy – factors like poor pre-pregnancy control of diabetic, severe pre-pregnancy retinopathy, rapid control of diabetes in early stages of diabetes, eclampsia and fluid imbalance.
6. Anaemia – Hb% < 6.0 gms / dl
7. Obesity – BMI – Normal 18-25kg / sq. mt.
8. Genetic factors – HLA type – DR3 / DR4

   Worsening genes: Aldose reductase gene (AKR1B1), z-2 microsatellite, NOS3, VEGF, ITGA2, ICAM1

   Protective genes: Z+2 microsatellite, T allele of AKR1B1promoter rs759853 variant in type 1

9. Nephropathy
10. Intraocular surgery

PATHOGENESIS OF DIABETIC RETINOPATHY

Hyperglycemia, a hallmark in diabetes, results in cellular damage. Endothelial cells are more prone to hyperglycemic damage as they poorly regulate intracellular glucose. Increase in blood glucose sets in a chain of metabolic events that leads to **overproduction of reactive oxygen species** in the mitochondria which in turn increases the hexosamine and polyol pathways, increased formation of advanced glycation end products and activation of protein kinase C. Hyperglycemia being the central feature as it is directly linked to the above changes causes ischemia of tissues.

Biochemical and cellular events which initiates vascular lesions is complex. It is not clearly known whether retinopathy is due to direct effect of insulin deficiency / resistance / due to other metabolic derangement such as hyperlipidemia associated with diabetes. Vascular dysfunction is due to breakdown of blood-retinal barrier that leads to diabetic macular edema, which is responsible for visual loss in diabetic retinopathy patients.

POLYOL PATHWAY:

Increased glucose metabolism through polyol pathway results in the increase in sorbitol, reduction in myo-inositol with reduced activity of sodium-potassium-ATPase which are responsible for vascular dysfunction. Key enzyme in the polyol pathway is **Aldose reductase**.

Under normal circumstances glucose is metabolized through hexokinase pathway but in the hyperglycemia with high glucose levels hexokinase pathway is saturated and glucose is metabolized by the polyol pathway. **Sorbitol which is not easily dissolved across cell membranes accumulates inside the cell**, increases cellular osmolality, and ultimately leads to damage of cells.

FORMATION OF NON-ENZYMATIC GLYCATION PRODUCTS:

Another cause of vascular damage in the development of diabetic retinopathy involves accumulation of Advanced Glycosylation End Products (AGEs). As consequence of hyperglycemia, a carbohydrate combines with protein side non enzymatically to form amadori products which subsequently form advanced glycosylation end products.

AGE formation in the basement membrane of endothelial cell **inactivates endothelial-derived nitric oxide**, which have action on peri-vascular smooth muscle resulting in vasodilation and also impairs the blood flow.

PROTEIN KINASE C ACTIVITY:

Enzyme Protein kinase that modifies other proteins by adding phosphate groups chemically to them is called phosphorylation. Protein kinase C is involved in transduction of signal to growth factors, neurotransmitters and hormones. Activation of Protein kinase C phosphorylates the proteins which results in alterations in the blood flow of retina and **breakdown of blood–retina barrier.**

MATRIX CHANGES:

Capillary Basement Membrane Thickening (CBMT) forms the histological hallmark of microangiopathy , which occurs because of increased extracellular matrix components deposition that contribute to the development of retinal haemodynamics abnormality and abnormal retinal blood flow auto-regulation.

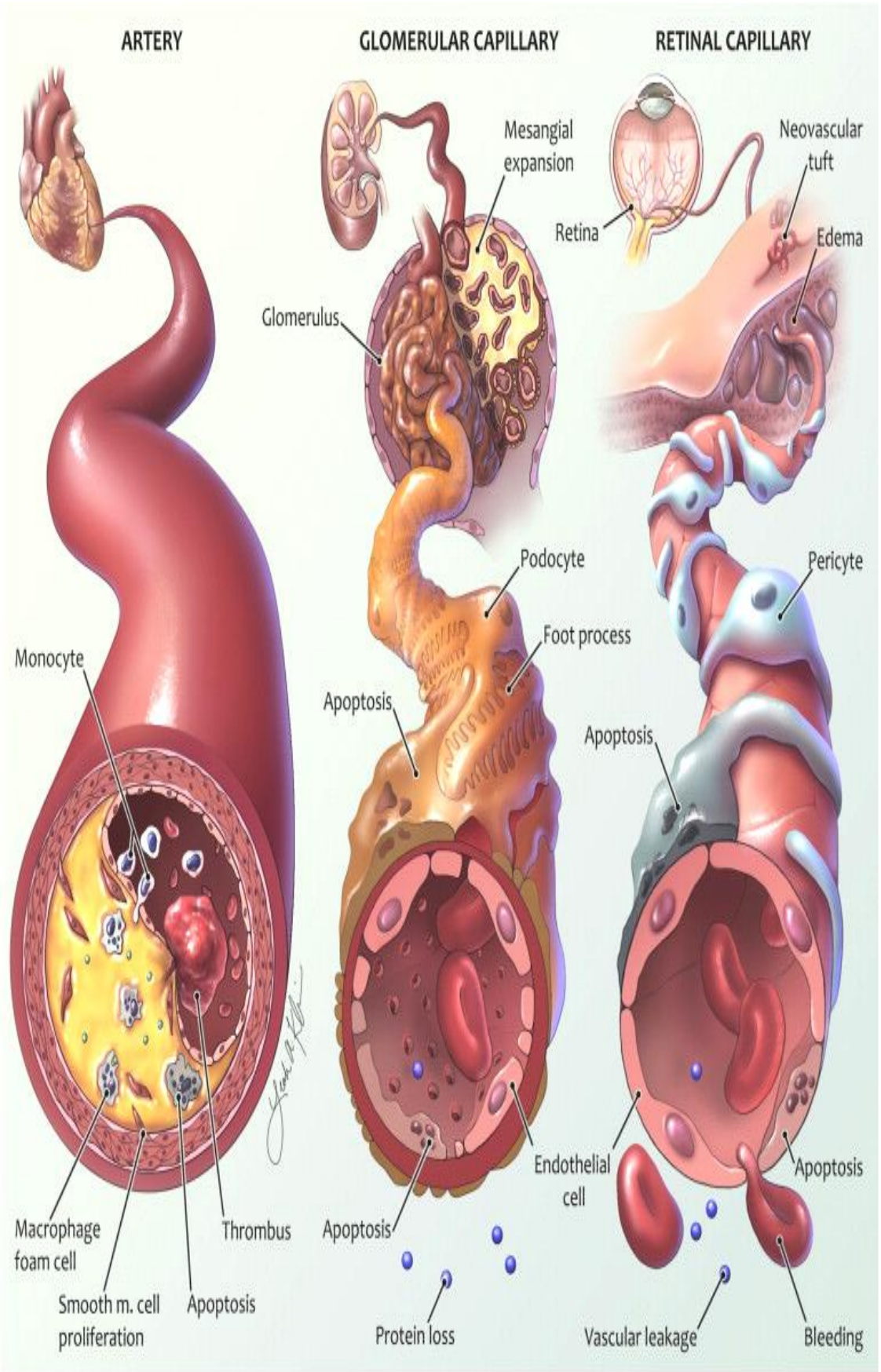
The important elements in the CBMT are type IV collagen, heparin sulphate (proteoglycan), along with

fibronectin and laminin. Endothelial cells produce heparin sulphate which is negatively charged and forms a regular lattice structure in the anionic sites which hinders the filtration of proteins like albumin that are negatively charged. In diabetes mellitus synthesis of proteoglycan is impaired and increases the in hydroxylysine and its glycosidally related disaccharide units. These alterations results in abnormal **peptide chains packing which causes excessive leaking of the membrane**. e.g. microalbuminuria occurring in diabetics.

Extracellular matrix degradation by matrix metalloproteinase (MMPs) affects endothelial cell function and increases the vascular permeability. Elevated level of MMP-9 and MMP-2 has shown in diabetic neovascular membranes.

RETINAL LEUKOSTASIS:

Retinal leukostasis also play an important role in the pathogenesis of diabetic retinopathy, particularly **leukocytes are less deformable** in diabetes .The increased leukostasis affects the retinal perfusion, endothelial function of retina, vascular permeability, and angiogenesis.


ARTERY
GLOMERULAR CAPILLARY
RETINAL CAPILLARY
Mesangial expansion
Neovascular tuft
Retina
Edema
Glomerulus
Podocyte
Pericyte
Foot process
Monocyte
Apoptosis
Apoptosis
Endothelial cell
Apoptosis
Macrophage foam cell
Thrombus
Smooth m. cell proliferation
Apoptosis
Apoptosis
Protein loss
Vascular leakage
Bleeding

Capillaropathy occurs due to **pericytes death, capillary basement membrane thickening, vascular smooth muscle loss and endothelial cell proliferation**. Leakage and occlusion indicates capillary dysfunction. Increased vascular permeability occurs due to cell-cell junction alteration leads to retinal edema, haemorrhages and exudates.

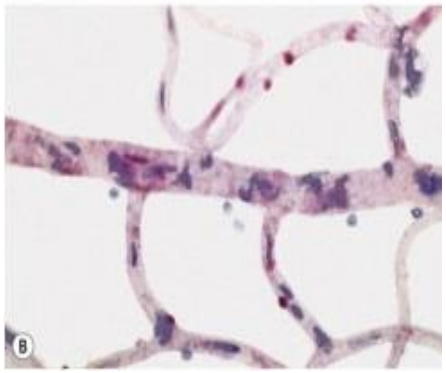
Disruption of endothelial integrity leads to capillary non perfusion which in turn results in retinal ischemia which in turn leads to vascular endothelial growth factor (VEGF) mediated neovascularization such as intraretinal microvascular abnormality (IRMA) and vitreous haemorrhage, NVD and NVE in proliferative diabetic retinopathy.

Hematological abnormalities such as **increased stickiness of platelets, abnormalities of erythrocytes, increased plasma viscosity also contribute to the development of diabetic retinopathy**. In diabetics thromboxane A2 which is released from platelets is increased which causes significant vasoconstriction and also leads to platelet aggregation. All the above factors lead to occlusion of small vessel due to micro-thrombus formation.

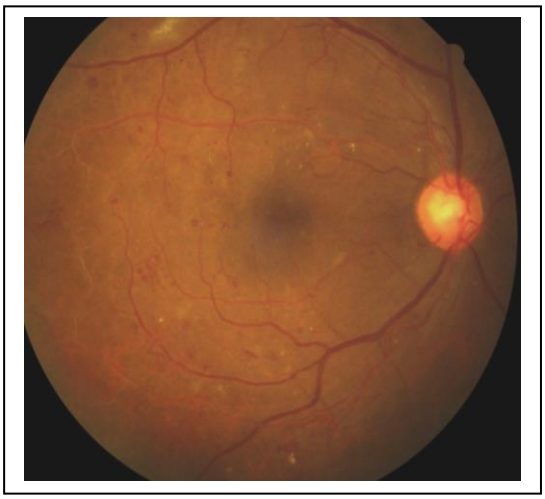
All the above sequences result in retinal capillary occlusion, resulting in retinal ischemia which stimulates a pathological neovascularization that are mediated by angiogenic factors like vascular endothelial growth factor (VEGF), that ultimately leads to proliferative retinopathy.

Capillary closure with adjacent dilated and elongated capillaries.

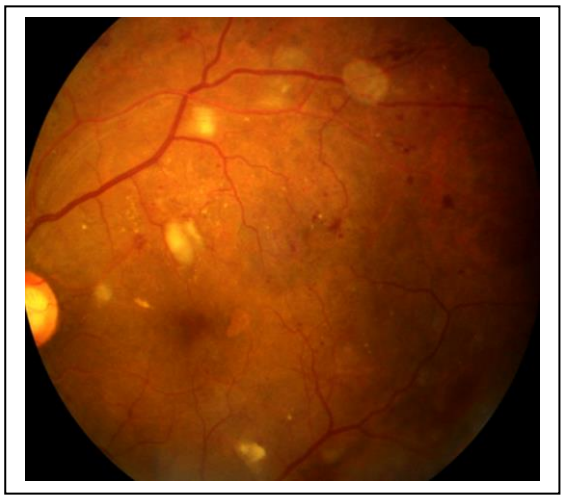
Degenerate pericytes which are eosinophilic- Trypsin digest preparation



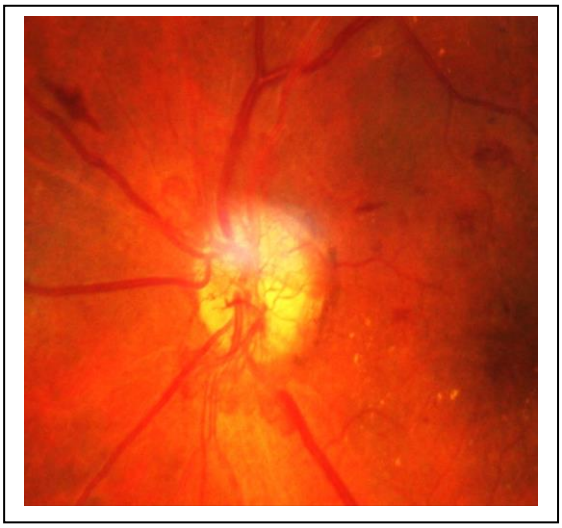
New capillaries (arrow) in relation to non-perfusion areas.



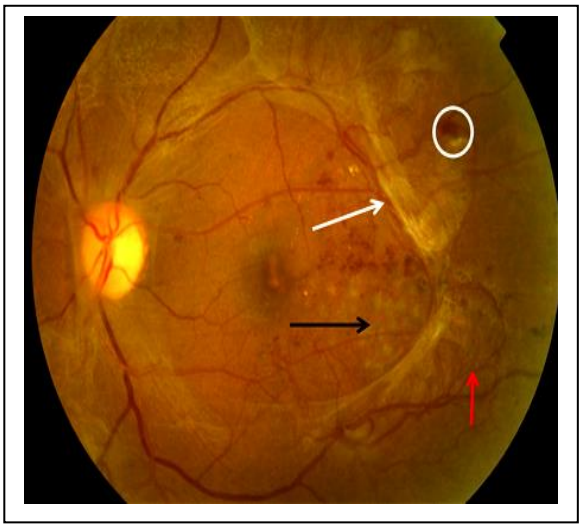
Mild NPDR



Moderate NPDR



Neovascularization disc



Tractional retinal detachment

DIABETIC RETINOPATHY MANIFESTATIONS & GRADING

It begins as Non Proliferative Diabetic Retinopathy (NPDR) and in some patients it progresses to Proliferative Diabetic Retinopathy (PDR) in stages.

MICROANEURYSM:

- Microaneurysm are located in inner nuclear layer
- range from 10-100 micrometer.
- Those larger than 30 micromater are visible ophthalmoscopically.
- Appear as tiny spot which are bright red in color.
- In FFA it appear as hyperfluorescent.

FLAME-SHAPED HEMORRHAGE:

- Hemorrhages located within the superficial nerve fibre layer

DOT HEMORRHAGES:

- These occur in inner nuclear and outer plexiform layer.
- In FFA these appear as hypofluorescent.

- These are difficult to differentiate from microaneurysm in the fundoscopy.

BLOT HEMORRHAGES:

- Less distinct margins
- Deep capillary network gives raise to blot hemorrhage
- Lies in outer and inner plexiform layer
- Because of its location and compactness of retinal structure, it appears dark in color
- Mostly found scattered in posterior pole

HARD EXUDATES:

- Principal source is from aneurysm
- It is the representation of the leakage of retinal circulation
- Concentrated lipoprotein gives rise to waxy appearance
- Located within the outer plexiform layer
- Re-absorbed by phagocytes spontaneously or by photocoagulation

COTTON WOOL SPOTS:

- Represents the occlusion of capillaries in nerve fibre layer which are along the long axis
- Axoplasmic stasis occurs in nerve fiber layer so these fibres are swollen giving fluffy cotton wool appearance
- Commonly seen near major retinal vessels in the posterior pole

VENOUS CHANGES:

- Includes increased tortuosity as well as dilatation
- Looping or beading represents the hypoxia
- Venous loops may also be formed due to focal traction in the retina

INTRA RETINAL MICROVASCULAR ANOMALIES: (IRMA)

- Precursor of the proliferative diabetic retinopathy
- Indicator of severe NPDR

1. Background diabetic retinopathy (BDR):

Characterized by exudates, micro aneurysms, occasional dot and blot haemorrhages.

2. Non proliferative Diabetic Retinopathy early stage:

First ophthalmoscopically detectable changes in diabetic retinopathy are venous dilation and micro aneurysms, due to localized out pouching of capillary walls. When the capillary wall is weakened it ruptures and gives rise to deep intra retinal hemorrhage – if it occurs in the nuclear layers it appears dot or blot, If it occurs in the superficial nerve fiber layer it is flame or splinter shape.

` The presence of plenty of splinter hemorrhages in a diabetic should prompt to check blood pressure. Systemic hypertension if co-exists, increases the rapidity of progression of retinopathy.

Non proliferative Diabetic Retinopathy advanced stage:

In advanced NPDR, signs include multiple retinal hemorrhages, hard exudates cotton-wool spots, venous beading and loops, intraretinal microvascular abnormalities (IRMA).

Hard exudates are leaked lipoprotein and lipid filled macrophages situated within the outer plexiform layer which resolves spontaneously when leakage stops.

Cotton-wool spots or soft exudates result from local axonal ischemia that causes obstruction to axoplasmic flow with subsequent focal edema of the nerve fibers giving white fluffy appearance.

Venous beading is the sign of sluggish retinal circulation. Venous loops present adjacent to areas of no perfusion. IRMA - dilated capillaries that function as collateral shunts develop in reaction to ischemia.

Proliferative Diabetic Retinopathy

About 50% of individuals with very severe NPDR progresses to proliferative retinopathy within one year. Abnormal new vessels within one disc diameter of the optic nerve is called NVD (New Vessels over the Disc) and when

arise further away, they are known as NVE (New Vessels Elsewhere). New vessels are seen as irregular vessels, start as thin strands and attain normal retinal vessel dimension. New vessels over the disc seen as fine network of vessel bridging the physiological cup.

Vitreous changes:

New vessels on the retina cross the internal limiting membrane and are adherent to posterior vitreous surface. When vitreous starts separating from retina, the new vessels are pulled and bleeding occurs. Vitreous undergoes fibrosis and split (schisis). Fibrosis is more commonly seen in posterior pole and equator.

Retinal Detachment:

Retina is pulled by pre retinal fibro vascular vitreo retinal attachments leading to Tractional retinal detachments. Sometimes a break occurs at the base of proliferation leading to Rhegmatogenous – Tractional retinal detachments.

ATYPICAL PRESENTATION IN DR:

1. Feature less retina
2. Asymmetrical presentation
3. Carotid artery occlusion – worsens DR
4. Neovascular glaucoma
5. Florid appearance – rare seen mostly in type 1
6. Cilio retinal artery cause asymmetrical presention of retinopathy

ETDRS CLASSIFICATION OF DIABETIC RETINOPATHY

A. Mild NPDR: Presence of at least one micro aneurysm in the posterior pole, definition not met for B, C, D, E, or F.

B. Moderate NPDR: Haemorrhages and / or micro aneurysms, presence of soft exudates, venous beading, IRMA definitely present in the mid periphery, definition not met for C, D, E, or F.

C. Severe NPDR: Haemorrhages and / or micro aneurysm in all four quadrants, or venous beading in two or more quadrants, or IRMA at least in one quadrant, definition not met for D, E, or F.

D. Very severe NPDR: Any two or more of the changes seen in severe NPDR, definition not met for E, or F.

E. Early PDR: Presence of new vessels over the disc or elsewhere, definition not met for F.

F. High-risk PDR: Includes any of the following characteristics -

   a. Neovascularization of disc (NVD) > $1/3^{rd}$ to $1/4^{th}$ disc diameter, NVD < $1/3^{rd}$ to $1/4^{th}$ disc diameter with vitreous / pre-retinal haemorrhage,

b. NVE with vitreous/pre-retinal haemorrhage.

c. High-risk characteristics (HRC) were defined by Diabetic Retinopathy Study as the patient if not treated urgently is at a high risk of severe irreversible visual loss.

G. Diabetic maculopathy: involvement of macula is describes as

   a. **Focal Maculopathy**- presence of micro aneurysms, haemorrhage, macular oedema and hard exudates arranged in circinate pattern.

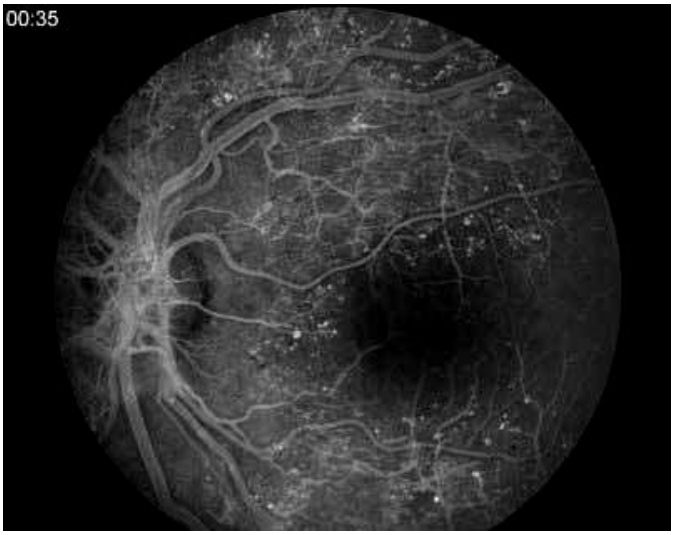
   b. **Diffuse maculopathy**- presence of diffuse retinal oedema and thickening.

   c. **Ischemic maculopathy**- marked visual loss with micro aneurysms, haemorrhage, mild or no macular oedema.

H. Clinically significant macular oedema **(CSME)** – Retinal thickening within 500 micrometre of centre of macula. Exudates within 500 micrometre of centre of macula. Retinal thickening of 1disc area (1500

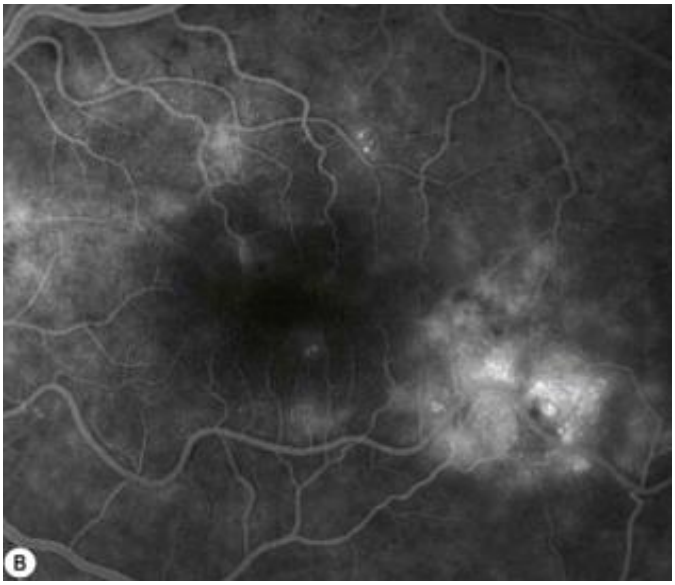
micrometre) or larger, part of which is within centre of macula.

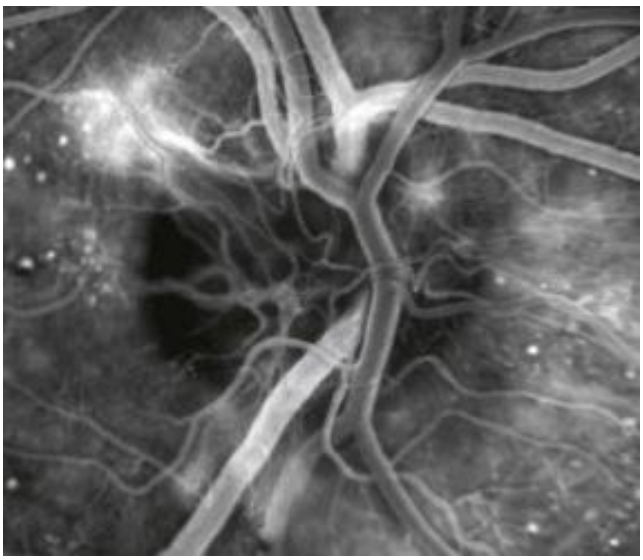
l. Advanced eye disease includes – **Persistent vitreous haemorrhage, Tractional retinal detachment, and Neovascular glaucoma.**



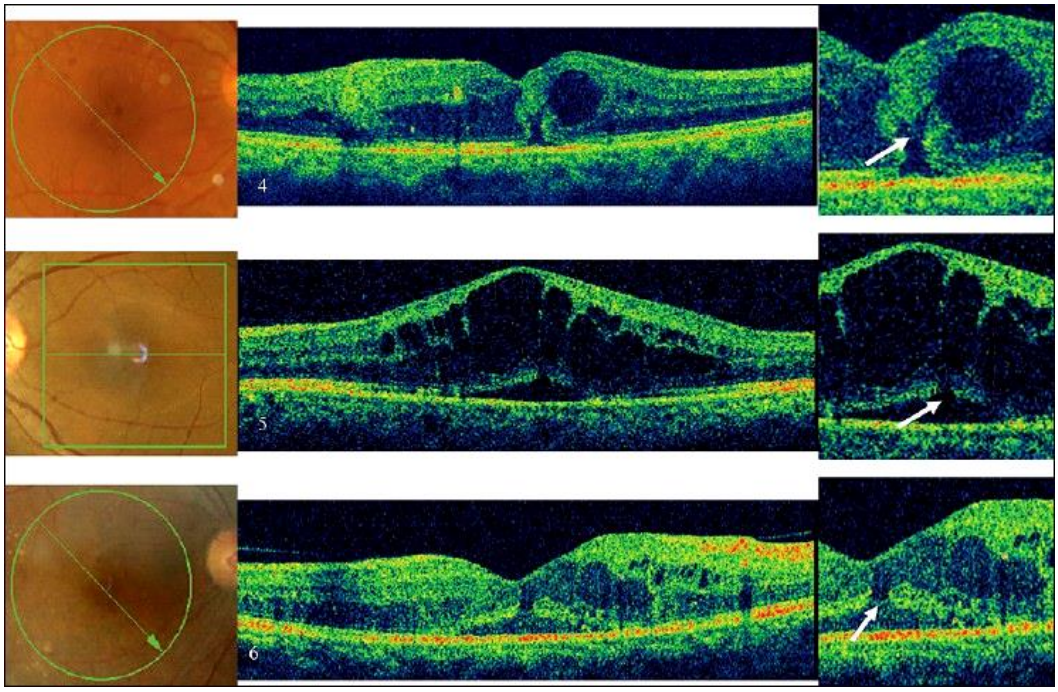
MICROANEURYSM , IRMA

DIABETIC MACULAR EDEMA

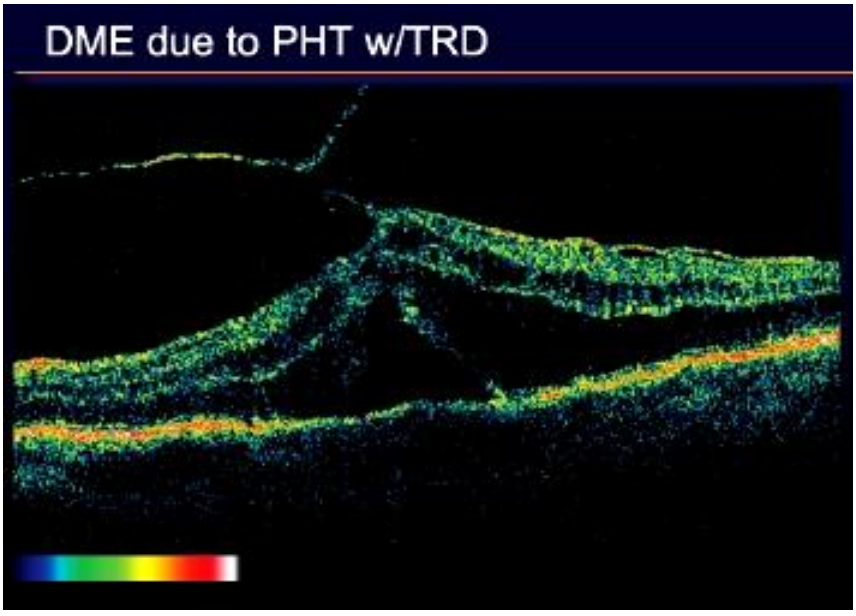




NEOVASCULARIZATION OF DISC



OCT PICTURE OF DIABETIC MACULAR EDEMA



INVESTIGATIONS:

The three main investigations to assess the retinal involvement in diabetes are the invasive Fundus Fluorescein Angiography, non-invasive Optical coherence tomography and screening tool the Stereoscopic fundus photography.

Fundus Fluorescein Angiography (FFA):

Introduction

Sodium fluorescein injected intravenous, present as bound and unbound form in the circulation. 80% bound to albumin remaining unbound form freely diffuse through chorio capillaries, bruch's membrane, sclera as well as optic nerve

Physiological barrier exist at the retinal capillaries. Break down of inner retinal barrier results in leakage of dye. And also outer blood retinal barrier forms tight junction which are impermeable to dye.

Mechanism:

Electrons are excited to higher level by particular wavelength of light, longer wavelength are emitted once the electron come back to its original level. Blue light of 465-490nm wavelength excites the fluorescein. Blue filter is present in fundus camera blocks the other light to enter the eye. Whereas reflected light from the fundus pass through the filter which prevents other light except for yellow green light.

Technique

Patient is comfortably seated in front of fundus camera. 5ml of 10% or 3ml of 25% fluorescein is injected intravenously. Red free photographs are taken at 1second interval from 5-10 seconds of injection. Then photos are taken from all areas of fundus in both eyes to find lesions of retina namely capillary non perfusion areas lined by micro aneurysms, leaks from new vessels, diffuse macular edema etc. Late photographs are taken after 10 minutes to show leakage.

Indication for FFA:

1. The leakage area is uncertain
2. The location of leakage with respect to the foveal is uncertain.
3. Foveal centre involvement with diffuse edema in macula
4. Capillary closure extent is uncertain
5. Suspected other pathology
6. Re-treatments cases and non-responsive

ADVERSE EFFECTS OF FLUORESCEIN:

1. Discolouration
2. Nausea, vomiting
3. Anaphylactic reactions
4. Dizziness
5. Papulomacular rashes
6. Itching

HYPERFLUORESCENCE CAUSES:

1. Autofluorescence
2. Pseudofluorescence – occurs before dye injection
3. Window effect – RPE atrophy / absence
4. Pooling – due to outer blood retinal barrier break down
5. Leakage due to dysfunction of tight junction or congenital absence of tight junction
6. Staining – in drusen, optic nerve, sclera and fibrous tissue in late phase

HYPOFLUORESCENCE CAUSES

1. Retinal Fluorescence Masking – hemorrhages / exudates – deeper lesions
2. Choroidal fluorescence masking – sub retinal / Choroidal lesion / hypertrophy of RPE
3. Filling defects – vessel occlusion – artery, vein, and capillary

Myopic degeneration and choroideremia

Optical Coherence Tomography (OCT):

Indication – Diabetic macular edema

Type 1: focal macular thickening

Type 2: Diffuse thickening without cyst

Type 3: Diffuse cystoids macular edema

Type 4: Tractional macular edema

4A: posterior hyaloids traction

4B: Epiretinal membrane

4C: both

Type 5: macular serous detachment

Stereoscopic photography of fundus:

- It is the visual record that documents the patient's current ophthalmological appearance of retina. It also forms an important tool for telemedicine and computer aided analysis of the fundus picture and then interpretation by ophthalmologist.

DIABETIC RETINOPATHY MANAGEMENT

SYSTEMIC MANAGEMENT FOR DIABETES

1. Dietary changes, Low saturated fat, each day fruits and vegetables given in five proportion
2. Regular physical activity, > 30minutes of increased physical activity is suggested by WHO
3. Low salt intake with type 2 diabetes who has raised blood pressure;
4. Individualized BMI
5. Oral hypoglycaemic agents: indications When HbA1C > 6.5 % or fasting plasma glucose > 6.0 m mol/dl
6. Insulin therapy
    a. When no residual activity of beta cell
    b. Uncontrolled diabetes with multiple OHA
    c. HbAIC >8%
    d. treatment of dyslipidemia
7. Stop smoking

MANAGEMENT FOR DIABETIC RETINOPATHY:

Apart from glycemic control and reversal of angiopathy, to prevent blindness we do photocoagulation to control ischemia. Photocoagulation is a focused laser beam of a discrete wavelength to penetrate the ocular media and reach RPE of retina. It is absorbed by different intraocular layers of retinal, particularly the retinal pigment epithelium, that locally rise the temperature to about 30°C, which in turn results in denaturation of tissue proteins and coagulative necrosis. Wavelength and pigment distribution determines the target and level of photocoagulation inside the retina. Pigments that absorb LASER are

1. Melanin in the RPE
2. Xanthophyll a macular pigment
3. Hemoglobin in Red Blood Cells
4. Melanin within scleral and choroidal melanocytes
5. Lipofuscin in ageing eyes.

The mechanisms by which photocoagulation inhibits exudation and the resultant involution of neovascularization of retina are not completely understood, but includes

1. Laser induces RPE-mediated release of cytokines and growth factors to restore the outer and inner blood-retinal barriers in diabetic maculopathy.
2. Laser may cause damage to the RPE which allow movement of anti-angiogenic agents into the inner retina.
3. Oxygenation is improved in the inner retina because of laser-induced destruction of RPE / photoreceptor complex. This is the mode of action of pan retinal photocoagulation.

Precautions in eyes with photophobia:

- Lower illumination to start with initially slowly raised
- Red free photography is used
- Blink after each laser application
- Sub tenon block can be given

Argon laser is commonly used laser in treating diabetic retinopathy. Mainly are 488 nm (blue) and 514 nm (green) i.e. relatively shorter wavelengths which penetrate and travel to the retina. Frequency-doubled YAG (532 nm) laser is of green wavelength similar to argon (514 nm).

Diode laser (810 nm) less up taken by hemoglobin thereby allowing laser through vitreous haemorrhages. Disadvantage is that it produces deeper burns with more pain.

Indications for LASER Photocoagulation:

1. Ischemic diabetic maculopathy
2. Proliferative retinopathy with recurrent vitreous hemorrhage.

Diabetic Maculopathy:

Laser treatment for diabetic maculopathy is aimed at arresting the leakage from microvascular abnormality which allows fluid and hard exudates to re-absorb.

Types of maculopathy are focal, diffuse, and ischemic maculopathy

1. Focally exudative maculopathy with circinate exudates- good prognosis 10% patients suffer moderate visual loss in 1 year – Focal laser
2. Diffusely edematous maculopathy - worst prognosis – Grid photocoagulation. 20% of them have moderate visual loss within 1 year;
3. Ischemic maculopathy: 30% has suffer moderate visual loss in 1 year.

The treatment of choice depends on the pattern and extent of macular edema and extension of lesions being treated. With the focal or grid-pattern laser photocoagulation has significantly reduced the incidence of moderate visual loss by 50%.

Focal treatment:

Used for circumscribed small macular edema, here laser burns applied to areas of microvascular lesions, within the centre of rings of hard exudates. Burns size 100 microns for 0.08–0.1 second and power is 100 mW.

Following conditions where threshold is lowered,

1. Priorly planned for PRP
2. About to undergo Cataract surgery
3. Poor patient compliance
4. Pregnancy
5. Worsening of retinopathy in spite of glycemic control

Grid treatment:

Used when generalized diffuse edema of macula with foveolar involvement, spot size 100–200 microns in grid pattern of 100–200 burns for 0.08–1.0 s surrounding the macula.

Modified grid:

Similar to grid except it is concentrated in a particular sector of retina rather a general pattern surrounding the fovea. In practice this is more commonly used.

It excludes papillo-macular bundle. Macular laser treatment has most important adverse effect such as inadvertent foveal burns and secondary Choroidal neovascularization mainly due to high power of laser and small spot size that are close to fovea.

FIXATION IDENTIFICATION:

It is more important to identify the fovea before the start of laser therapy in the prevention of foveal damage.

Individual is instructed to fix eccentrically,

This is achieved by asking the individual to look at a aiming beam or small illumination.

TITRATION OF POWER OF LASER:

It is important to titrate the power, as it varies depending on the eye.

- Power of laser burns are titrated by placing the sample of preliminary burns near the retinal arcades and see for the grey appearance of area to which burns are applied.
- More power- in the areas with increased thickness.
- Phakic eyes require more power than the pseudophakic eyes.
- Pigmented patients require low power.
- High power needed in myopes.

Other treatments of diabetic maculopathy:

1. Lasers – like frequency doubled Nd:YAG lasers and micropulse diode lasers
2. Intravitreal injections: anti-VEGF

   like ranibizumab 0.5mg or triamcinolone acetonide.
3. Pars plana vitrectomy :

   edema with tangential traction of taut and thickened posterior hyaloids

Drugs like lipid lowering agents may reduce laser treatment requirement.

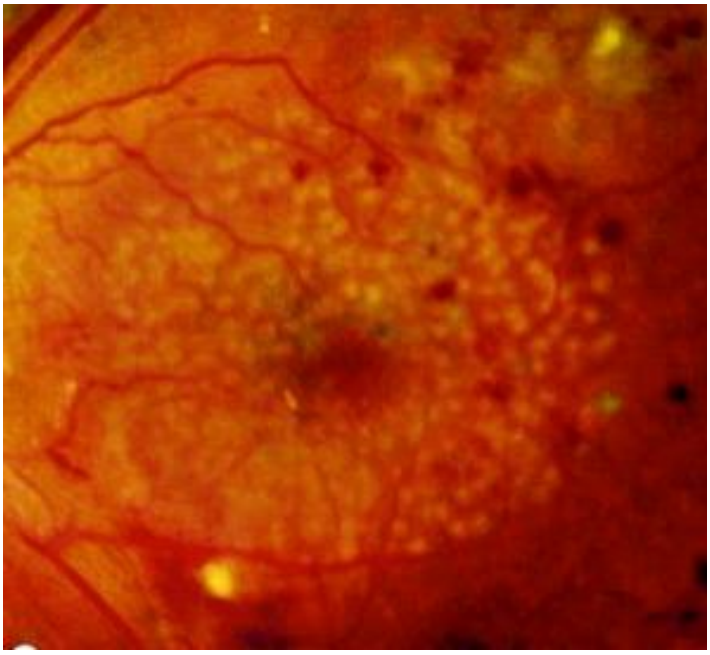
NON-RESONSE OF DM REASONS:

In non responsive cases FFA is done to assess the extent of ischemia. Reasons for non responsiveness of diabetic maculopathy are,

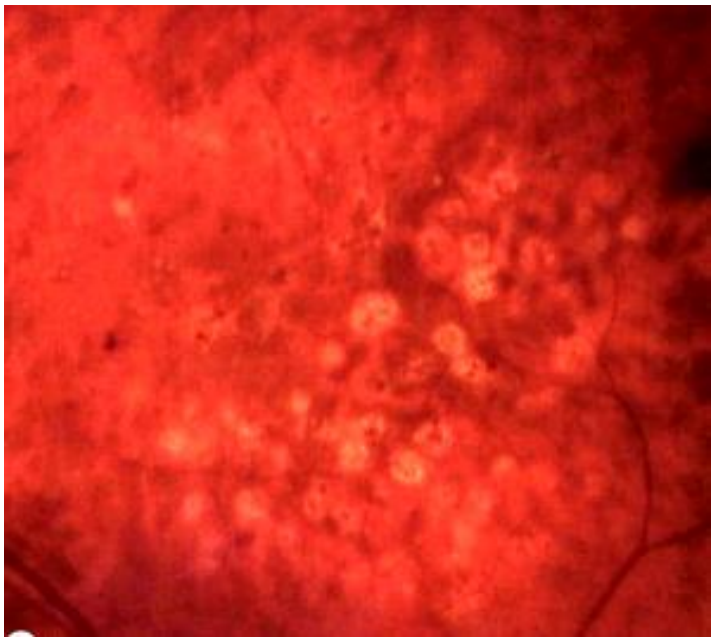
- Hypertension
- Poor glycemic status
- Traction of vitreous
- Renal failure

- Choroidal neovascularition
- Obstruction in the vascular system – confirmed by the angiogram.

GRID LASER



FOCAL LASER



Complications:

1. Choroidal effusion
2. Para central scotoma
3. Transient worsening of vision
4. Choroidal neovascularization
5. Sub retinal fibrosis
6. Laser scar expansion
7. Non resolving macular edema
8. Increased ischemia
9. contrast sensitivity and Color vision are reduced
10. sclera perforation

Proliferative diabetic retinopathy:

Aim: PRP laser therapy in PDR is aimed to induce involution of new vessels so that visual loss from Tractional retinal detachment and vitreous haemorrhage are prevented.

Indications for PRP: Presence of High risk characteristics

1. NVD – 1/3-1/4 disc area or larger
2. NVD associated with pre retinal or vitreous haemorrhage
3. NVE – at least ½ disc area larger or associated with haemorrhage

If clinically significant macular edema is co-existent grid photocoagulation is done 4 weeks prior to PRP or with first session of PRP since scatter photocoagulation may worsen macular edema as persistent / non- resolving.

Technique of PRP is 1200–1600 burns approximately of 500 microns retinal spot size , 0.1-0.2 second duration with routinely used a Volk Quadraspheric lens for mid-periphery and Mainster standard lens for posterior treatment that magnifies the spot for about 2x. Inferior half of retina is photocoagulated first because if any vitreous haemorrhage occurs will gravitate inferiorly and obscure the area, precludes further treatment.

The retinal vessels or IRMA should be differentiated from new vessels by their superficial location or extension into vitreous cavity whereas retinal vessels and IRMA lie within the retina.

When left untreated, these new vessels eventually regress and are partially replaced with fibrous tissue, which in turn strongly adhers to the vitreous and retina. With the fibrous tissue contraction or vitreous humour contraction occurs, retinal pigment epithelium is detached from the retina,

resulting in Tractional detachment of retina. Or else, rupture of new blood vessels may fill the vitreous cavity with blood resulting in vitreous haemorrhage. Vision is severely decreased with either of the above complications.

Another complication of PDR is the formation of new vessels in the iris and angle of anterior chamber (rubeosis iridis). When untreated, scarring of these structures occur, which may result in increase in intraocular pressure and Neovascular glaucoma.

Chorio retinal burn intensity:

1.Barely, visible blanching retina	Light
2.Faint and white retina	Mild
3.Opaque and dirty white	Moderate
4.Dense and white	Heavy

Technique of PRP is 1200–1600 burns approximately of 500 microns retinal spot size, 0.1-0.2 second duration with routinely used a Volk Quadraspheric lens for mid-periphery and Mainster standard lens for posterior treatment that magnifies the spot for about 2x. Laser indirect ophthalmoscopy or Goldmann 3-mirror lens for very

peripheral retina. In very severe cases peripheral cryoablation can be added. Power initially started with 180mW gradually endpoint is achieved to moderate intensity burns. Inferior half of retina is photocoagulated first because if any vitreous haemorrhage occurs will gravitate inferiorly and obscure the area, which precludes further treatment.

The amount of PRP depends on patient's tolerance. PRP is completed in 3 sessions, 10 days to 3 weeks. Topical anesthesia or sub-tenon or peribulbar block used. Consent prior to laser is essential. Performed with patient in supine position. Other mode of delivery of LASER are with indirect ophthalmoscope, endolaser during 3 port Pars Plana Vitrectomy surgery and Trans scleral for cilio ablation.

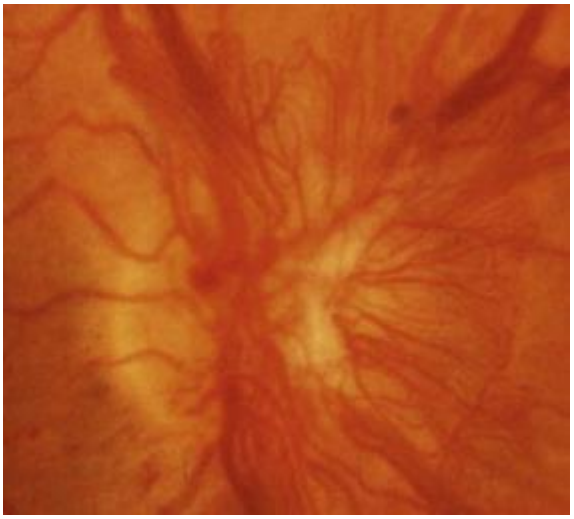
Sequence of PRP,

Step 1: close to disc, below inferior temporal arcade

Step 2: macula surrounded by a protective barrier

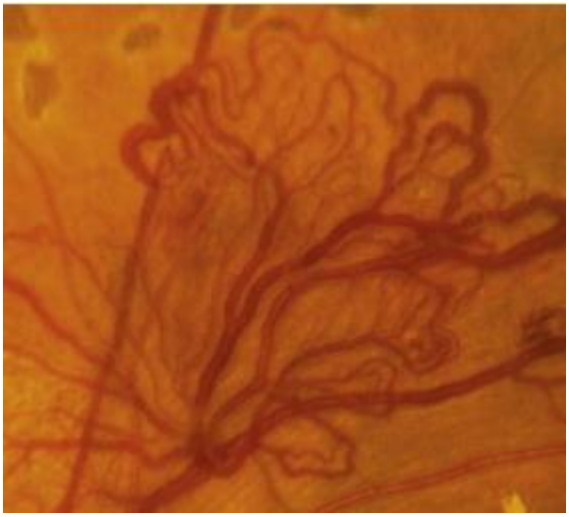
Step 3: Nasal to disc completion of posterior pole treatment

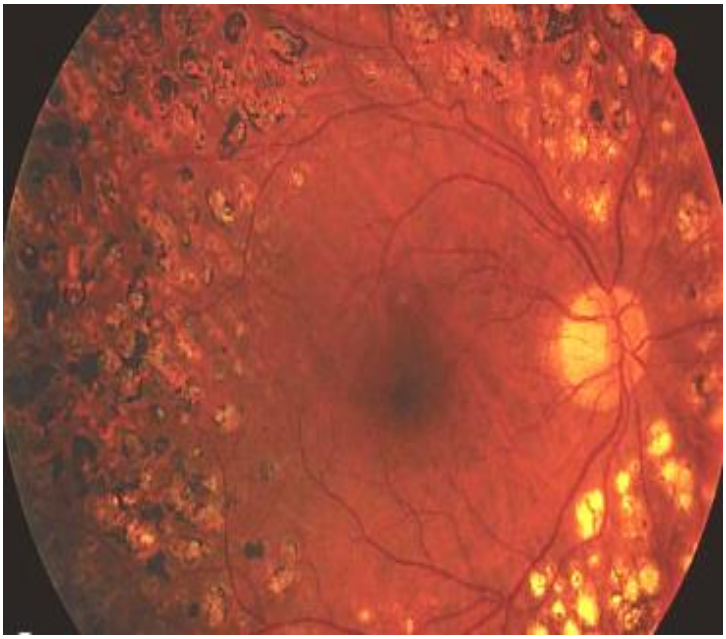
Step 4: Treatment of periphery until completion



NEOVASCULARITION OF DISC

NEOVASCULARITION OF ELSEWHERE





PAN RETINAL PHOTOCOAGULATION

Follow up is after 4-6 weeks, in recurrent or persistent cases laser photocoagulation fill in any gaps between the previous laser marks. Laser indirect ophthalmoscopy or Goldman 3-mirror lens for very peripheral retina.

Complications of PRP:

1. Central visual acuity loss – due to disruption of the blood retinal barrier, which allows fluid to leak from the choroid into the neuro-sensory retina so macular edema temporary occurs with reduction in vision.
2. Field loss - PRP burns results in atrophy which coalesce in time and visual field defects increase in extent.
3. Vitreous haemorrhage
4. Impaired accommodation and pupillary abnormalities
5. Impaired night vision and dark adaptation
6. Choroidal detachment
7. Glare, color and contrast sensitivity loss

Pars Plana Vitrectomy - in PDR in following situation

1. Clearing of media opaucities like hemorrhage in vitreous cavity
2. Removal of fibrovascular tissue leading to traction over the retina
3. Prevention of further neovascularization
4. Retinal detachment repairing

Procedure:

Vitrectomy is done through pars plana incisions placed 3.0–3.5 mm posterior to limbus in pseudophakic eyes and 3.5–4.0 mm in phakic eyes with 20 gauge-size incisions mostly used but 25G, 23G and 30G systems are available now used in indicated cases.

Three sclerotomies (ports) are needed. One is for Infusion cannula to maintain IOP, BSS is used to infuse through sutured or self-retaining cannula placed in infero-temporal quadrant. Two other sclerotomies at the 10 o'clock and 2 o'clock positions for Vitrectomy probe – cutter and fiber optic cable for illumination. Probe contains in it a oscillating or guillotine type cutter that oscillates 2500 times per minute, aspiration line is connected to the Vitrectomy probe. Another instrument inserted is fiber optic cable attached to a halogen or xenon light source which illuminates within the eye during the surgery.

Complications:

1. Post - Vitrectomy vitreous haemorrhage
2. Retinal tears and holes
3. Cataracts may develop following Vitrectomy
4. Corneal epithelial problems
5. Rubeosis

NEWER TREATMENT MODALITIES:

1. Anti – VEGF – Avastin, Lucentis, Macugen
2. PKC inhibitors – ruboxistaurin mesylate / LY 333531
3. Pigment endothelium derived factor inducers
4. Growth factor modulators – somatostatin, interferon alpha 2a, COX 2 inhibitors, ACE inhibitors
5. Anti oxidants
6. Extra cellar matrix modifiers
7. Aspirin
8. Matrix metalloproteinase inhibitors – AG3340
9. Statins
10. Oxygen
11. Aldose reductase inhibitors
12. Carbonic anhydrase inhibitors
13. Intra vitreal steroids

Co existing diseases:

1. Cataract
2. Uveitis
3. Infection - Endophthalmities
4. Posterior capsular opaucification / anterior capsule phimosis

DIABETIC NEPHROPATHY

Diabetic nephropathy otherwise known as Kimmelstiel–Wilson syndrome or nodular diabetic glomerulosclerosis is a progressive disease of kidney due to angiopathy of capillaries of glomeruli.

Diabetes being the most important cause of End-Stage Renal Disease (ESRD) particularly type 2.

RISK FACTORS:

Includes genetic factors, duration of diabetes, hyperglycaemia, hypertension, renal hyper filtration and hypertrophy and smoking.

PATHOGENSIS:

Angiopathy of capillaries is the main cause. Various mechanisms includes metabolic pathway, hemodynamic and hormonal pathway.

Metabolic pathway:

Hyperglycaemia results in activation of polyol pathway, accumulation of advanced glycation of end products, increases the reactive species and oxidative stress along with hypertension activates the intracellular signalling pathway.

These in turn release growth factors, cytokines and other inflammatory mediators that alter vessel wall permeability and proteinuria occurs. Biosynthesis of extracellular matrix and glomerular basement membrane are increased in diabetic nephropathy.

Hemodynamic and hormonal events:

Increased GFR, elevated capillary pressure, increased blood flow to glomeruli results in hyper filtration. This disrupts normal barrier in the glomeruli so plasma protein and lipoproteins accumulates and matrix degradation is suppressed causing histological alteration which is pathognomonic of diabetic nephropathy. Hormones like angiotensin 2 and endothelin are responsible for these hemodynamic changes.

ASSESSMENT OF NEPHROPATHY:

1. Spot urinary Albumin-

Normoalbuminuria -	< 30mgs
Microalbuminuria -	30-300mgs
Macroalbuminuria -	>300mgs

2.	Blood urea :	10-40 mg/dl
3.	Serum creatinine	0.6 - 1.2 (women) 0.5-1.5 (men)

CLINICAL FEATURES:

Includes - Proteinuria, fluid retention, hypertension, retinopathy, neuropathy, arterial disease. Renal biopsy showing presence of kimmelsteil Wilson nodules suggestive of poor prognosis

STAGE	TITLE	FEATURES
Stage 1	hyper function and hypertrophy	Characterized by large kidneys, glomerular hyper filtration, and hypertrophy. Blood pressure normal, Urinary Albumin Excretion (UAE) may be increased.
Stage 2	silent stage	Blood pressure and UAE are normal
Stage 3	Incipient	Hypertension and Persistent

	diabetic nephropathy	micro albuminuria present
Stage 4	Overt diabetic nephropathy	Characterized by proteinuria, low GFR, hypertension
Stage 5	End stage renal disease (ESRD):	Uremia due to renal failure with greatly reduced GFR. Cortico medullary difference is lost in ESRD

.

INVESTIGATIONS:

1. Urinary albumin excretion and albumin/creatinine ratio
2. Renal function test – glomerular function test, blood urea and serum creatinine
3. Renal biopsy

MANAGEMENT:

1. Control of hyperglycaemia – oral hypoglycaemic agents like Gliclazide, Glipizide, Glimepiride or Insulin therapy.
2. Control of hypertension
3. Low dietary proteins – 40g/day

4. Diuretics – Frusemide
5. Control urinary infection if present because it worsens renal function
6. Management of uremic diabetic – haemodialysis, continuous ambulatory peritoneal dialysis, hemofiltration and finally renal transplantation.

DIABETIC NEUROPATHY

Axonal degeneration / demyelination of a segment of a nerve due to hypoxia is seen in diabetes. Therefore patients complain of peripheral neuropathy, tropic complications and autonomic dysfunction.

PATHOGENESIS:

Hyperglycaemia causes activation of polyol pathway, myo-inositol depletion , accumulation of advanced glycation of end products, increased reactive oxygen species leads to oxidative stress in turn causes endothelial dysfunction with reduced blood flow in capillaries so endo neural hypoxia occurs causing nerve dysfunction so nerve conduction and regeneration reduced , structural damage occurs . Axonal degeneration and demyelination of a segment is the histological hallmark in diabetic neuropathy.

PATHOLOGY:

Functions of different types of peripheral nerves are

1. large myelinated A alpha fibres – for motor power, proprioception and coordination

2. Thinner myelinated A delta fibres – for cold thermal and deep sited pain
3. Thin unmyelinated C fibres – warm thermal and touch

CLASSIFICATION:

Diabetic neuropathy classified as

1. Mononeuropathies – isolated , truncal, cranial, multiple nerve involvement
2. Polyneuropathies – sensory , proximal or truncal motor
3. Autonomic neuropathy – Parasympathetic or sympathetic

STAGING:

1. No neuropathy

2 a. Acute painful – burning, shooting pain increased in night absent reflexes

b. Chronic painful – signs absent

c. Painless with partial or complete sensory loss

3. Late complications – Neuropathic deformity, foot lesion, non-traumatic amputation.

CLINICAL FEATURES:

LARGE FIBRE INVOLVEMENT – absent reflexes, unsteady gait, decreased position sense and vibration, Charcot's joint

SMALL FIBRE INVOLVEMENT – more Painful, reflexes variable, variable position sense, vibration and Charcot's joint with sensory loss.

SCREENING TESTS:

1. Pin prick test with disposable dressmakers pin
2. Light touch – with wisp of cotton
3. Vibration test – 128 Hz turning fork is used
4. Ankle reflex – both legs compared
5. Pressure perception – monofilament used to assess the risk of foot ulcer

ASSESSMENT OF NEUROPATHY: **Neuropathy disability score**

1. Vibration perception test - Normal - 0 , Abnormal - 1
2. Temperature perception - Dorsum of foot - Normal - 0 , Abnormal - 1
3. Pin prick - Normal - 0 , Abnormal - 1
4. Ankle jerk - present - 0, Present with reinforcement – 1 and Absent - 2

TREATMENT:

Good glycaemic control is the mainstay in treating the diabetic neuropathy. As there is no cure for neuropathy, by having good control over blood sugar within a target range symptoms can be reduced and preventing them from progression.

REVIEW OF LITERATURE

In the study conducted in **Main University Hospital of Alexandria Intensive Care Unit - study** showed positive correlation between Random Blood Sugar and all complication which denotes great impact of glycemic level and development and progress of diabetic complications.

In above study 250 patients admitted in intensive care unit was taken among them 148 were females and 102 were male patients. Results showed 95.6% patients had complication. The fundamental in the management of diabetes is glycemic control and its control is associated with reduction of the rate of multiple diabetes complications.

Cardiovascular complications, nephropathy and retinopathy found to be more in hypertensive patients with diabetes. The most common complications are diabetic **nephropathy, neuropathy, followed by cardiovascular complications**.

Diabetic neuropathy incidence in this study was 56.0%. At any time diabetic patient can develop problems in nerve but risk rises with duration, age and glycemic status of diabetes.

The highest rates of neuropathy are among patients who had diabetes for at least 25 years.

Valensi *et al* - explained that the presence sensorimotor peripheral neuropathy co related with hypertension, micro angiopathy and retinopathy.

Hypertension and hyperglycemia are two important risk factors in the development diabetic retinopathy. Cross-sectional study was conducted in outpatient clinic at Hvidore Hospital –

In conclusion study data indicate patients with micro albuminuria has increased risk of progression to nephropathy, proliferative retinopathy, blindness, arterial hypertension, peripheral neuropathy and foot ulcers. **Several longitudinal studies showed micro albuminuria is the early predicto**r in the progression of diabetic nephropathy.

Progression in diabetic nephropathy by the presence of raised blood pressure so effective control of blood pressure reduces albuminuria. Several studies have done previously have shown the slightly higher A1C hemoglobin concentration particularly in patients with micro albuminuria than those with the Normo albuminuria.

Marre et al showed a decrease in persistent micro albuminuria in patients who are normotensive started on insulin therapy. Indication for more frequent follow up in glycemic control, arterial hypertension, and diabetic complications is micro albuminuria.

Prospective case control study was conducted at Aravind Eye Hospital in 2001 – on 102 type 2 diabetic patients. In the conclusion of the study irrespective of the severity of retinopathy, **macular ischemia found to be important marker for nephropathy** in type 2 diabetes mellitus.

The association between macular ischaemia and diabetic nephropathy would possibly be explained based on ischemic micro angiopathy in the two end-organs in retina and kidney respectively. Similar to the capillaries in the retina in diabetic retinopathy and glomeruli of kidneys also shows basement membrane thickening in diabetic kidney disease at early stage , results in characteristic diffuse , exudative and nodular glomerular lesions. Glomerular hyalinisation is the common final point of renal lesions.

Diagnosis of ischaemia in macular region in with type 2 diabetes, irrespective of the retinopathy severity, should alert the ophthalmologist to suspect of nephropathy so patient can get appropriate investigations and management before development of complication.

Klein et al (ARIC study group) showed a significant correlation of retinopathy with hypertension and carotid artery stenosis extra cranially.

A group of 8,187cases of type 2 and 488 cases of type 1 diabetic patients were studied on population bases in north – east of spain which showed the microalbuminuria occurs due prolonged duration of uncontrolled diabetes and associated risk factors such as hypertension and increased cholesterol.

Cross-sectional population based study was conducted in Chennai in type 2 diabetic patients. It was done on cases more than 40 years. Mean of age in study group was 56+/- 10years. In all the cases urine analysis was done in morning sample to assess the presence of albuminuria. Individuals who had retinopathy were graded and documented with fundus photograph. Among 1414 cases 15.8% of patients had microalbuminuria and 2.7% patients had macroalbuminuria.

Diabetic retinopathy prevalence was more in patients with macroalbuminuria than in patients microalbuminuria as well as Normoalbuminuria. This study concludes that diabetic retinopathy incidence is more in patients with macroalbuminuria.

Cross sectional study was done in Tehran university in iran with 100patients. The study included patients with the age 58+/- 8 years of age. There was female subject slightly more. All presented with +/- 8 years of diabetes mellitus and average HbA1C was 8.4%. out of 100 75 were on oral hypoglycemic. Aim of the study was to known the correlation between diabetic retinopathy with peripheral neuropathy risk factors were also analysed.

The study interpreted 78% of diabetic retinopathy patients presented with diabetic neuropathy; and around 79% of diabetic peripheral neuropathy had presented with diabetic retinopathy. The analysis made based on this study showed no correlation between diabetic neuropathy and retinopathy significantly. 90% of individual who suffered from proliferative retinopathy had diabetic peripheral neuropathy. 27% of peripheral neuropathy patients had proliferative

diabetic retinopathy. This correlation was statistically significant.

The study further wants to extend the importanc of screening of DPN patients for retinopathy as both these are major microvascular complication of diabetes. Hence early detection by screening of diabetic retinopathy in patient with DPN helps in prevention of blindness in these patients. Newer laser modalities helps in successful management of retinopathy when detected earlier which prevents blindness.

PART TWO

AIMS AND OBJECTIVES OF THE STUDY

1. To analyse the incidence of Nephropathy, Neuropathy among Type 2 DM patients.
2. To analyse the severity of retinopathy among Type 2 Diabetic patients.
3. To Analyse Nephropathy and Neuropathy depending on severity of Retinopathy in Type 2 Diabetic patients.

MATERIALS AND METHODS

STUDY DESIGN: Analytical study

This is an analytical study. This study is to be conducted among 100 Type 2 DM patients attending the OPD as well as in the wards at Govt. Rajaji Hospital, Madurai. Subjects shall be evaluated for entry into the study if they are 40 years and above. Subjects believed to fulfill all eligibility criteria, and none of the exclusion criteria, will be invited to participate in the study.

SELECTION OF STUDY SUBJECT:

A total of 100 patients attending the O.P units and in the wards of the Department of Ophthalmology, Govt. Rajaji Hospital, Madurai who satisfy the inclusion criteria

DURATION OF THE STUDY:

5 months (2014 April to 2014 august)

ETHICAL COMMITTEE CLEARANCE: Obtained

FINANCIAL SUPPORT: NIL

INCLUSION CRITERIA:

1. Patients diagnosed with Type 2 Diabetes on treatment.
2. Duration > 5 years of diabetes mellitus.
3. Age- 40 years and above

EXCLUSION CRITERIA:

1. Patients with known history of thyroid disorders.
2. Pregnant women.
3. Patients in whom fundus cannot be examined.
4. Patients not consenting for the study.
5. Glaucoma patients.
6. Patient undergone photocoagulation.
7. Dialysis patient.

METHODOLOGY:

100 Type 2 Diabetic patients, who came to Retina clinic, Department of Ophthalmology, Govt. Rajaji Hospital, Madurai, during the period April 2014 to August 2014, were completely evaluated.

Assessment of diabetic retinopathy (Graded as per ETDRS classification), Diabetic Nephropathy (urinary albumin and blood urea, creatinine), Diabetic Neuropathy (Neuropathy disability score) was done for all patients. Analysis of Nephropathy and Neuropathy depending on severity of Retinopathy in Type 2 diabetic patients was done.

All the patients with Diabetic Retinopathy was examined with + 90 D Lens under Slit lamp Microscope and Grading done as per ETDRS classification. Those patients with Diabetic Retinopathy underwent **urine analysis for albuminuria and blood investigation** (blood urea and serum creatinine) to rule out Diabetic Nephropathy and assessment for Diabetic Neuropathy was done according to **Neuropathy Disability Score** which includes Vibration perception test, Temperature perception test, Pin prick test, Ankle jerk.

OBSERVATION AND ANALYSIS

STATISTICAL METHOD:

The information collected regarding all the selected cases were recorded in a Master Chart.

Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square, ‘t’ value and ‘p’ values were calculated.

Student’s ‘t’ test was used to test the significance of difference between quantitative variables and Yate’s and Fisher’s chi square tests for qualitative variables.

A 'p' value less than 0.005 is taken to denote significant relationship.

ANALYSIS:

Table 1: Age Distribution:

Age distribution of cases varied from 40-80years. There was no age preponderance.

Age Group	Cases	
	No	%
Upto 50 yrs	28	28
51 – 60 yrs	43	43
61 – 70 yrs	26	26
Above 70 yrs	3	3
Total	**100**	**100**

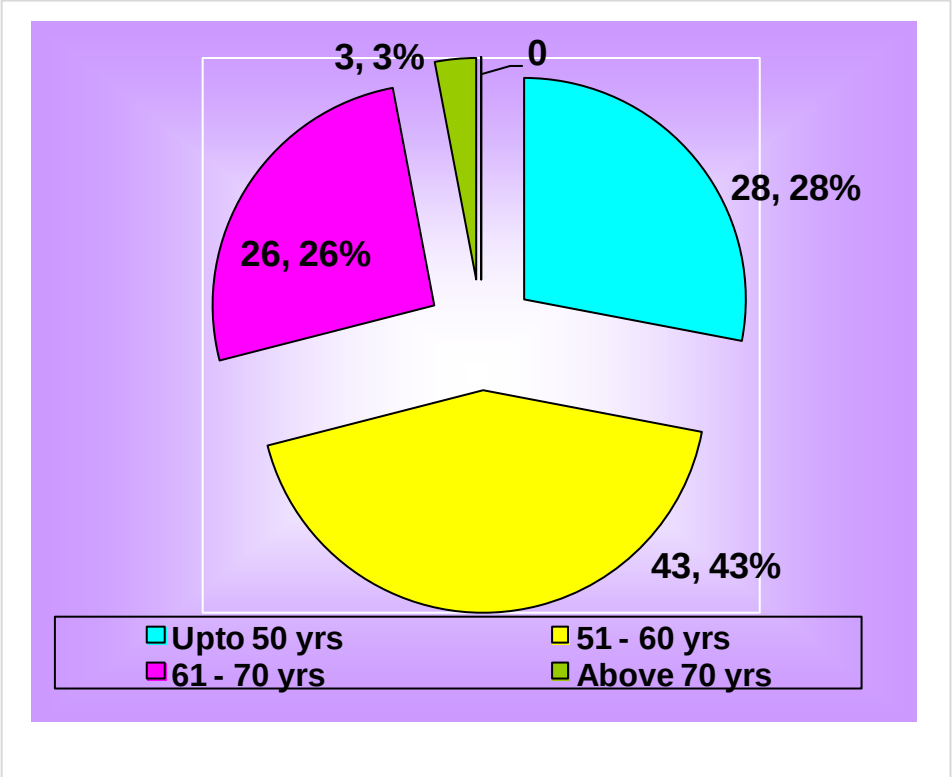


Table 2: Sex Distribution:

In total 100patients 55 were male and 45 were female patient. There was male preponderance.

Sex	Cases	
	No	**%**
Male	55	55
Female	45	45
Total	**100**	**100**

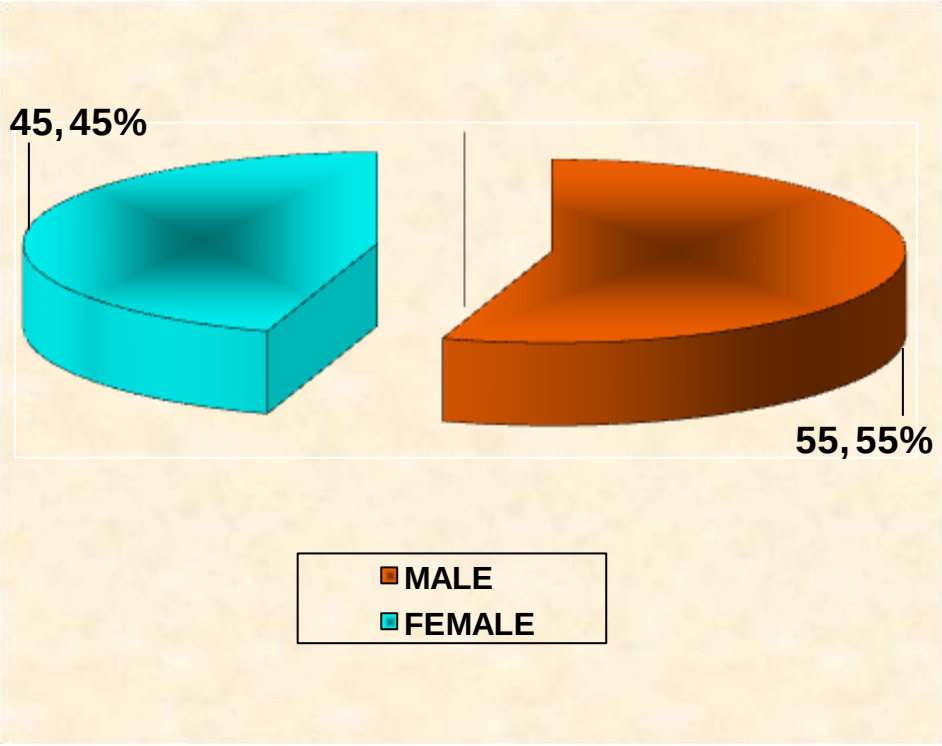


Table 3: Occupation:

Among 100patients 89% of patients were cooly, remaining patients below to various other occupation like housewife – 4%, carpenter -1%, teacher - 3%,driver – 1%, Electrician -1%, plumber -1%.

Occupation	Cases	
	No	**%**
Cooly	89	89
House wife	4	4
Carpenter	1	1
Teacher	3	3
Driver	1	1
Electrician	1	1
Plumber	1	1
Total	**100**	**100**

Table 4: Duration of diabetes:

Patients with diabetes for > 5years showed diabetic retinopathy, with the mean duration was 8.02 years

Duration of diabetes	Cases	
	No	%
Upto 5 yrs	15	15
6 – 10 yrs	70	70
11 – 15 yrs	14	14
Above 15 yrs	1	1
Total	**100**	**100**
Range	5 – 17 yrs	
Mean	8.02 yrs	
SD	2.75 yrs	

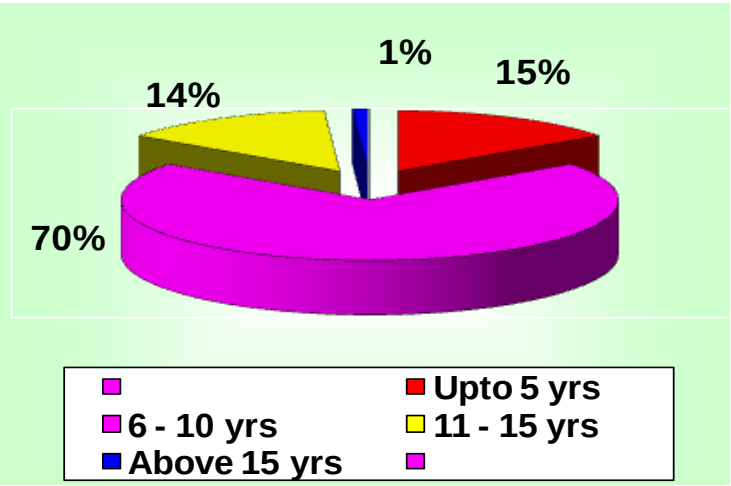


Table 5: Visual Acuity:

Visual acuity was normal in 41 eyes, 6/6p to 6/12 in 98 eyes and 6/18 to 6/60 in 61 eyes. The defective visions in these patients are due to lens changes, posterior capsular opacification and diabetic retinopathy.

VA	Right Eye		Left Eye	
	No	**%**	**No**	**%**
PH 6/6	20	20	21	21
PH 6/6P	7	7	4	4
PH 6/9	29	29	22	22
PH6/69P	4	4	3	3
PH 6/12	10	10	19	19
PH 6/18	1	1	-	-
PH 6/24	10	10	13	13
PH 6/36	5	5	6	6
PH 6/60	14	14	12	12
Total	**100**	**100**	**100**	**100**

Table 6: ANTERIOR SEGMENT:

On anterior segment evaluation 54 eyes showed lens changes, 22 eyes had posterior capsular intraocular lens and 99 eyes were normal.

AS	Right Eye		Left Eye	
	No	**%**	**No**	**%**
Immature Cataract	18	18	15	15
Posterior Capsular Intraocular lens	22	22	25	25
Minimal Lens Changes	10	10	11	11
Normal	50	50	49	49
Total	**100**	**100**	**100**	**100**

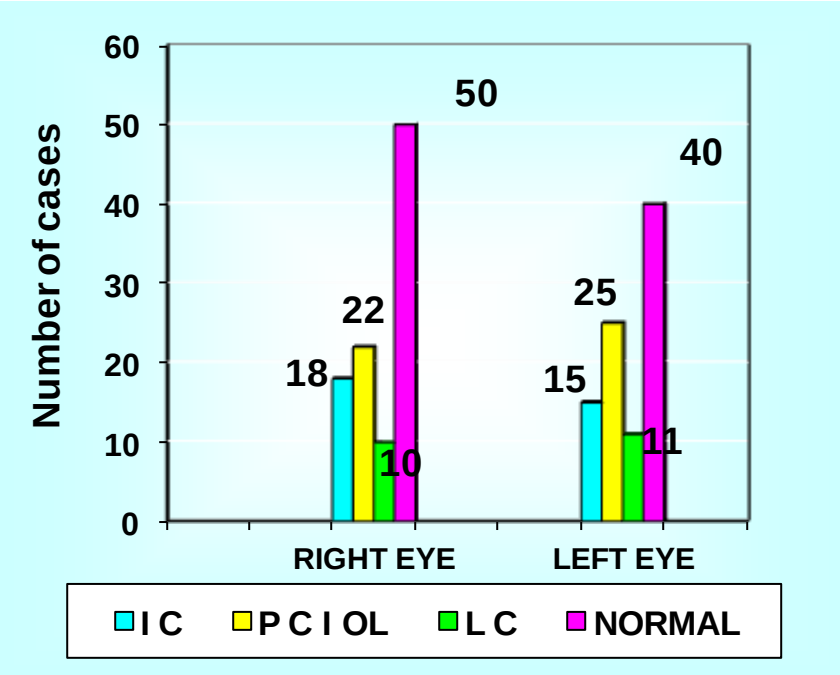


Table 7: Retinopathy:

On fundus examination showed 34% with mild NPDR, 41% with Moderate NPDR , 8% with Severe NPDR , 7% with Very severe NPDR , 4% with Early PDR, 3% with HR PDR , 3% with DM.

Retinopathy	**Cases**	
	No	**%**
Early PDR	4	4
HR PDR	3	3
Mild NPDR	34	34
Moderate NPDR	41	41
Severe NPDR	8	8
Very severe NPDR	7	7
DM	3	3
Total	**100**	**100**

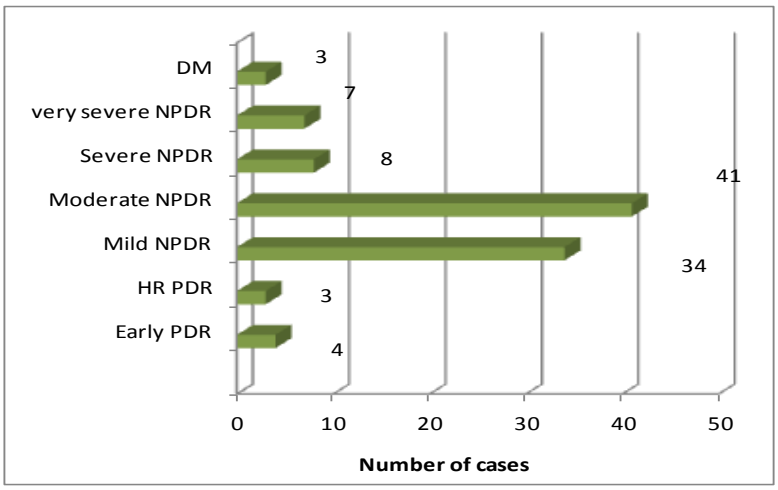


Table 8: Blood Urea:

66% patients showed abnormal blood urea level and in 34% patients blood urea was normal.

Blood Urea	Cases	
	No	%
Normal (10-40 mg / dl)	34	34
Abnormal (>40 mg/dl)	66	66

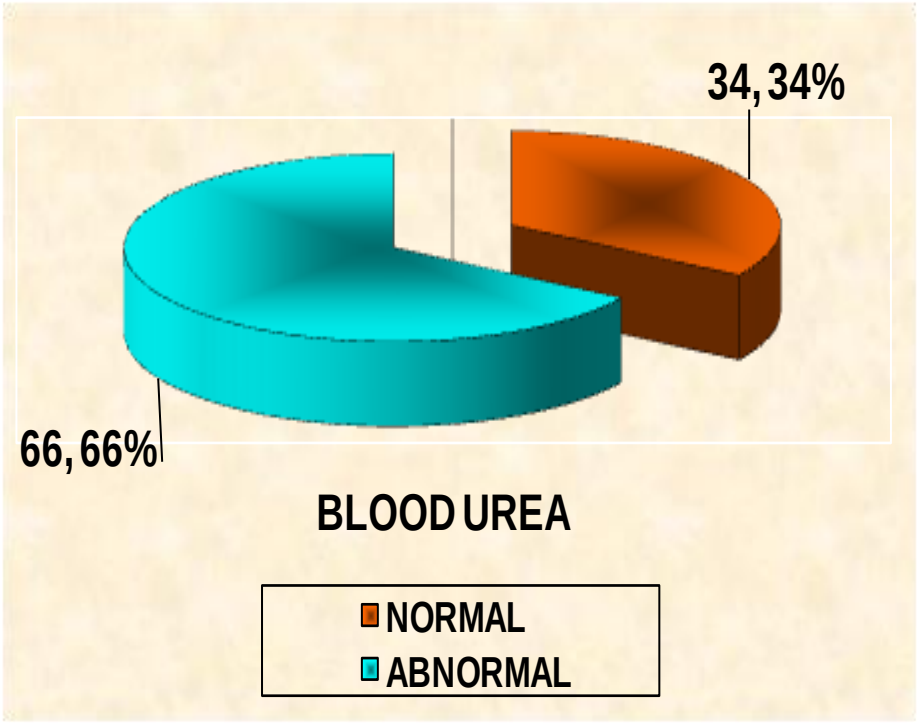


Table 9: Serum Creatinine:

70 patients had abnormal serum creatinine level and in 30 patients serum creatinine was normal.

Serum Creatinine	Cases	
	No	%
Normal	30	30
Abnormal	70	70

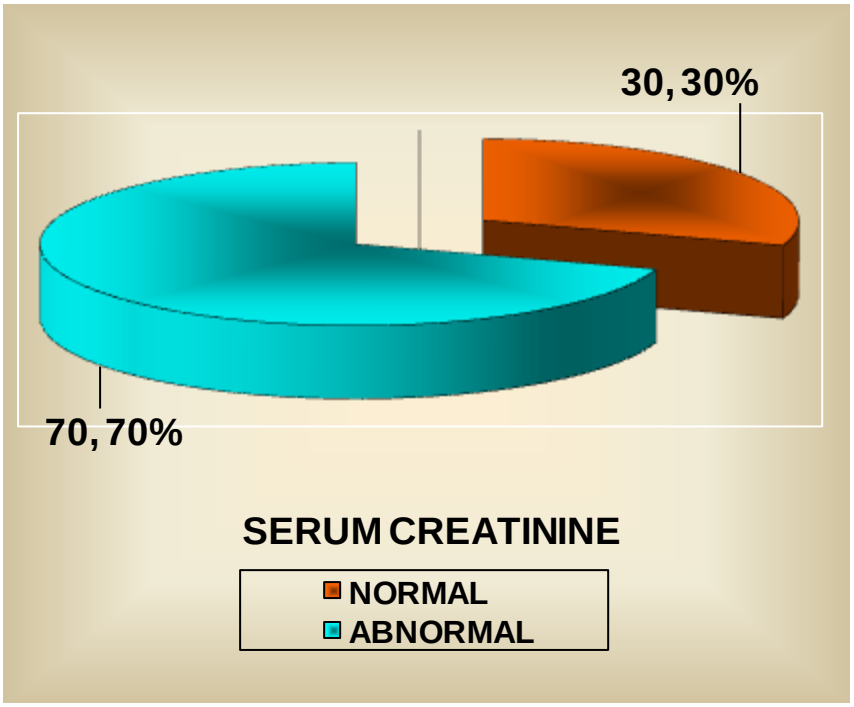


Table 10: Urine Albumin:

Out of 100 patients 34 patients showed Normoalbuminuria, 41 patients showed Microalbuminuria and 25 patients showed Macroalbuminuria

Urine Albumin	Cases	
	No	%
Normoalbuminuria (<30 mgs)	34	34
Microalbuminuria (30 – 300 mgs)	41	41
Macroalbuminuria (>300 mgs)	25	25
Total	100	100

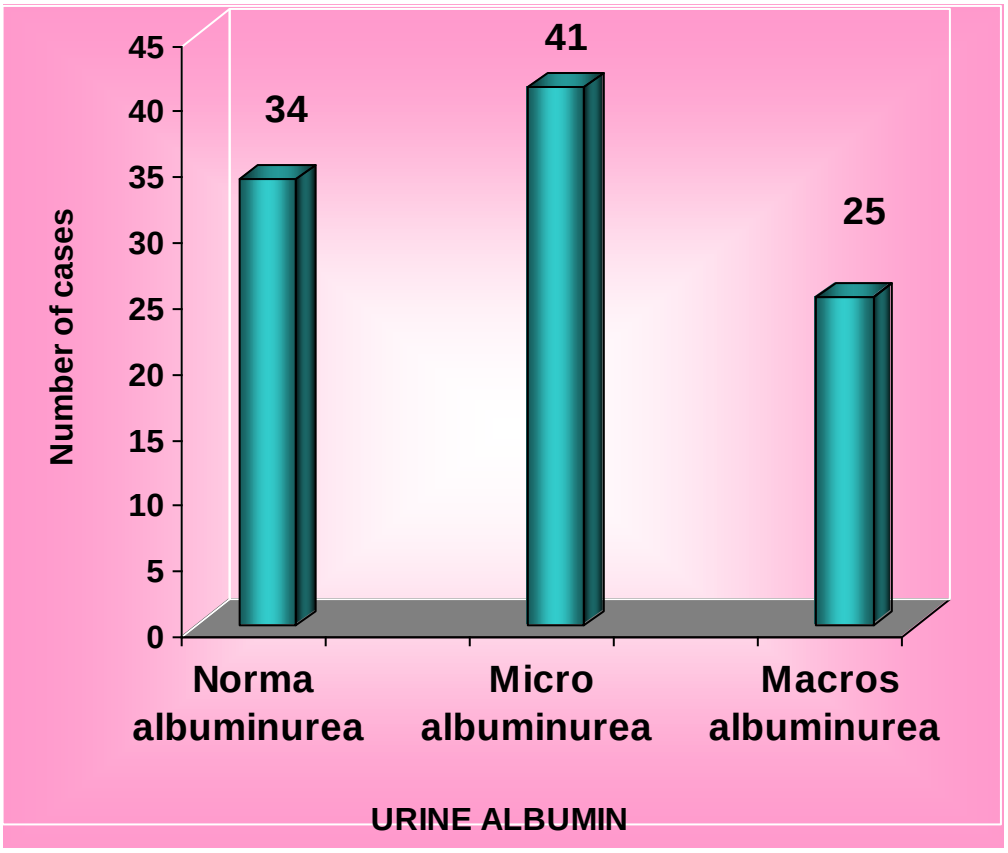


Table 11: Age and Nephropathy:

Significant correlation with age and nephropathy. ‘p’ value 0.0001

Nephropathy	Age (in yrs)	
	Mean	SD
Normoalbuminuria	52.7	8.6
Microalbuminuria	56.4	7.1
Macroalbuminuria	61.4	6.3
‘p’	0.0001 Significant	

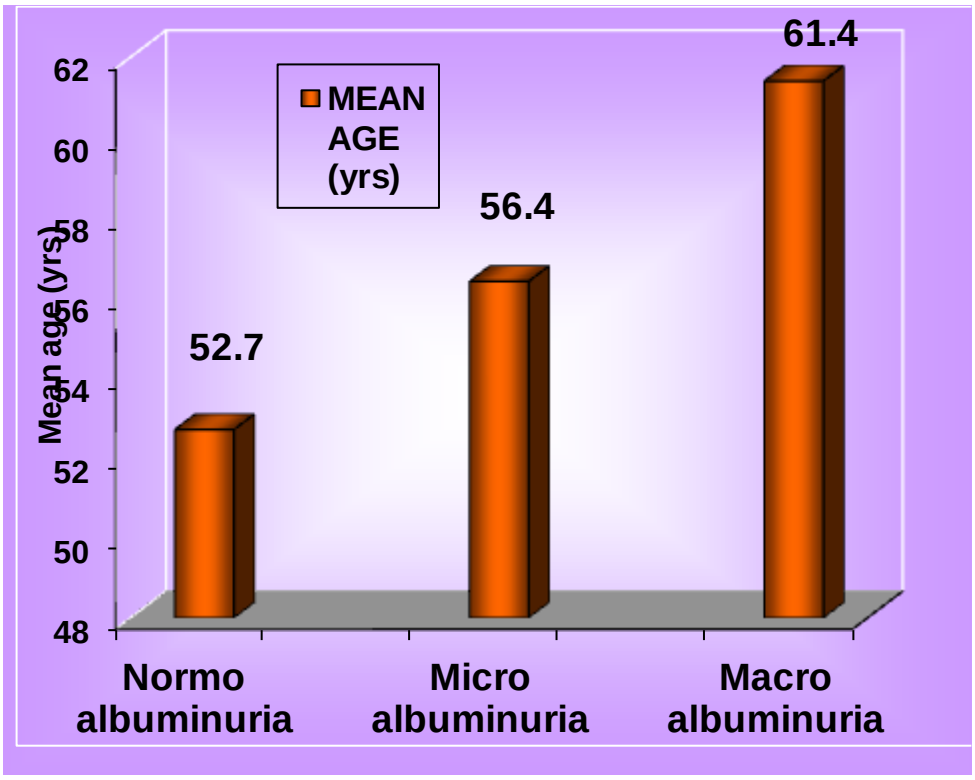


Table 12: Neuropathy:

On assessment of right and left leg for diabetic neuropathy 71% legs were normal and 29% legs showed abnormal.

Neuropathy	Right Leg				Left Leg			
	Normal		Abnormal		Normal		Abnormal	
	No	%	No	%	No	%	No	%
Vibration Perception test	71	71	29	29	71	71	29	29
Temperature perception test	71	71	29	29	71	71	29	29
Pin prick test	71	71	29	29	71	71	29	29
Ankle jerk	71	71	29	29	71	71	29	29

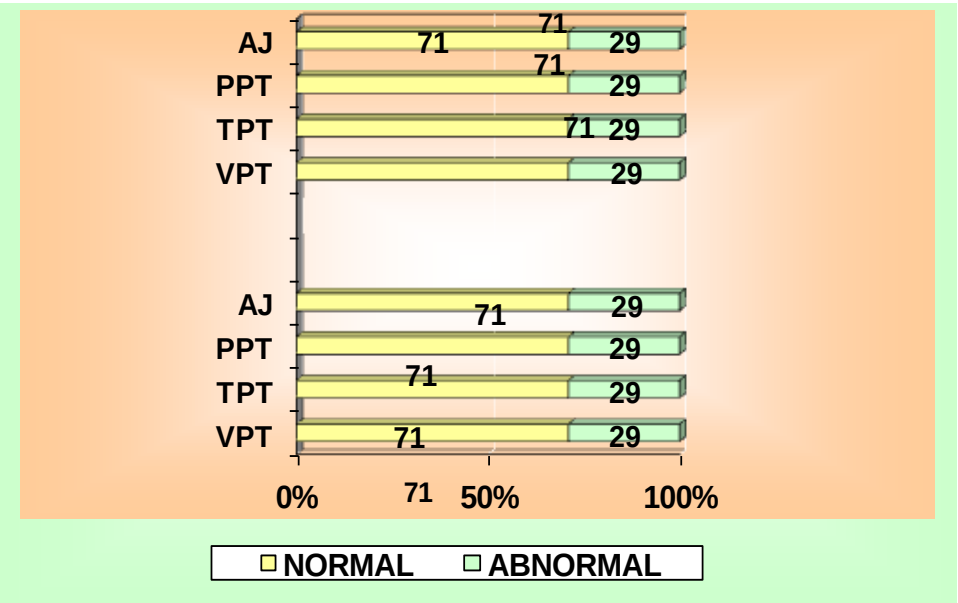


Table 12: Age and Neuropathy:

Significant correlation between age and neuropathy. p value <0.0001

Age Group	Neuropathy			
	Present		Absent	
	No.	%	No.	%
Upto 50 yrs	1	3.6	27	96.4
51 – 60 yrs	12	27.9	31	72.1
61 – 70 yrs	11	42.3	15	57.7
Above 70 yrs	1	33.3	2	66.7
Mean	54.4		62.3	
S.D.	7.6		6.6	
'p'	**< 0.0001 Significant**			

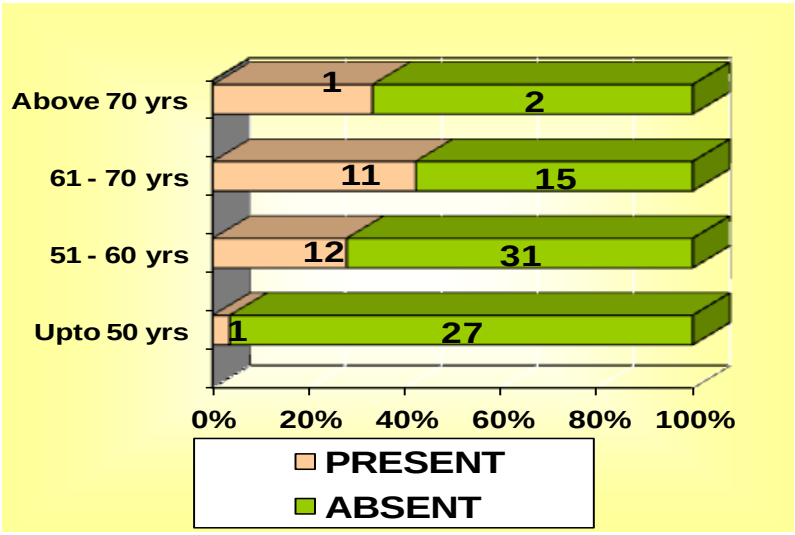


Table 13: Age and Retinopathy:

Has significant correlation p value 0.0002

Fundus	Age (in yrs)	
	Mean	SD
Mild NPDR	52.7	8.6
Moderate NPDR	55.8	6.5
Severe NPDR	57.6	6.1
Very severe NPDR	62.3	4.2
Early PDR	62.3	6.7
HR PDR	69.3	1.2
DM	68.0	7.5
'p'	**0.0002 Significant**	

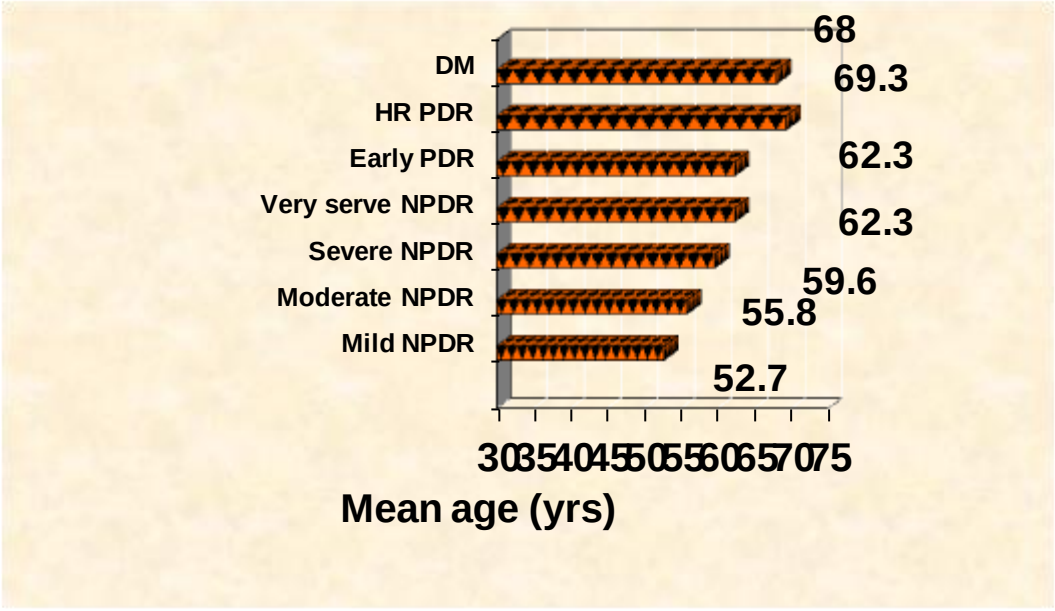


Table 14: Retinopathy and Nephropathy:

Patients with mild NPDR showed 100% Normoalbuminuria , moderate NPDR patients showed 97.6% Microalbuminuria and 2.4% showed Microalbuminuria and patients with severe NPDR, very severe NPDR, early PDR, HR PDR, DM showed 100% Macroalbuminuria.

Retinopathy	**Nephropathy**					
	Normo albuminuria		**Micro albuminuria**		**Macro albuminuria**	
	No.	**%**	**No.**	**%**	**No.**	**%**
Mild NPDR	34	100	-	-	-	-
Moderate NPDR	-	-	40	97.6	1	2.4
Severe NPDR	-	-	-	-	8	100
Very severe NPDR	-	-	-	-	7	100
Early PDR	-	-	-	-	4	100
HR PDR	-	-	-	-	3	100
DM	-	-	1	33.3	2	66.7

Retinopathy and Nephropathy:

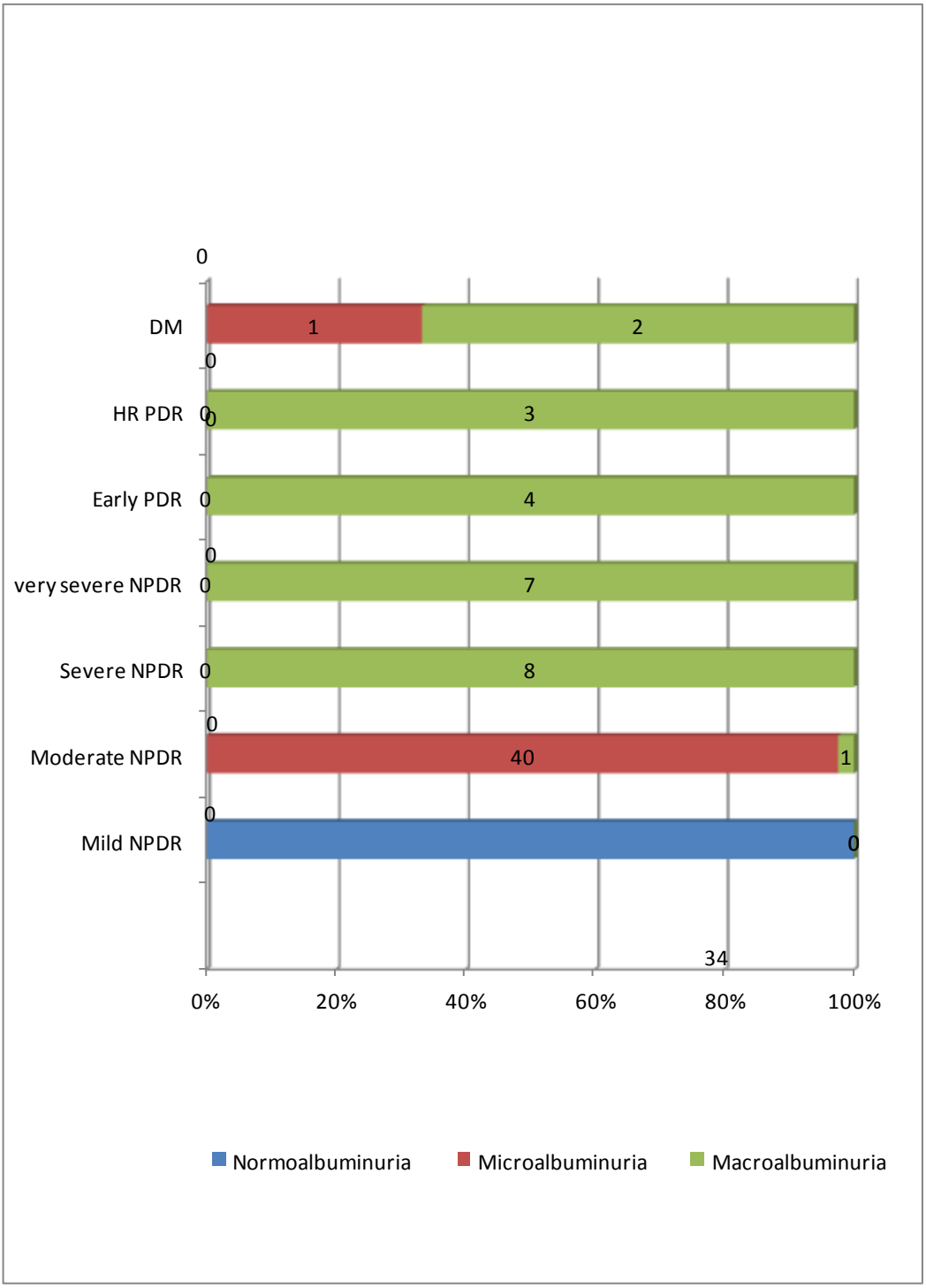


Table 15: Retinopathy and Blood Urea / Serum Creatinine:

Diabetic retinopathy has significant association with blood urea and serum creatinine. p value was proved to be significant (0.0001).

Retinopathy	Blood Urea		Serum Creations	
	Mean	SD	Mean	SD
Mild NPDR	30.0	6.2	0.92	0.28
Moderate NPDR	50.3	3.6	2.66	0.56
Severe NPDR	60.6	5.4	4.21	1.27
Very serve NPDR	66.2	4.7	5.13	1.43
Early PDR	65.5	9.3	5.13	1.27
HR PDR	62.3	3.5	6.03	0.81
DM	55.3	9.1	4.07	1.06
'p'	**0.0001 Significant**		**0.0001 Significant**	

Retinopathy and Blood Urea / Serum Creatinine

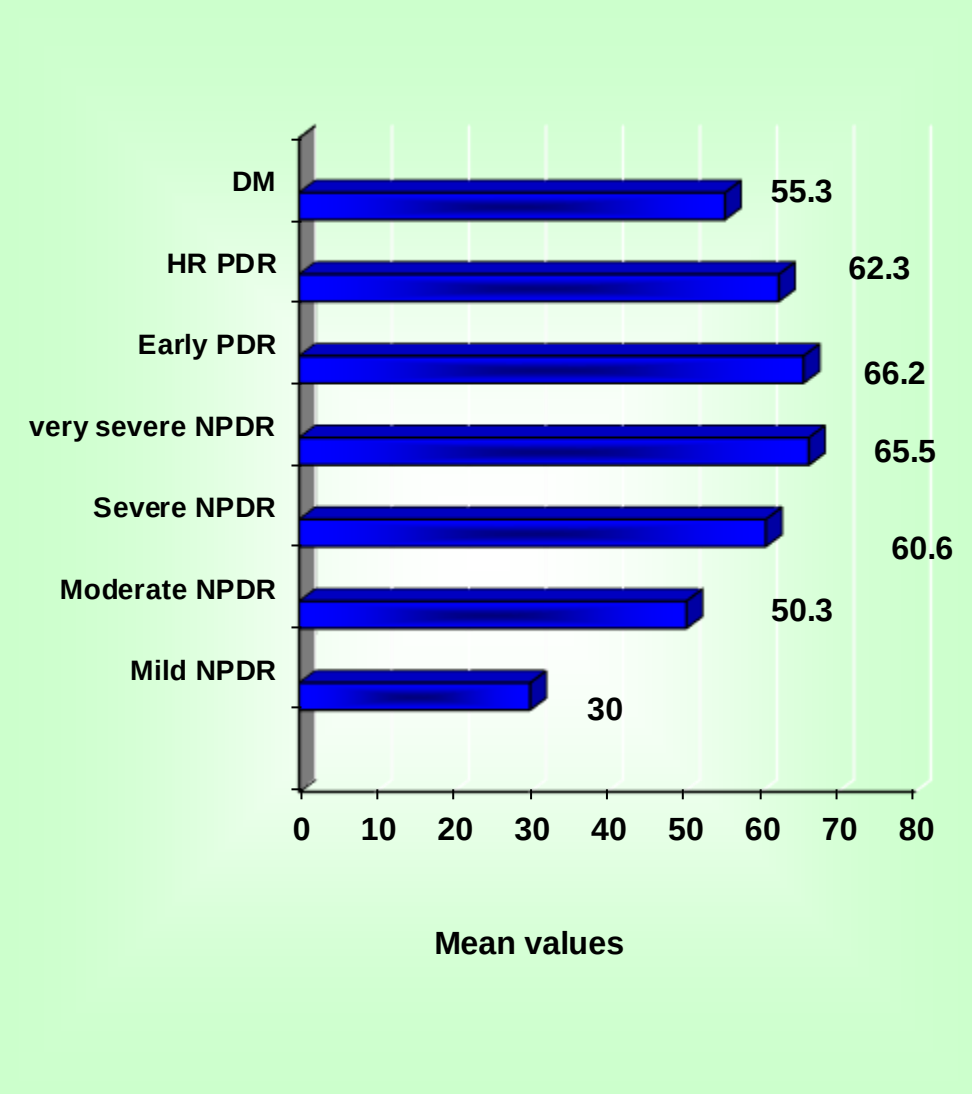
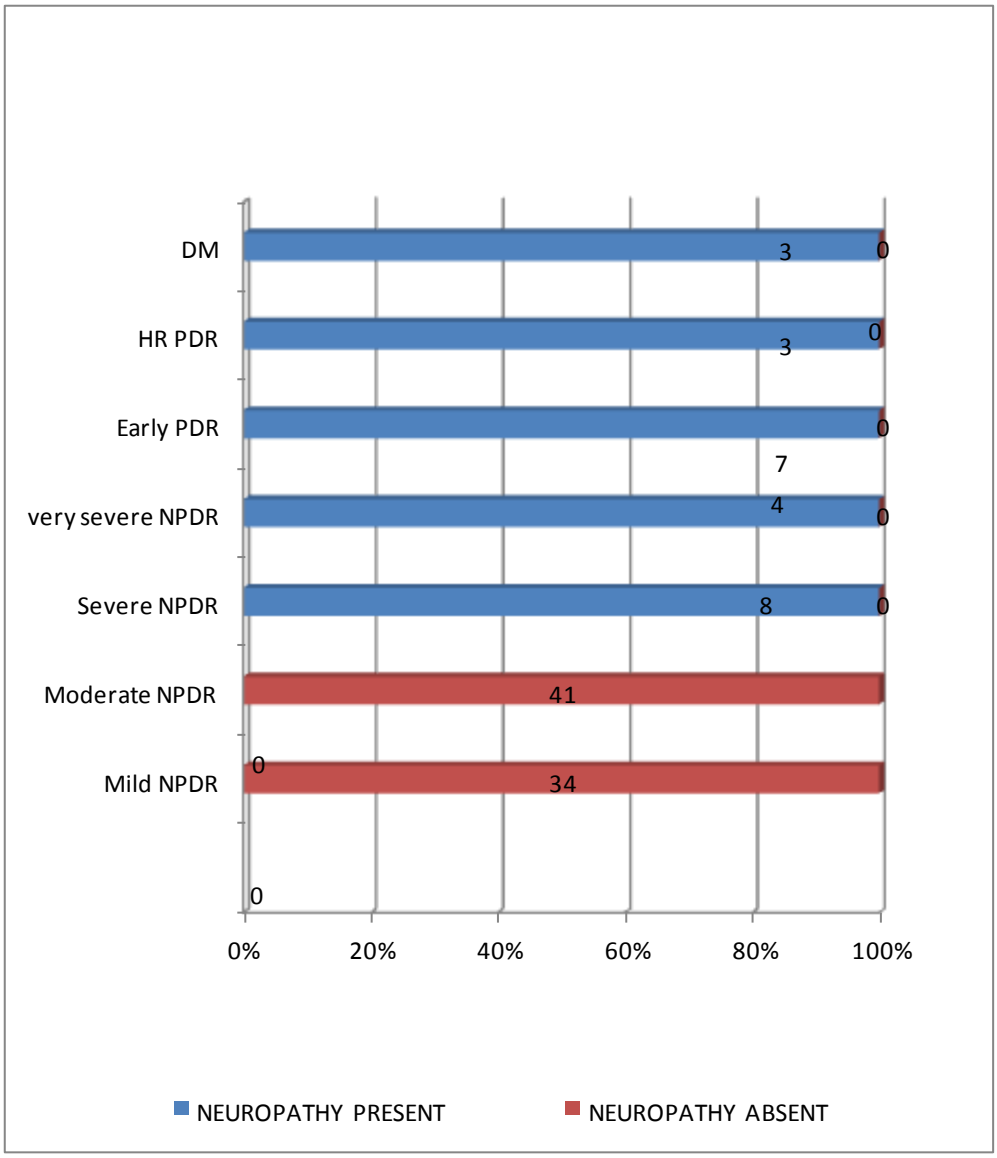


Table 16: Retinopathy and Neuropathy:

Neuropathy was 100% absent in mild and moderate NPDR and 100% present in severe NPDR, very severe NPDR, early PDR, HR PDR, DM.

Retinopathy	Neuropathy			
	Present		Absent	
	No.	%	No.	%
Mild NPDR	-	-	34	100
Moderate NPDR	-	-	41	100
Severe NPDR	8	100	-	-
Very severe NPDR	7	100	-	-
Early PDR	4	100	-	-
HR PDR	3	100	-	-
DM	3	100	-	-

Retinopathy and Neuropathy:



SUMMARY

- The average age group of patient presenting with diabetic retinopathy in this study was 50-70years – with 55% males and 45% females, with preponderance to males.
- Among the 100 patients 89% of patients were cooly, remaining patients belong to various other occupation like housewife – 4%, carpenter -1%, teacher -3%,driver – 1%, Electrician -1%, plumber -1%.
- Patients with diabetes for > 5years showed diabetic retinopathy, with the mean duration was 8.02 years.
- Visual acuity was normal in 41 eyes, 6/6p to 6/12 in 98 eyes and 6/18 to 6/60 in 61 eyes. The defective visions in these patients are due to lens changes, posterior capsular opacification and diabetic retinopathy.
- On anterior segment evaluation 54 eyes showed lens changes, 22 eyes had posterior capsular intraocular lens and 99 eyes were normal.
- On fundus examination showed 34% with mild NPDR, 41 eyes with Moderate NPDR , 8 eyes with Severe NPDR , 7%

with Very severe NPDR , 4% with Early PDR, 3% with HR PDR , 3% with DM

- 66% patients showed abnormal blood urea level and in 34% patients blood urea was normal.
- 70% patients had abnormal serum creatinine level and in 30% patients serum creatinine was normal.
- Out of 100 patients 34% patient showed Normoalbuminuria, 41% patients showed Microalbuminuria and 25% patients showed Microalbuminuria.
- On assessment of right and left leg for diabetic neuropathy 71% legs were normal and 29% legs showed abnormal.
- Patients with mild NPDR showed 100% Normoalbuminuria, moderate NPDR patients showed 97.6% Microalbuminuria and 2.4% showed Microalbuminuria and patients with severe NPDR , very severe NPDR, early PDR, HR PDR, DM showed 100% Macroalbuminuria.

- Diabetic retinopathy has significant association with blood urea and serum creatinine. p value was proved to be significant (0.0001).
- Neuropathy was 100% absent in mild and moderate NPDR and 100% present in severe NPDR, very severe NPDR, early PDR, HR PDR, and DM.

DISCUSSION

One of the major health problems that have significant impact on socioeconomic life of individual is diabetes mellitus. It results in long term damage, dysfunction and failure of various organs such as eyes, kidneys, nerves, heart and blood vessels. Diabetes mellitus develops due inadequate insulin or insensitivity of cells to insulin, classified accordingly as Type 1 (insulin dependent diabetes mellitus) or Type 2 (Non Insulin Dependent Diabetes Mellitus) respectively. Glycaemia control being the fundamental for the management of diabetes. It has been proved by Diabetes Control and Complication trial (DCCT).

The second major cause for blindness in the world is diabetic retinopathy. It has recently got greater attention because it is potentially treatable with laser photocoagulation when retinopathy is detected early and blindness can be prevented. Significant increase in risk of visual loss in patients with delayed diagnosis.

Multiple risk factors that has influence on development of diabetic retinopathy such as >5years duration of diabetes, poor glycemic control, hyperlipidemia,

hypertension, nephropathy, pregnancy, anaemia, obesity, Genetic factors - HLA type – DR3/DR4.

Most important cause for nephropathy is diabetes both in type 1 and type 2. The diabetic nephropathy incidence in type 2 patients various considerably among different ethnic group and small fraction of them progress to end stage renal disease compared to type 1 diabetic patients.

Hyperglycemia most important factor in the development of microvascular complication. Most of the previous studies showed that there is significant relationship between the presence of albuminuria and diabetic retinopathy.

Also the severity of retinopathy grading is correlated with the presence of diabetic nephropathy. Other factors like hypertension, smoking, duration has influence on the development of nephropathy.

Another important microvascular complication of diabetes is diabetic peripheral neuropathy. Diabetic neuropathy has heterogeneous clinical presentation.

Neuropathy has variety of clinical presentation such as mononeuropathies,

polyneuropathy,

autonomic neuropathy,

peripheral neuropathy

As the severity of diabetic retinopathy increases there is high risk of association of diabetic peripheral neuropathy.

CONCLUSION

- Patient with diabetic mellitus should undergo thorough history taking regarding the onset and duration of diabetes mellitus and diabetic retinopathy screening including visual acuity, fundus examination with slit lamp and +90D lens.
- Diabetic retinopathy is graded according to ETDRS classification.
- Diabetic patients should also undergo screening for diabetic nephropathy and diabetic neuropathy. From the above study conducted, it is found that
- Patients with mild NPDR showed 100% Normoalbuminuria, moderate NPDR patients showed 97.6% Microalbuminuria and 2.4% showed Macroalbuminuria and patients with severe NPDR , very severe NPDR, early PDR, HR PDR, DM showed 100% Macroalbuminuria.
- Diabetic Nephropathy was 100% absent in mild NPDR and

Diabetic Nephropathy was 100% present in moderate NPDR, severe NPDR, very severe NPDR, early PDR, HR PDR, DM

- Diabetic Neuropathy was 100% absent in mild and moderate NPDR and

  Diabetic Neuropathy was 100% present in severe NPDR, very severe NPDR, early PDR, HR PDR and

  DM.

PART

THREE

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PROFORMA

PATIENT DETAILS:

Patient Name	:		
Age	:	Date of Examination	:
Sex	:	Duration of DM	:
Occupation	:	Type of DM	:
Address & Mobile Number	:	Others	:

OCULAR EXAMINATION:

OD		**OS**
	LIDS	
	CONJUCTIVA	
	CORNEA	
	ANTERIOR	
	CHAMBER	
	IRIS	
	PUPIL	
	LENS	
	VISUAL ACUITY	

RETINOPATHY

STATUS

MILD NPDR

MODERATE

NPDR

SEVERE NPDR

VERY SEVERE

NPDR

EARLY PDR

HIGH RISK PDR

SYSTEM EXAMINATION:

	RIGHT	**LEFT**
PERIPHERAL NEUROPATHY		
Grade		
VIBRATION PERCEPTION TEST		
TEMPERATURE PERCEPTION TEST		
PIN PRICK TEST		
ANKLE JERK		
NEPHROPATHY		
Blood Urea		
Sr. Creatinine		
Urine albumin		

MASTER

CHART

S.No.	Name	Age	Sex	Occupation	Place	DM-D	Type-DM	Eye	VA	AS	Fundus	NEPHROPATHY BU	SC	UA	Leg	NEUROPATHY VPT	TPT	PPT	AJ
1	ramaiyapillai	60	m	cooly	madurai	10	2	RE	PH 6/36	IMC	moderate NPDR	52	2.1	MICRO A	R	0	0	0	0
								LE	PH 6/9	PCIOL	moderate NPDR				L	0	0	0	0
2	anwar	48	m	plumber	madurai	6	2	RE	PH 6/9	N	moderate NPDR	50	2	MICRO A	R	0	0	0	0
								LE	PH 6/12	N	moderate NPDR				L	0	0	0	0
3	vijayalakshmi	45	f	hw	madurai	5	2	RE	PH 6/6	N	mild NPDR	24	0.6	NA	R	0	0	0	0
								LE	PH 6/12	N	mild NPDR				L	0	0	0	0
4	padma	59	f	hw	dindugal	15	2	RE	PH 6/60	PCIOL	very severe NPDR	68	4.2	MACRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	very severe NPDR				L	1	1	1	1
5	karuputhevar	62	m	cooly	madurai	8	2	RE	PH 6/24	PCIOL	moderate NPDR	50	2	MICRO A	R	0	0	0	0
								LE	PH 6/36	IMC	moderate NPDR				L	0	0	0	0
6	valli	47	f	teacher	madurai	6	2	RE	PH 6/6	N	mild NPDR	33	0.8	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
7	andi	55	m	cooly	madurai	10	2	RE	PH 6/36	IMC	severe NPDR	53	3.2	MACRO A	R	1	1	1	1
								LE	PH 6/36	IMC	severe NPDR				L	1	1	1	1
8	pappathi	62	f	cooly	madurai	7	2	RE	PH 6/24	PCIOL	moderate NPDR	48	1.9	MICRO A	R	0	0	0	0
								LE	PH 6/24	PCIOL	moderate NPDR				L	0	0	0	0
9	logambal	52	f	hw	madurai	9	2	RE	PH 6/12	N	moderate NPDR	66	2.8	MACRO A	R	0	0	0	0
								LE	PH 6/12	N	moderate NPDR				L	0	0	0	0
10	peayammal	46	f	cooly	madurai	5	2	RE	PH 6/6	N	mild NPDR	26	0.6	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
11	kumar	46	m	electrician	madurai	7	2	RE	PH 6/24	N	moderate NPDR	49	4	MICRO A	R	0	0	0	0
								LE	PH 6/12	N	moderate NPDR				L	0	0	0	0
12	sagili	59	m	cooly	madurai	12	2	RE	PH 6/36	IMC	moderate NPDR	50	3.3	MICRO A	R	0	0	0	0
								LE	PH 6/12	IMC	moderate NPDR				L	0	0	0	0
13	vasanthi	47	f	cooly	madurai	7	2	RE	PH 6/60	N	severe NPDR	55	3.1	MACRO A	R	1	1	1	1
								LE	PH 6/9	N	severe NPDR				L	1	1	1	1
14	vanmagam	68	m	cooly	madurai	13	2	RE	PH 6/9	PCIOL	early PDR	64	5.1	MACRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	early PDR				L	1	1	1	1
15	nagammal	50	f	cooly	madurai	5	2	RE	PH 6/24	N	moderate NPDR	45	1.9	MICRO A	R	0	0	0	0
								LE	PH 6/6	N	moderate NPDR				L	0	0	0	0
16	andiappan	63	m	cooly	maduri	8	2	RE	PH 6/6	N	mild NPDR	33	0.8	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
17	veeranan	75	m	cooly	madurai	17	2	RE	PH 6/24	PCIOL	DM	54	4.1	MICRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	DM				L	1	1	1	1
18	rasu	69	m	cooly	madurai	6	2	RE	PH 6/9	IMC	mild NPDR	40	0.7	NA	R	0	0	0	0
								LE	PH 6/6	PCIOL	mild NPDR				L	0	0	0	0
19	rakku	53	f	cooly	madurai	10	2	RE	PH 6/6	N	mild NPDR	31	0.6	NA	R	0	0	0	0
								LE	PH 6/9	N	mild NPDR				L	0	0	0	0
20	buvana	48	f	cooly	madurai	6	2	RE	PH 6/6	N	mild NPDR	32	0.6	NA	R	0	0	0	0
								LE	PH 6/9	N	mild NPDR				L	0	0	0	0
21	andisamy	43	m	cooly	madurai	5	2	RE	PH 6/6	N	mild NPDR	29	1.2	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
22	durai	66	m	cooly	madurai	8	2	RE	PH 6/9	IMC	moderate NPDR	49	2.3	MICRO A	R	0	0	0	0
								LE	PH 6/9	IMC	moderate NPDR				L	0	0	0	0
23	lakshmi	62	f	cooly	madurai	7	2	RE	PH 6/60	IMC	moderate NPDR	59	3	MICRO A	R	0	0	0	0
								LE	PH 6/9	PCIOL	moderate NPDR				L	0	0	0	0
24	ochammal	59	f	cooly	madurai	15	2	RE	PH 6/60	PCIOL	very severe NPDR	74	3.9	MACRO A	R	1	1	1	1
								LE	PH 6/9	PCIOL	very severe NPDR				L	1	1	1	1
25	andichi	73	f	cooly	madurai	6	2	RE	PH 6/6	PCIOL	mild NPDR	22	0.7	NA	R	0	0	0	0
								LE	PH 6/9	ACIOL	mild NPDR				L	0	0	0	0
26	ramuthai	54	f	cooly	madurai	8	2	RE	PH 6/9	N	moderate NPDR	50	1.9	MICRO A	R	0	0	0	0
								LE	PH 6/12	N	moderate NPDR				L	0	0	0	0
27	kaliammal	47	f	cooly	madurai	7	2	RE	PH 6/6	N	mild NPDR	21	0.9	NA	R	0	0	0	0
								RE	PH 6/12	N	mild NPDR				L	0	0	0	0
28	deivam	54	m	cooly	madurai	10	2	RE	PH 6/6	N	moderate NPDR	49	2.2	MICRO A	R	0	0	0	0
								LE	PH 6/12	N	moderate NPDR				L	0	0	0	0
29	mariappan	45	m	cooly	madurai	8	2	RE	PH 6/60	N	moderate NPDR	52	2.2	MICRO A	R	0	0	0	0
								LE	PH 6/24	N	moderate NPDR				L	0	0	0	0
30	mayandi	62	m	cooly	madurai	6	2	RE	PH 6/6	PCIOL	mild NPDR	38	0.8	NA	R	0	0	0	0
								LE	PH 6/9	PCIOL	mild NPDR				L	0	0	0	0
31	poornam	56	f	cooly	madurai	7	2	RE	PH 6/9P	N	moderate NPDR	58	2.9	MICRO A	R	0	0	0	0
								LE	PH 6/9	N	moderate NPDR				L	0	0	0	0
32	meenachi	66	f	cooly	madurai	13	2	RE	PH 6/60	N	severe NPDR	66	5.1	MACRO A	R	1	1	1	1
								LE	PH 6/9	N	severe NPDR				L	1	1	1	1
33	meena	44	f	cooly	madurai	5	2	RE	PH 6/6	N	mild NPDR	23	0.6	NA	R	0	0	0	0
								LE	PH 6/9	N	mild NPDR				L	0	0	0	0
34	thirumee	57	f	cooly	madurai	7	2	RE	PH 6/9	IMC	moderate NPDR	51	3.1	MICRO A	R	0	0	0	0
								LE	PH 6/12	IMC	moderate NPDR				L	0	0	0	0
35	krishnan	69	m	cooly	madurai	11	2	RE	PH 6/24	PCIOL	DM	65	5.1	MACRO A	R	1	1	1	1
								LE	PH 6/24	IMC	DM				L	1	1	1	1
36	rajapandi	52	m	cooly	madurai	8	2	RE	PH 6/9	N	moderate NPDR	49	2	MICRO A	R	0	0	0	0
								LE	PH 6/24	N	moderate NPDR				L	0	0	0	0
37	solaiammal	51	f	cooly	madurai	6	2	RE	PH 6/6	N	mild NPDR	26	1.3	NA	R	0	0	0	0
								LE	PH 6/6P	N	mild NPDR				L	0	0	0	0
38	sodalai	60	f	carpenter	madurai	7	2	RE	PH 6/9	LC	moderate NPDR	48	2.9	MICRO A	R	0	0	0	0
								LE	PH 6/24	LC	moderate NPDR				L	0	0	0	0
39	mayar	53	m	cooly	theni	5	2	RE	PH 6/9	N	mild NPDR	40	0.7	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
40	peechi	50	f	cooly	madurai	6	2	RE	PH 6/6P	N	mild NPDR	33	1.4	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
41	ramalingam	64	m	cooly	madurai	5	2	RE	PH 6/9	IMC	moderate NPDR	52	2.4	MICRO A	R	0	0	0	0
								LE	PH 6/24	IMC	moderate NPDR				L	0	0	0	0
42	malliga	47	f	teacher	madurai	5	2	RE	PH 6/6P	N	mild NPDR	28	0.6	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
43	nagarajan	58	m	cooly	madurai	12	2	RE	PH 6/36	IMC	very severe NPDR	61	4.4	MACRO A	R	1	1	1	1
								LE	PH 6/60	IMC	very severe NPDR				L	1	1	1	1
44	eswaran	52	m	cooly	madurai	7	2	RE	PH 6/9P	LC	moderate NPDR	50	3.4	MICRO A	R	0	0	0	0
								LE	PH 6/24	LC	moderate NPDR				L	0	0	0	0
45	ramesh	48	m	cooly	madurai	6	2	RE	PH 6/6P	N	mild NPDR	20	0.9	NA	R	0	0	0	0
								LE	PH 6/6P	N	mild NPDR				L	0	0	0	0
46	karupayee	57	f	cooly	madurai	15	2	RE	PH 6/60	LC	early PDR	74	5.1	MACRO A	R	1	1	1	1
								LE	PH 6/60	LC	early PDR				L	1	1	1	1
47	kannaki	66	f	cooly	madurai	6	2	RE	PH 6/12	N	mild NPDR	38	0.6	NA	R	0	0	0	0
								LE	PH 6/6P	N	mild NPDR				L	0	0	0	0
48	vijaiyan	46	m	cooly	madurai	8	2	RE	PH 6/6	N	mild NPDR	38	1.2	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
49	solai	55	f	cooly	madurai	8	2	RE	PH 6/24	LC	moderate NPDR	49	3.1	MICRO A	R	0	0	0	0
								LE	PH 6/12	PCIOL	moderate NPDR				L	0	0	0	0
50	esakiammal	60	f	cooly	madurai	10	2	RE	PH 6/9	N	moderate NPDR	50	2.2	MICRO A	R	0	0	0	0
								LE	PH 6/9	N	moderate NPDR				L	0	0	0	0
51	pethiammal	54	f	cooly	madurai	14	2	RE	PH 6/9	N	severe NPDR	64	6.1	MACRO A	R	1	1	1	1
								LE	PH 6/60	N	severe NPDR				L	1	1	1	1
52	sarathy	64	m	cooly	madurai	8	2	RE	PH 6/9	IMC	moderate NPDR	51	2.9	MICRO A	R	0	0	0	0
								LE	PH 6/36	IMC	moderate NPDR				L	0	0	0	0
53	kandasamy	44	m	cooly	madurai	5	2	RE	PH 6/6P	N	mild NPDR	37	1.1	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
54	dhanam	53	f	cooly	madurai	9	2	RE	PH 6/60	N	moderate NPDR	51	2.8	MICRO A	R	0	0	0	0
								LE	PH 6/9P	N	moderate NPDR				L	0	0	0	0
55	jayaram	67	m	cooly	madurai	10	2	RE	PH 6/9	PCIOL	very severe NPDR	62	4.5	MACRO A	R	1	1	1	1
								LE	PH 6/9	PCIOL	very severe NPDR				L	1	1	1	1
56	nataraj	60	m	cooly	madurai	6	2	RE	PH 6/6P	IMC	mild NPDR	29	0.8	NA	R	0	0	0	0
								LE	PH 6/6	IMC	mild NPDR				L	0	0	0	0
57	mohammed	47	m	cooly	madurai	9	2	RE	PH 6/6	N	moderate NPDR	53	2.5	MICRO A	R	0	0	0	0
								LE	PH 6/6	N	moderate NPDR				L	0	0	0	0
58	rajammal	68	f	cooly	madurai	15	2	RE	PH 6/60	PCIOL	HR PDR	66	5.1	MACRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	HR PDR				L	1	1	1	1
59	kathiresan	47	m	cooly	madurai	7	2	RE	PH 6/6	N	mild NPDR	31	1.2	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
60	maniammal	48	f	cooly	madurai	7	2	RE	PH 6/6P	N	moderate NPDR	48	2.6	MICRO A	R	0	0	0	0
								LE	PH 6/24	N	moderate NPDR				L	0	0	0	0
61	rajagam	60	m	cooly	madurai	8	2	RE	PH 6/9	IMC	DM	47	3	MACRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	DM				L	1	1	1	1
62	vellaithai	65	f	cooly	madurai	5	2	RE	PH 6/9	PCIOL	mild NPDR	39	1.3	NA	R	0	0	0	0
								LE	PH 6/9P	PCIOL	mild NPDR				L	0	0	0	0
63	muthukani	73	m	cooly	madurai	9	2	RE	PH 6/18	PCIOL	moderate NPDR	53	2.4	MICRO A	R	0	0	0	0
								LE	PH 6/12	PCIOL	moderate NPDR				L	0	0	0	0
64	chinnan	59	m	cooly	madurai	7	2	RE	PH 6/60	IMC	severe NPDR	60	3.1	MACRO A	R	1	1	1	1
								LE	PH 6/24	IMC	severe NPDR				L	1	1	1	1
65	shanmugavel	57	m	cooly	madurai	6	2	RE	PH 6/9	LC	mild NPDR	32	0.8	NA	R	0	0	0	0
								LE	PH 6/9	LC	mild NPDR				L	0	0	0	0
66	ganesan	52	m	cooly	madurai	6	2	RE	PH 6/12	N	moderate NPDR	48	1.9	MICRO A	R	0	0	0	0
								LE	PH 6/9	N	moderate NPDR				L	0	0	0	0
67	selvaraj	67	m	cooly	madurai	8	2	RE	PH 6/9	PCIOL	very severe NPDR	67	5.3	MACRO A	R	1	1	1	1
								LE	PH 6/24	PCIOL	very severe NPDR				L	1	1	1	1
68	mari	58	f	cooly	madurai	6	2	RE	PH 6/6	IMC	mild NPDR	33	1.1	NA	R	0	0	0	0
								LE	PH 6/6P	LC	mild NPDR				L	0	0	0	0
69	sandanam	49	m	cooly	madurai	5	2	RE	PH 6/12	N	moderate NPDR	50	2.6	MICRO A	R	0	0	0	0
								LE	PH 6/24	N	moderate NPDR				L	0	0	0	0
70	papammal	60	f	cooly	madurai	7	2	RE	PH 6/12	LC	moderate NPDR	49	2.8	MICRO A	R	0	0	0	0
								LE	PH 6/6	LC	moderate NPDR				L	0	0	0	0
71	pandi	48	m	cooly	madurai	5	2	RE	PH 6/9	N	mild NPDR	31	1.2	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
72	gomathi	65	f	cooly	madurai	8	2	RE	PH 6/9	LC	moderate NPDR	47	3.7	MICRO A	R	0	0	0	0
								LE	PH 6/12	LC	moderate NPDR				L	0	0	0	0
73	pitchai	58	m	cooly	madurai	11	2	RE	PH 6/60	LC	severe NPDR	59	4.1	MACRO A	R	1	1	1	1
								LE	PH 6/60	LC	severe NPDR				L	1	1	1	1
74	angammal	61	f	cooly	madurai	6	2	RE	PH 6/9	PCIOL	mild NPDR	37	0.7	NA	R	0	0	0	0
								LE	PH 6/6	PCIOL	mild NPDR				L	0	0	0	0
75	radha	57	m	cooly	madurai	7	2	RE	PH 6/12	N	moderate NPDR	49	2.3	MICRO A	R	0	0	0	0
								LE	PH 6 9P	N	moderate NPDR				L	0	0	0	0
76	singaram	68	m	cooly	madurai	9	2	RE	PH 6/36	PCIOL	early PDR	53	3.6	MACRO A	R	1	1	1	1
								LE	PH 6/9	PCIOL	early PDR				L	1	1	1	1
77	setharam	55	m	cooly	madurai	5	2	RE	PH 6/6P	N	mild NPDR	27	1.3	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
78	palanisamy	52	m	cooly	madurai	7	2	RE	PH 6/9	N	moderate NPDR	51	4.2	MICRO A	R	0	0	0	0
								LE	PH 6/12	N	moderate NPDR				L	0	0	0	0
79	chidhambaram	70	m	cooly	madurai	10	2	RE	PH 6/9	PCIOL	HR PDR	63	6.4	MACRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	HR PDR				L	1	1	1	1
80	sethalakshmi	65	f	cooly	madurai	9	2	RE	PH 6/12	IMC	moderate NPDR	48	2.2	MICRO A	R	0	0	0	0
								LE	PH 6/9	PCIOL	moderate NPDR				L	0	0	0	0
81	solaimalai	45	m	cooly	madurai	5	2	RE	PH 6/6	N	mild NPDR	23	0.8	NA	R	0	0	0	0
								LE	PH 6/9	N	mild NPDR				L	0	0	0	0
82	masilamani	59	m	cooly	theni	10	2	RE	PH 6/9	PCIOL	moderate NPDR	47	2.9	MICRO A	R	0	0	0	0
								LE	PH 6/12	IMC	moderate NPDR				L	0	0	0	0
83	seethammal	65	f	hw	madurai	11	2	RE	PH 6/9	PCIOL	severe NPDR	59	4.4	MACRO A	R	1	1	1	1
								LE	PH 6/24	PCIOL	severe NPDR				L	1	1	1	1
84	krishnamoorthy	43	m	driver	madurai	6	2	RE	PH 6/9	N	mild NPDR	33	0.9	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
85	gurusamy	47	m	cooly	madurai	7	2	RE	PH 6/24	N	moderate NPDR	48	3.1	MICRO A	R	0	0	0	0
								LE	PH 6/12	N	moderate NPDR				L	0	0	0	0
86	annamalai	60	m	cooly	madurai	9	2	RE	PH 6/60	IMC	very severe NPDR	68	5.5	MACRO A	R	1	1	1	1
								LE	PH 6/36	IMC	very severe NPDR				L	1	1	1	1
87	rajeswari	42	f	teacher	madurai	6	2	RE	PH 6/9	N	mild NPDR	22	1.2	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
88	annakili	52	m	cooly	madurai	8	2	RE	PH 6/12	N	moderate NPDR	51	2.5	MICRO A	R	0	0	0	0
								LE	PH 6/24	N	moderate NPDR				L	0	0	0	0
89	vijayaraj	55	m	cooly	madurai	8	2	RE	PH 6/6	LC	moderate NPDR	47	2.8	MICRO A	R	0	0	0	0
								LE	PH 6/9	LC	moderate NPDR				L	0	0	0	0
90	nallathambi	56	m	cooly	madurai	12	2	RE	PH 6/24	LC	early PDR	71	6.7	MACRO A	R	1	1	1	1
								LE	PH 6/36	LC	early PDR				L	1	1	1	1
91	saraswathi	48	f	cooly	madurai	6	2	RE	PH 6/9P	N	mild NPDR	20	0.7	NA	R	0	0	0	0
								LE	PH 6/6	N	mild NPDR				L	0	0	0	0
92	shanthi	57	f	cooly	madurai	7	2	RE	PH 6/24	N	moderate NPDR	49	2.8	MICRO A	R	0	0	0	0
								LE	PH 6/12	LC	moderate NPDR				L	0	0	0	0
93	govindhan	57	m	cooly	madurai	10	2	RE	PH 6/60	IMC	severe NPDR	69	4.6	MACRO A	R	1	1	1	1
								LE	PH 6/36	IMC	severe NPDR				L	1	1	1	1
94	soman	65	m	cooly	madurai	5	2	RE	PH 6/12	PCIOL	mild NPDR	29	0.9	NA	R	0	0	0	0
								LE	PH 6/12	PCIOL	mild NPDR				L	0	0	0	0
95	mani	46	m	cooly	madurai	6	2	RE	PH 6/6	N	moderate NPDR	50	2.9	MICRO A	R	0	0	0	0
								LE	PH 6/6	N	moderate NPDR				L	0	0	0	0
96	lakshmi	66	f	cooly	madurai	8	2	RE	PH 6/12	PCIOL	very severe NPDR	65	8.1	MACRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	very severe NPDR				L	1	1	1	1
97	sempatiyan	70	m	cooly	madurai	14	2	RE	PH 6/60	PCIOL	HR PDR	59	6.6	MACRO A	R	1	1	1	1
								LE	PH 6/60	PCIOL	HR PDR				L	1	1	1	1
98	irulayee	53	f	cooly	madurai	7	2	RE	PH 6/9P	N	mild NPDR	23	1.6	NA	R	0	0	0	0
								LE	PH 6/12	N	mild NPDR				L	0	0	0	0
99	chandra	55	f	cooly	madurai	6	2	RE	PH 6/9	N	moderate NPDR	47	3.1	MICRO A	R	0	0	0	0
								LE	PH 6/9	N	moderate NPDR				L	0	0	0	0
100	vairum	57	f	cooly	madurai	8	2	RE	PH 6/9	IMC	moderate NPDR	48	2.6	MICRO A	R	0	0	0	0
								LE	PH 6/12	IMC	moderate NPDR					0	0	0	0

KEY TO MASTER CHART:

S.No – serial number

m – male, f - female

hw – house wife

DM-D – diabetes mellitus duration

Type DM – type of diabetes mellitus

RE – right eye

LE – left eye

PH – pinhole

IMC – immature cataract

PCIOL – posterior capsular intraocular lens

LC – lens changes

N – normal

VA – visual acuity

AS – anterior segment

NPDR – non-proliferative diabetic retinopathy

PDR – proliferative diabetic retinopathy

HR PDR – high risk proliferative diabetic retinopathy

DM – diabetic maculopathy

BU – blood urea

SC – serum creatinine

UA – urine albumin

MICRO A – microalbuminuria

MACRO A – macroalbuminuria

NA – normoalbuminuria

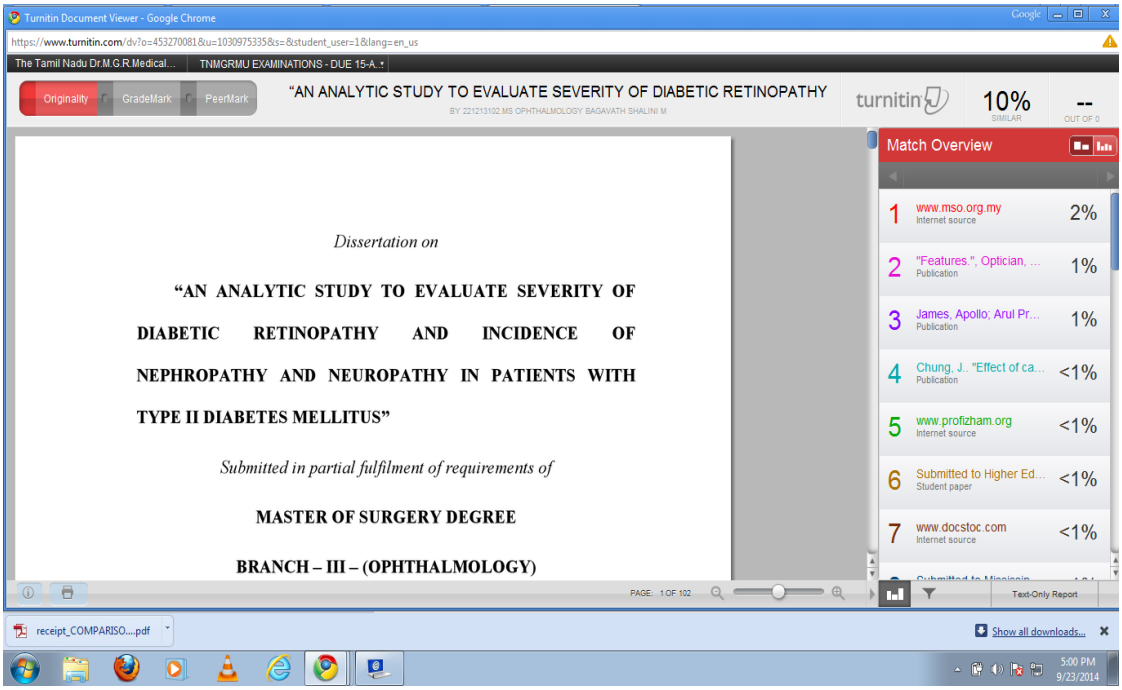
R – right, L – left

VPT – vibration perception test

TPT – temperature perception test

PPT – pin prick test

AJ – ankle jerk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