A Dissertation on

"INCIDENCE OF RETINOPATHY CHANGES IN NEW CASES OF DIABETES MELLITUS TYPE 2"

Submitted to the

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of degree of

M.S. (Branch-III)

OPHTHALMOLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI, TAMILNADU

APRIL 2013

CERTIFICATE

This is certify that study entitled **"INCIDENCE OF RETINOPATHY CHANGES IN NEW CASES OF DIABETES MELLITUS TYPE 2"** is the result of original work carried out by **Dr.Kavitha.K**, under my supervision and guidance at STANLEY MEDICAL COLLEGE, CHENNAI The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of **M.S Degree in Ophthalmology**, course from May 2010 to April 2013 at the Stanley Medical College, Chennai.

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DECLARATION

I hereby declare that this dissertation entitled **"INCIDENCE OF RETINOPATHY CHANGES IN NEW CASES OF DIABETES MELLITUS TYPE 2"** is a bonafide and genuine research work carried out by me under the guidance of **Prof. Dr.K.Kanmani**, M.S., D.O., Associate Professor, Department of Ophthalmology, Government Stanley Medical College and Hospital, Chennai – 600 001.

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Finally, I am deeply indebted to all my patients for their sincere cooperation for completion of this study.

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Incidence of retinopathy changes in newly detected Diabetes mellitus patients – A Hospital based Study

Principal Investigator : Dr.K. Kavitha

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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.07.2011 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
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5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

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IEC, SMC, CHENNAI

INCIDENCE OF RETINOPATHY CHANGES IN PATIENTS WITH NEWLY DETECTED DIABETES MELLITUS-TYPE-II

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PART-I

PART-II

PART-I

INTROUCTION

DIABETES MELLITUS

Diabetes mellitus belongs to a type of metabolic disease characterised by hyperglycemia resulting from [1]

- Defects in insulin secretion
- Reduction in the effectiveness of secreted insulin
- Combination of the above

The raised blood sugar levels thus produces the characteristic symptoms of

- polyuria
- polydipsia
- polyphagia .

Diabetes mellitus (DM) mainly fall into three categories

Type 1 DM

It is an autoimmune disease due to which the immune system of ones own body attacks and destroys the insulin-producing cells of the pancreas. It requires the injection insulin. This was originally called as insulin-dependent diabetes mellitus (IDDM) or juvenile diabetes.

Type 2 DM

Resistance to insulin causes the cells not to respond to the secreted insulin along with a probable association with an absolute insulin deficiency. Earlier termed as adult-onset diabetes or non insulin-dependent diabetes mellitus (NIDDM).

The third category, **gestational diabetes** occurs in pregnancy, who develop raised blood glucose levels during pregnancy in the absence of previous history suggestive of raised glucose levels. It may even precede development of type 2 DM. It disappears after delivery, although it increases the chances of developing diabetes at a future date.

Other forms of diabetes mellitus include

- congenital diabetes, associated with gene defects causing insulin secretion defects,
- cystic fibrosis-related diabetes,
- steroid induced diabetes due to intake of high dose steroids and other forms.

With insulin becoming available, all categories of diabetes are now treatable, with type 2 also having being cured with medications. Both forms of the disease are chronic. Transplantation of the pancreas has been tried as an alternative but success rates were low.

Long term complications are usually high, usually manifest 10 to 20 years later, although it may be the presenting feature in a few patients. Damage to the vascular system is the main reason for complications.

The risk of cardiovascular disease is greatly increased in a patient with diabetes.

Macrovascular diseases associated are-

- ischemic heart disease
- stroke
- peripheral vascular disease.

Diabetes damages the small calibre vessels like capillaries.

Diabetic retinopathy, causes formation of new blood vessels in the retina, thus producing symptoms of decreased visual acuity and sometimes potential blindness.

Diabetic nephropathy leads to chronic scar formation in the kidneys resulting in loss of proteins in the urine, culminating in chronic kidney disease.

Diabetic neuropathy usually produces symptoms like numbness, tingling and pain in the feet and also causes an increased damage to the skin due to alteration in sensation.

Vascular disease affecting the legs together with neuropathy results in an increases risk of developing diabetes-related foot problems such as diabetic foot ulcers, which are chronic, making treatment difficult ,ultimately leading to amputation of the affected part.

OCULAR MANIFESTATIONS OF DIABETES MELLITUS

S.No.	STRUCTURE	MANIFESTATIONS
1.	Lids	Xanthelasma and recurrent stye or internal hordeolum, chalazion
2	Conjunctiva	Telangiectasia, sludging of the blood in conjunctival vessels and subconjunctival haemorrhage
3	Cornea	Pigment dispersal at back of cornea, decreased corneal sensations (due to trigeminal neuropathy), punctate kerotapathy, Descemet's folds, higher incidence of infective corneal ulcers and delayed epithelial healing due to abnormality in epithelial basement membrane
4	Iris	Rubeosis iridis (neovascularization)
5	Lens	Snow-flake cataract in patients with IDDM, posterior subcapsular cataract, early onset and early maturation of senile cataract
6	Vitreous	Vitreous haemorrhage and fibre- vascular proliferation secondary to diabetic retinopathy Posterior vitreous detachment, Asteroid bodies.

7	Retina	Diabetic retinopathy and lipaemia retinalis, Decreased contrast sensitivity and colour vision.
8	Intraocular pressure	Increased incidence of POAG, neovascular glaucoma and hypotony in diabetic ketoacidosis (due to increased plasma bicarbonate levels)
9	Optic nerve	Optic neuritis. Anterior ischaemic optic neuropathy, optic atrophy
10	Extraocular muscles	Ophthalmoplegia due to diabetic neuropathy
11	Changes in refraction	Hypermetropic shift in hypoglycemia, myopic shift in hyperglycemia and decreased accommodation

DIABETIC RETINOPATHY

DR is a microangiopathy affecting the retinal precapillary arterioles, capillaries, and venules.2 Loss of vision caused by DR,can be prevented by timely diagnosis . One of the essential components of diabetic care is to diagnose the earliest signs of DR, based on which, early preventive and treatment methods can be implemented,thus arresting the disease pregression and its sequelae.

The development of microvascular complications in diabetes depends on the level of control of blood gloucose and blood pressure .

Various modalities available for the treatment of severe non proliferative and proliferative DR are

- laser photocoagulation
- ,anti-VEGF drugs
- vitrectomy.

Raised levels of glycosylated haemoglobin indicates that the prior control of blood glucose levels were not satisfactory.[2]

Thus leading on to

- retinopathy
- cardiovascular disease
- nephropathy .

It would be worthwhile to quote Sir. Stewart Duke Elder's words on diabetic retinopathy. "It is one of the major tragedies of ophthalmology in the present generation, always common and rapidly becoming still more common, affecting the young as well as the aged, predictable but not preventable and relatively untreatable, chronic and progressive in its course and leading to blindness in a distressing % of cases". Bilateral involvement is a common feature in DR.

Facts on diabetic retinopathy

1. 85% to90% of cases of diabetes mellitus eventually develops DR (WESDR).
2. Leading causes of blindness in working age group world wide[3 .4]
3. Timely treatment can prevent upto 60% to 70% of visual loss.

Magnitude of problems in our country

- According to latest WHO report India has31.7 million diabetic subjects currently.This number is expected to increase to79.4 million by 2030[5]
- The incidence of diabetes in Indians varies from that of Europeans by
  - Having a younger age of onset[6]
  - Obesity being less [7]
  - Stronger genetic association [8]
- These differences and the incresed prevalence of the disease in India indicate the need for more diabetic based studies .[9]

REVIEW OF LITERATURE

1856 - Eduard Jaeger - reported yellow coloured spots and extravasations that permeated part or whole thickness of the retina as part of DR changes[10]

1855 - Newly developed direct ophthalmoscope

1872 - Edward Nettleship - cystoids degeneration of the macula diabetes

1876 - Wilhelm Manz - the proliferative changes, vitreous haemorrhages and tractional retinal detachments.

1899-1978 Charles Best - extracted insulin from the Pancreas

1933 - Macherer - pars plana vitrectomy to treat vitreous haemorrhages in PDR[(11)]

1943 - Arthur James Ballantyne - diabetic retinopathy - a vascular disease

1950 - Gerhard Meyer-Schwickerath reported treatment of retinal disorders with photocoagulation.[12]

1953 - Poulsen- regression of PDR following post partum pituitary necrosis [13]

1976 - Patz – stabilization of macular oedema with argon laser photocoagulation [14]

1979 - The Diabetic Retinopathy Study Research Group - argon laser panretinal photocoagulation (PRP) and xenon arc both decrease the severity of visual loss. [15]

1985 - DRVS- early vitrectomy improves visual outcome.[16]

1993 - DCCT-Intensive therapy sloes progression of DR[17]

1995 - Early Treatment Diabetic Retinopathy study Research Group (ETDRS) – Importance of panretinal photocoagulation in severe NPDR and PDR [18]

1998 - United kingdom prospective diabetes study (UKPDS)— Strict control of blood sugar and blood pressure management in prevention of long term complications[19]

ANATOMY OF RETINA

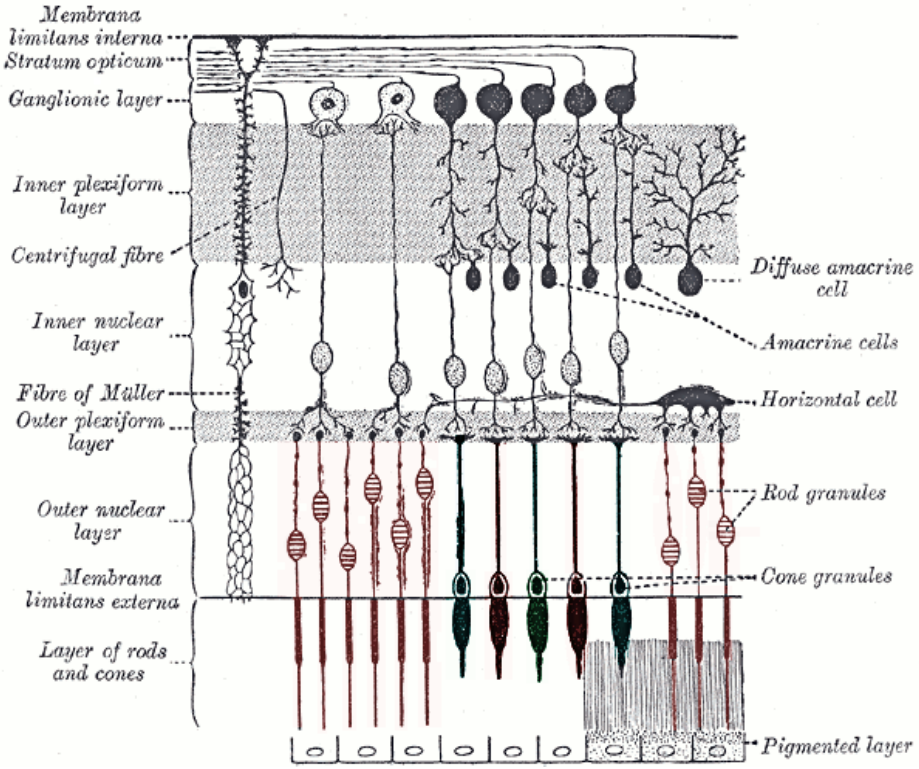
Retina is the innermost tunic of the eyeball. It is a thin ,transparent and delicate membrane. It is the most highly developed tissue of the eye. It appears purplish –red due to visual purple of rods. light-sensitive tissue which lines the inner surface of the eye.

Retina is a light sensitive layer. Light falling upon the retina is absorbed by the rods and cones which contain the photo sensitive pigments. They initiate photochemical changes which trigger a sequence of various changes which initiate visual sensations.

Total retinal area 100000 mm sq. The retinal thickness in the macular area 400 micro metres and around the fovea 150 micro metres. The retina thin out as it approaches to the equatorial region. This about 80 micro metres at ora serrata.

In retina there are several layers of neurons, which are interconnected through synapses. The photoreceptor cells are the one which are directly sensitive to light. They are the rods and cones.

ANATOMY OF RETINA



Anatomic Layers of the Retina

The layers of the retina from without inward are as follows:

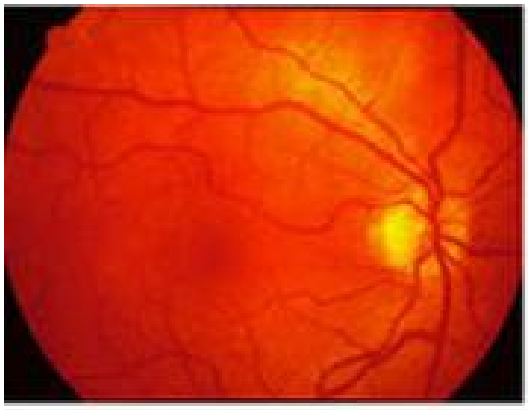
- Retinal pigment epithelium
- Layer of rods and cones
- External limiting membrane
- Outer nuclear layer
- Outer plexiform layer
- Inner nuclear layer
- Inner plexiform layer
- Ganglion cell layer
- The nerve fiber layer
- Internal limiting membrane

SPECIALISED AREA OF NEURAL RETINA

Optic Disc

It is placed 3-4 mm on to the nasal fovea. There is a central depression which has variable size, is called optic cup. The optic disc is the location where the axons of the ganglion cells exceed the eye to

NORMAL FNUDUS



form optic nerve. There are neither cones nor rods in this area which contributes physiological blind spot. At this optic disc the major blood vessels that supplies the retina are entering into the retina.

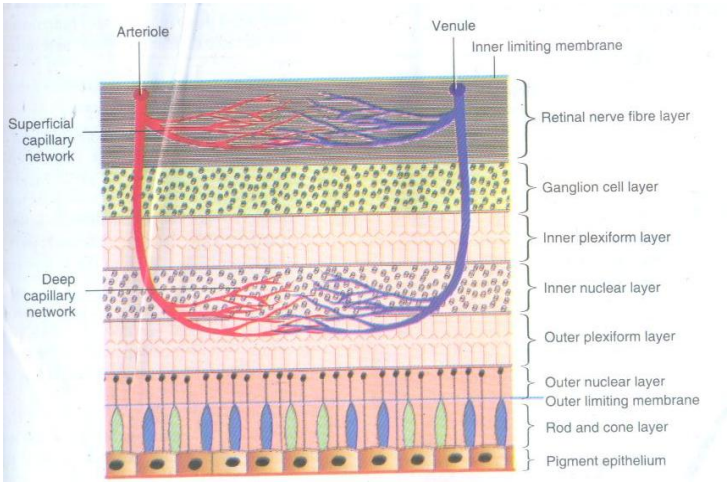
Macula

Macula represents shallow concavity lying in thickened retina 4 m.m. away from the optic disc at the posterior pole and forming a horizontal ellipse approximately 2 m.m. long 1.5 m.m. wide. In the centre of the fovea there is small depression about 0.2 m.m. in diameter called the foveola and it is at this that the greatest concentration of cones occurs and where the focal point of entering rays of light is situated.

Fovea centralis

It is the centrally depressed portion of macula .It is about 1.85 mm in diameter and 0.25mm in thickness.. They contain cone cells only each of being which is connected to one ganglion cell. Hence contributes to highest visual acuity . The fovea centralis represents visual field of 5 degrees.

BLOOD SUPPLY OF RETINA



Blood supply

The outer one third of retina are supplied by choroidal plexus. The inner two third of retina is supplied central retinal arteries. Both are branches of the ophthalmic artery, which arises from internal carotid artery.

The retinal blood vessels provide nourishment to inner retinal layers . Through diffusion from the choriocapillaris outer retinal layers get nourishment as they are avascular . Despite this dual circulation to the retina, functionally little overlap occurs, with the watershed zone at the outer plexiform layer. The central retinal artery is an end artery that has no significant anastomoses.

The retinal arteries and arterioles remain in the inner retina, while the inner nuclear layer contain only capillaries. The venous drainage of the retina usually follows the arterial supply of retina. The retinal veins (mainly venules) are present in the inner retina, where they occasionally interdigitate with their associated arteries.

Throughout the retina, the capillaries are arranged in laminar meshworks.[20]

Retinal capillaries consist of two layers—

a. inner endothelial cells

b. outer layer of pericytes.

They are 5–6μm in diameter. The pericyte:endothelial ratio is 1:1, which is relatively high compared with elsewhere in the central nervous system or body in general. The capillary basement membrane lies inbetween endothelial cells and pericytes .It is much thinner than the basement membrane that covers the two types of cells, which probably allows increased communications between the cells. The retinal capillary endothelial cells are the major component of the blood-retinal barrier. The retinal pericytes appear to be involved directly in the local control of retinal blood flow and may affect endothelial cell proliferation as well.

EPIDEMIOLOGY

Diabetic Retinopathy is one of the leading cause of blindness in both developed and developing countries. With the increase in incidence of diabetes in India, diabetic retinopathy has become more prevalent and is the sixth cause for blindness in our country. It is an important micro vascular complication in both type I and type II diabetes.

Demographic risk factors

- Age group
- General

Systemic risk factors

1. Longer duration of diabetes [21,22,23,24]
2. Severity of diabetes[24,25]
3. High systolic Bp [26,27]
4. Pregnancy[28]
5. Patients on insulin treatment

6. Nephropathy

7. Hyperlipidaemia[29,30]

8. Smoking

9. Anaemia[33,34.35]

10. Alcoholism[31,32]

Ocular factors

- ✓ In glaucoma there is reduced metabolic activity and reduced vascular perfusion.This ultimately protects from development of DR.

- ✓ Unilateral carotid artery stenosis protects the ipsilaeral eye from development of DR..

- ✓ High myopes with choroidal degeneration patients are protective against DR due to reduced retinal metabolism.

- ✓ Cataract removal exaggerate the progression of NPDR and already existing DME and lead onto the development of rubeosis.[36,37]

PATHOGENESIS OF DIABETIC RETINOPATHY

Diabetic retinopathy essentially is a microangiopathy affecting mainly the small caliber retinal vessels (i.e) precapillary arterioles and venules eventually resulting in either

1. Microvascular occlusion that causes hypoxia and development of new vessels.

2. Microvascular leakages resulting macular oedema

A number of factors at the molecular,cellular,biochemical and ulrastructural levels are implicated in the pathogenesis.

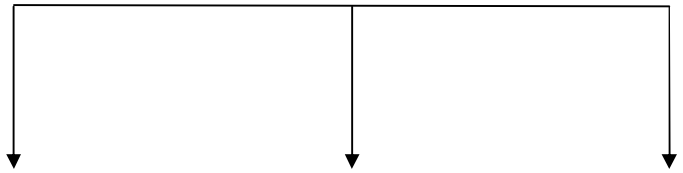
Hyperglycaemia induces these changes due to combination of factor including biochemical,haemodynamic and paracrine factors producing structural changes in the vessels including

1. Pericytes degeneration

2. Basement membrane thickening

3. Endothelial cell proliferation

Hyperglycaemia induced alternation



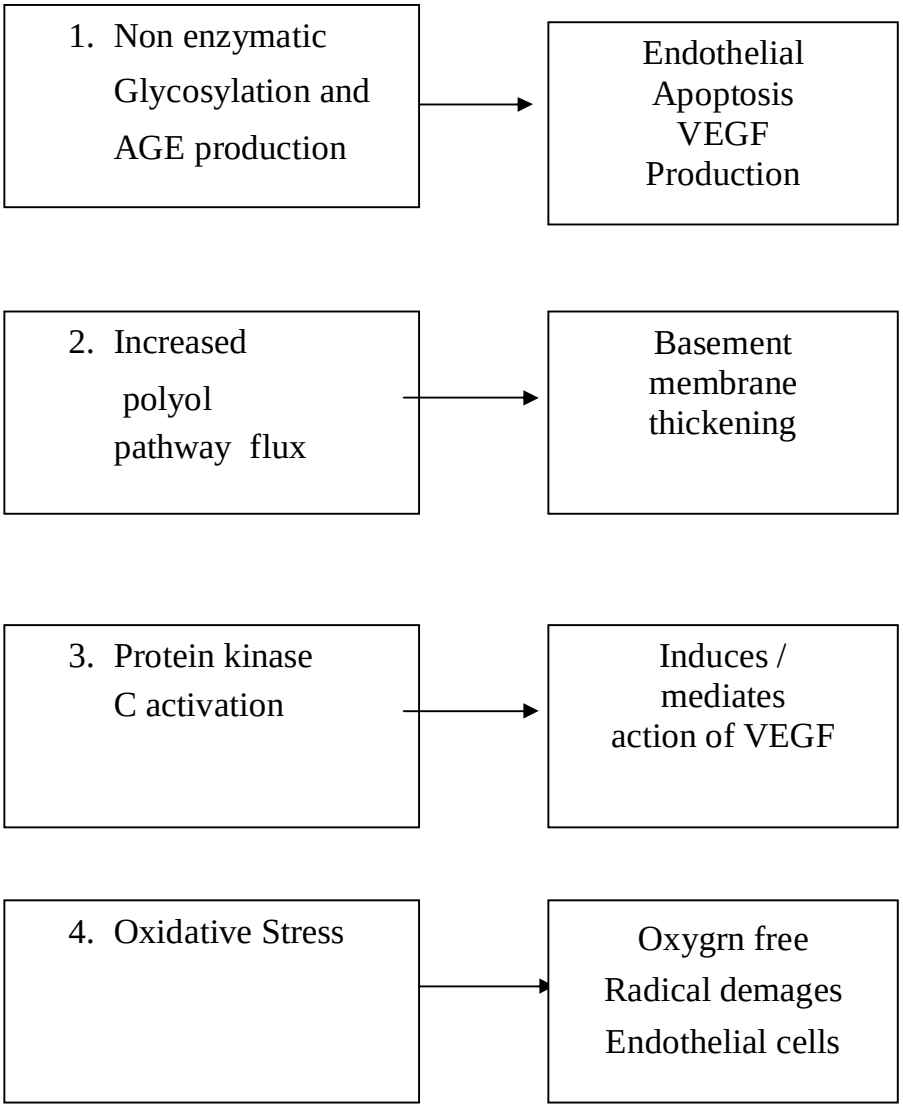
Biochemical Haemodynamic paracrine

BIOCHEMICAL ALTERATION

There are several biochemical mechanisms which include

- Non –enzymatic glycation
- Polyol pathway
- protein kinase C activation
- oxidative stress

BIOCHEMICAL ALTERATION



HAEMODYNAMIC ALTERATIONS

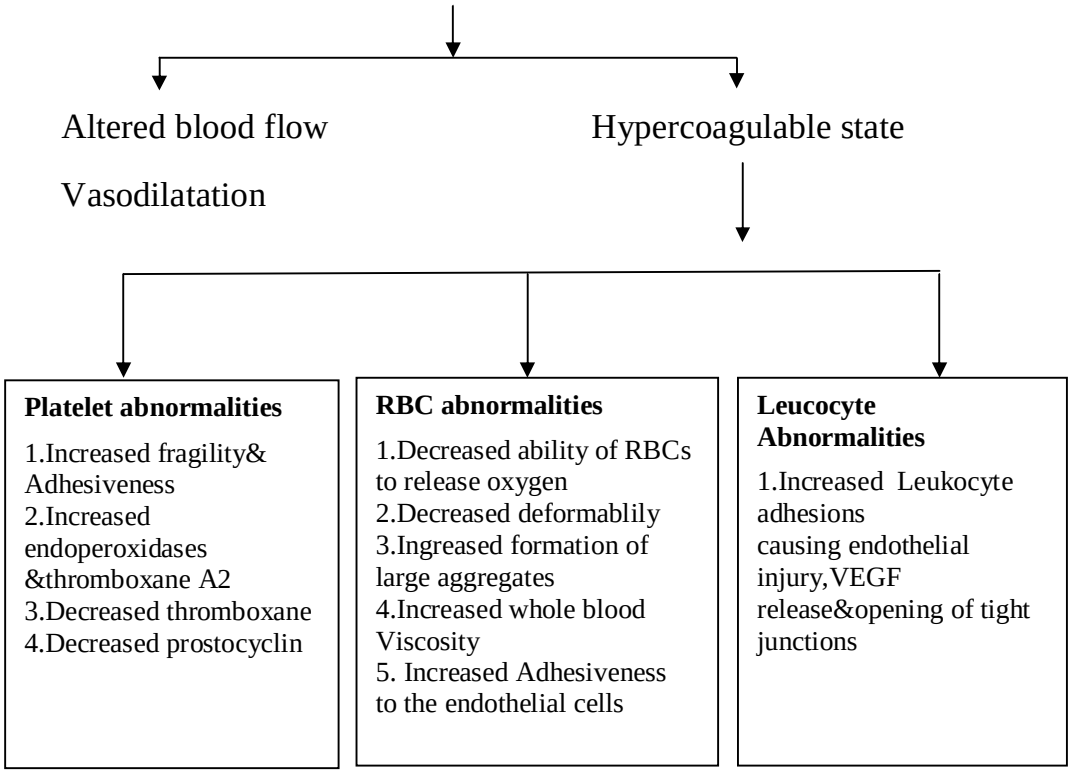
Altered blood flow

The earliest retinovascular change seen in diabetic retinopathy is vasodilatation due to abnormal tissue oxygenation.

Thickening of basement membrane associated with pericyte loss are the initial changes that occur in DR .The contractile properties possed by the pericytes allows changes in the lumen of the blood vessels, thus regulating blood flow.

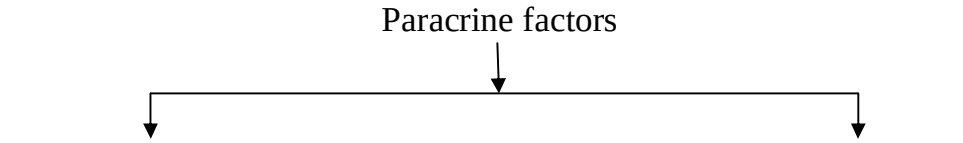
The changes in the vessel wall induce hypoxia. This induces functional changes inthe retinal vasculature resuling in a change of retinal tissue blood flow pattern. There occurs a hyperperfusion in retinal circulation of diabetic patients which leads to stress damage to the vessel wall.At the capillary level there is increased viscosity of blood,increased platelet aggregation which lead to further capillary occlusion .

Haemodynamic alteration



PARACINE FACTORS

Pathological abnormal neovascularization in patients with diabetes results due to imbalance between the the positive regulators/ pro-angiogenic and negative regulators/ anti-angiogenic factors.



Pro-angiogenic factors	Anti-angiogenic factors
1. Vascular endothelial growth factor(VEGF),	1.Endostatin
2. platelet derived growth factor(PDGF),	2.Angiostatin
3. hepatocyte growth factor(HGF),	3.Pigment endothelium
4. erythropoietin,	derived factor
5. leptin, angiogenin,	4.Genistein
6. insulin growth factor 1&2,	5.Thrombopondin 1
7. intracellular adhesion molecule 1 (ICAM1),	6. Platelet factor 4
8. angiotension 2,	
9. angiotensin converting enzyme,	

MECHANISM FOR DIABETIC MACULAR OEDEMA

1. Hyperglycaemia causes breakdown of blood-retina barrier by

   - Increased pericelullar permeablility of vascular endothelium
   - Loss of endothelial cells layer injury due to cell destruction
   - Increased transcellular transport through the endothelium

2. Increased blood flow along with increased hydrostatic pressure in the capillaries and venules are due to less resistance offered by the dilated retinal arterioles . This causes the fluid to move out of the vascular compartment as per Starling's law

3. The cellular structure of retinal capillaries consists of endothelial cells and pericytes with one to one ratio.Uncontrolled diabetes causes loss of pericytes resulting in saccular out pouching of the capillary wall clinically seen as microaneurysms,which tends to leak resulting in retinal edema.

4 Raised leukostasis in the retina affects endothelial function along with perfusion of retina and vascular permeability in diabetic patients which lead to development of DR.

MECHANISM FOR RETINAL NEOVASCULARIZATION

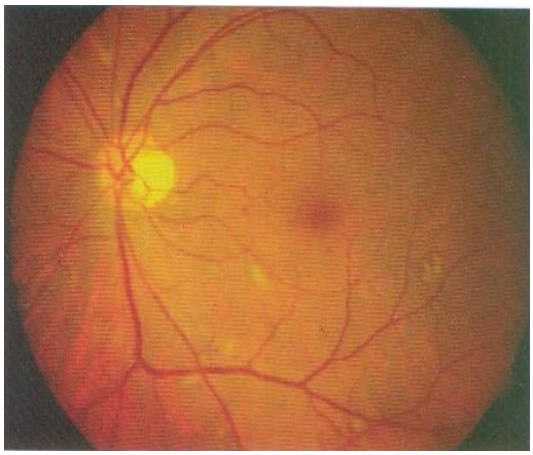
1. Astrocyte precursors form a meshwork from optic disc to retinal periphey that acts as a template for glial fibres for new vessels

2. New vessel follow the pre existing meshwork and tips of growing vessels extend along the processes of astrocytes that secrete VEGF and other angiogenic factors

3. The activated endothelial cells migrates and proliferate resulting in the formation of solid endothelial stumps into the stroma.

4. Recanalisation of the stumps results in the formation of capillary tubes with deposition of new basement menbrane and tightenig of endothelial junctions.

MILD NPDR



MODERATE NPDR

CLASSIFICATION

The most widely used classification is the ETDRS (The Early Treatment Diabetic Retinopathy Study) Classification .

It gives better understanding of the progression and management of the condition.

ETDRS Classification

I. NPDR (Non – Proliferative Diabetic Retinopathy)

Retinal hemorrhages, exudates and venous abnormalities are the hallmarks of NPDR which are classified as below :

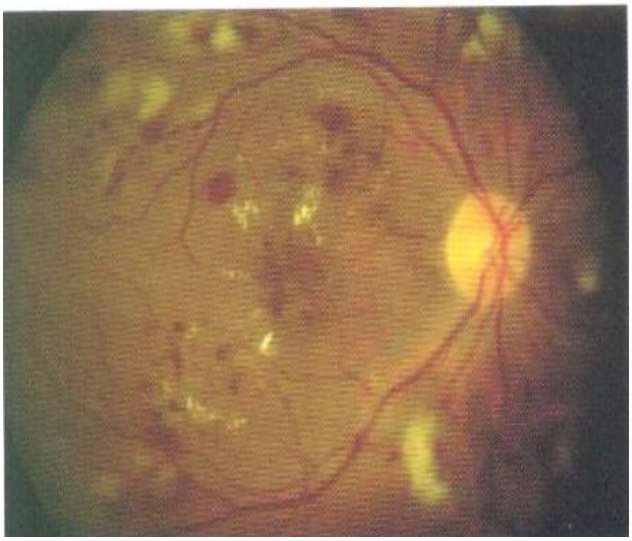
a. Mild NPDR :

At least one microaneurysm

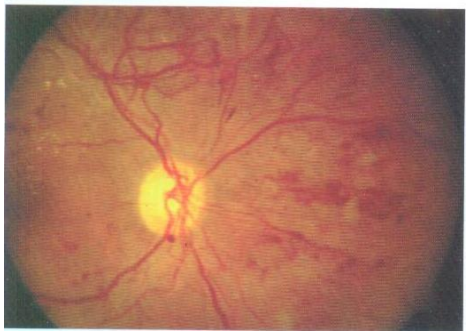
b. Moderate NPDR :

≥ Soft exudates, venous bleeding & IRMAS (Intraretinal microvascular abnormalities)

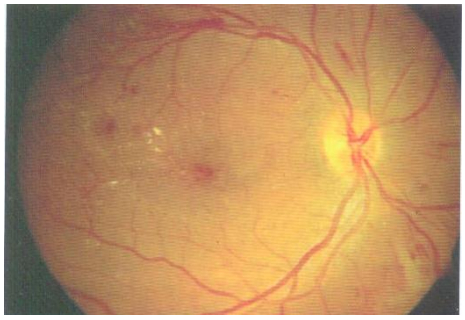
SEVERE NPDR



NVE



NVD



EARLY PDR WITH VH

PDR WITH PRE RETINAL HAEMORRAGE

c. <u>Severe NPDR :</u>

Presence of either of the following

i) Hemorrhages and microaneurysm in all 4 quadrants.

ii) Venous bleeding in 2 or > quadrant

iii) IRMA in at least 1 quadrant.

d. <u>Very Severe NPDR :</u>

Presence of either 2 or more of the criteria for severe NPDR.

II. PDR (Proliferative Diabetic Retinopathy)

It is composed of NVD (New Vessels on Disc) or NVE (New Vessels elsewhere), Vitreous or Pre-Retinal Hemorrhages and Fibrovascular proliferation.

1. Early PDR :

   New Vessels on Disc or elsewhere

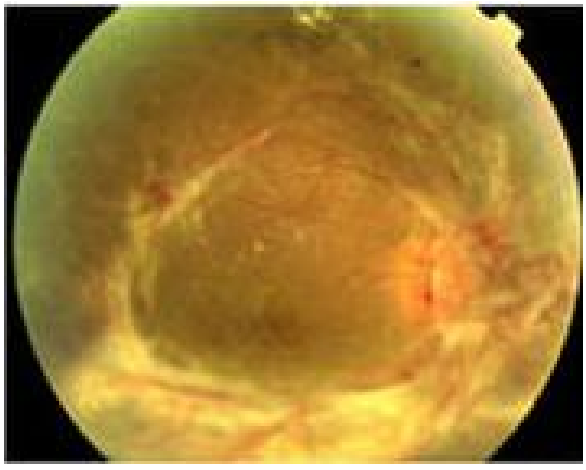
2. High risk PDR :

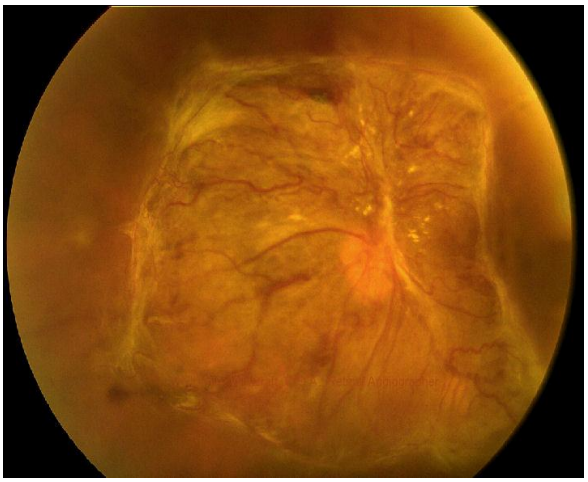
   i) NVD > 1/3 – 1/2 disc area

   ii) NVD & vitreous or pre-retinal hemorrhages

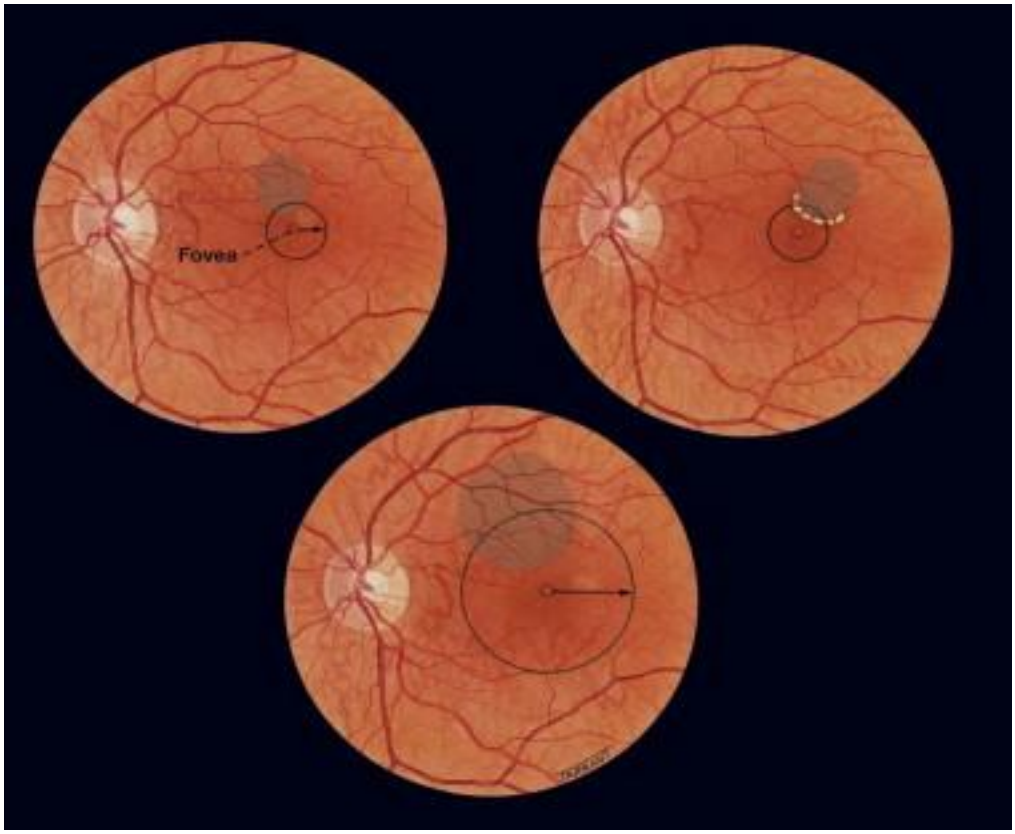
   iii) NVE > ½ disc area and vitreous or pre-retinal hemorrhages.

ADVANCED DIABETIC EYE DISEASE





CSME



3. Advanced PDR :

Extensive vitreous hemorrhages precluding grading. RD in the macula or Phthisis Bulbi or Enucleation secondary to a complication of DR.

CSME (Clinically Significant Macular Edema)

1. Retinal thickening within 500 micrometers of the centre of the macula.
2. Exudates within 500 u from centre of macula along with retinal thickening.
3. Retinal thickening 1 disc area or larger in size,any part of it located within 1 disc diameter from the centre of macula.

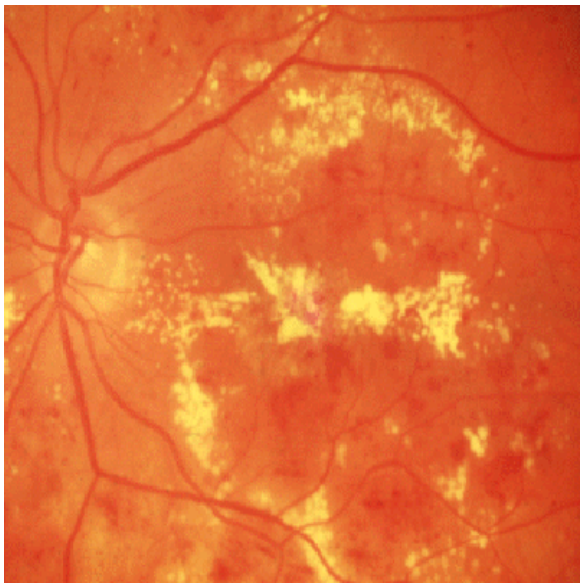
DIABETIC MACULOPATHY

Hammersmith Classification :

This classification is based upon anatomical & Pathological changes.

Type 1 : Exudate rings, microvascular lesions & focal fluorescein leakage.

CIRCINATE RETINOPATHY



Type 2 : Exudate plaques, increasing retinal hemorrhages and capillary loss.

Type 3 : Extensive central ischemia & edema, few or no exudates.

II. Selgelman's Classification :

Stage 1 : Back ground maculopathy

Stage 2 : Focal leakage on fluorescein angiogram

Stage 3 : Diffuse leakage on fluorescein angigram

Stage 4 : Cystoid degeneration of the macula.

III. ETDRS Classification :

1. Clinically Significant Macular Edema (CSME)
2. Ischemic Maculopathy
3. CME
4. Macular edema other than CSME

GRADING OF DIABETIC RETINOPATHY

International Clinical Diabetic retinopathy severity scale

Proposed disease severity level findings observable on dilated ophthalmoscopy;

Proposed disease severity level	Findings on dilated ophthalmoscopy
No apparent retinopathy	No abnormalities
Mild Non proliferative diabetic retinopathy	Microaneursms only
Moderate Non proliferative diabetic retinopathy	More than just microaneursms but less than severe non-proliferative diabetic retinopathy.
Severe Non proliferative diabetic retinopathy.	Any of the following more than 20 intraretinal hemorrhages in each of 4 quadrants; definite venous beading in 2+ quadrants; prominent intrarentinal microvascular abnormalities in 1+ quadrant and no signs of proliferative retinopathy.
Proliferative diabetic retinopathy	One or more of the following neovascularization, vitreous/ preretinal hemorrhage.,

Proposed disease severity level findings observable on dilated ophthalmoscopy	
Diabetic macular edema apperently absent. No apparent retinal thickening or hard exudates in posterior pole.	
If diabetic macular edema is present, it can be categorized as follows:	
Diabetic macular edema present	**Mild** diabetic macular edema : some retinal thickening or hard exudates in posterior pole but distant from the center of the macula
	Moderate diabetic macular edema: retinal thickening or hard exudates approaching the center of the macula but not invovling the center.
	Severe diabetic macular edema : retinal thickening or hard exudates involving the center of the macula

CLINICAL FEATURES

Microaneurysms

The first clinical manifestation of diabetic retinopathy is MICROANEURYSM which are nothing but capillary outpouchings due to loss of pericytes. It is seen as tiny red dots initially occuring temporal to fovea. The diameter varies from 12 to 100 microns..It tend to be the earliest sign of DR. Rupture of microaneurysm results in haemorrhages which can be either blot or flame shaped. Majority of microaneurysm occurs in the posterior pole and adjacent to the area of capillary nonperfusion.

Dot and blot hemorrhages

If microaneurysms rupture in the middle layers of the retina i.e at the inner nuclear and outer plexiform layers, dot and blot haemorrhage occur which can mimic microaneurysm if they are small and only FFA can differentiate a such haemoorhage from a microaneurysm.

Flame-shaped haemorrhages

These are nothing but splinter haemorrhages that occur in the larger superficial precapillary arterioles of the nerve fibre layer.

Hard exudates

Hard exudates are extracellular accumulation of lipids from the leaking vessels in the OPL of the retina.They are composed of lipoproteins and lipid filled macrophages.

Hard exudates are waxy yellow lesions with relatively distinct margins,occuring as clumps or rings surrounding the leaking microaneurysms most commonly at the posterior pole.

Cotton-wool spots

Capillary nonperfusion results in impaired axoplasmic flow and accumulation of neuronal debris within the nerve fibre layer. These accumulations are seen as cotton wool spots / soft exudates.

CWS are small, whitish fluffy superficial lesions obscuring underlying blood vessels. They are clinically evident in post equatorial region of retina.

Venous changes

Capillary non perfusion and resulting retinal ischemia can lead on to various venous changes like venous dilatation,venous loops and venous beading which frequently occurs adjacent to areas of non perfusion. Such changes corelates with the likelihood of progression to PDR.

Intra retinal micro vascular abnormalities

They are fine irregular red intraretinal lines that run from arterioles to venules without crossing major blood vessels.

Intraretinal microvascular abnormalities are arteriolar-venular shunts that run from arterioles to venules bypassing capillary bed.

IRMA is often seen adjacent to areas of marked capillary hypoperfusion.

Diabetic macular edema

Defective vision in diabetic patients is mainly due to macular oedema. It has the following features

1. Thickening of macula
2. Blurring of underlying choroidal vascular pattern.
3. Loss of FR
4. Cystoid spaces
5. Circinate retinopathy

Nonproliferative diabetic retinopathy

Occurence of at least 1 microaneurysm indicates mild NPDR. It reflects structural changes in the retina caused by the physiological and anatomical effects of diabetes.

Microaneurysms, hard exudates and superficial and deep haemorrhages occurs in moderate non proliferative diabetic retinopathy. Cotton wool spots,venous changes and intraretinal microvascular abnormalities occur less frequently than with severe NPDR.

Advancement of severe NPDR leads to increased retinal ischemia which can progress on to proliferative diabetic retinopathy,

Severe NPDR (4-2-1) is characterized by haemorrhages and microaneurysms in 4 quadrants, with venous beading in at least 2 quadrants and IRMA in at least 1 quadrant.

Proliferative diabetic retinopathy

PDR is characterised by the presence of neovascularisation occuring near the disc or elsewhere accordingly they are named as

NVD - neovascularisation on or within one disc diameter of the ONH.

NVE - neovascularisation located further away from the disc.

CLINICAL EVALUATION

I. Visual Acuity

Loss of vision mainly depends on the involvement of the macula.

II. Color Perimetry

Field charting by perimetry may reveal scotomas corresponding to areas of involvement in the fundus.

III. Color Vision

The most common defect observed is blue yellow. In diabetes the sensitivity of blue cones are depressed. These defects are best detected by Fransworth Munsell 100 hue test.

IV. Sterescopic Indirect Ophthalmoscope

This technique is of special importance because it allows to integrate the view of the entire retina.

V. Slit Lamp Biomicroscope

It is done with

- 90D

- 78D

- Hruby lens

By combining these maneuvers, it is possible to see virtually the entire retina.

VI. Direct Ophthalmoscopy

Though the area of field observed is smaller, increased magnification obtained with this method allows detailed examination of the various details of the fundus.

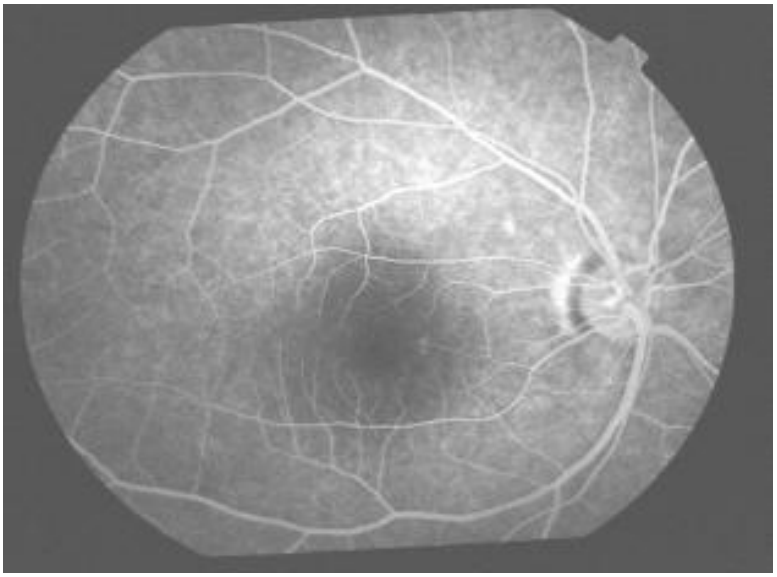
VII. Threshold Amster Grid Testing

This is a rapid, sensitive and high yield means of assessing the central fields in patents with diabetic retinopathy.

VIII. Photo Stress Test

After images and central scotomas persist after a long time. This explained the prolonged re-adaptation times in photo stress test in the affected eye.

FFA OF NORMAL FUNDUS



IX. Electrophysiology

i) Electro Retinography:

Early stages of diabetic retinopathy may reveal abnormalities of oscillatory potential in the ascending limit of the ‘b wave’. Delay of implicit time occurs as the macular edema progresses. (Brensick et al, 1984)

ii) Electro Oculography:

This test may reveal abnormal light to dark ratio (Arden’s ratio)

iii) Visually Evoked Responses:

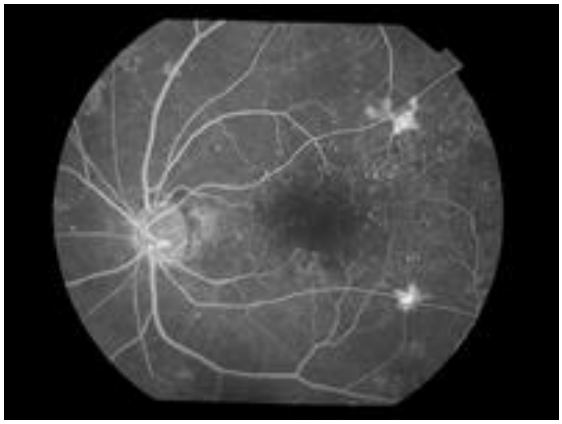
The macular disease with edema, the VER shows amplitude reduction depending on the reduced visual acuity with no change in latency.

X. Fluorescein Angiography

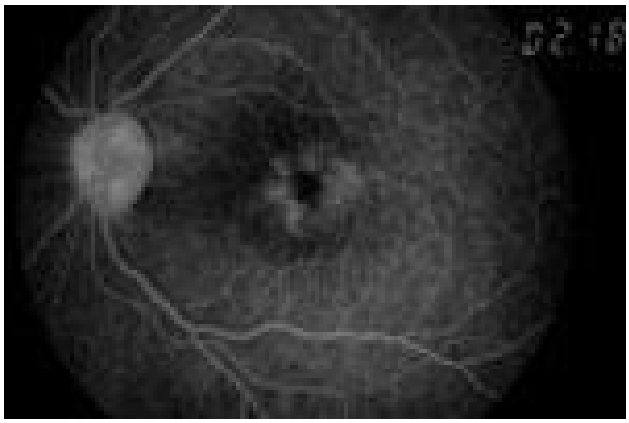
This is one of the mandatory investigations needed in diabetic retinopathy for :

- Confirmation of the diagnosis

LEAKAGE OF DYE IN FFA

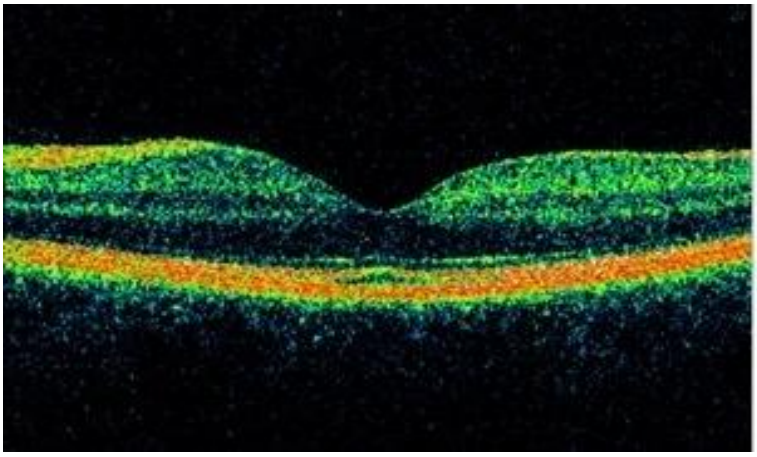


FFA IN MACULAR EDEMA

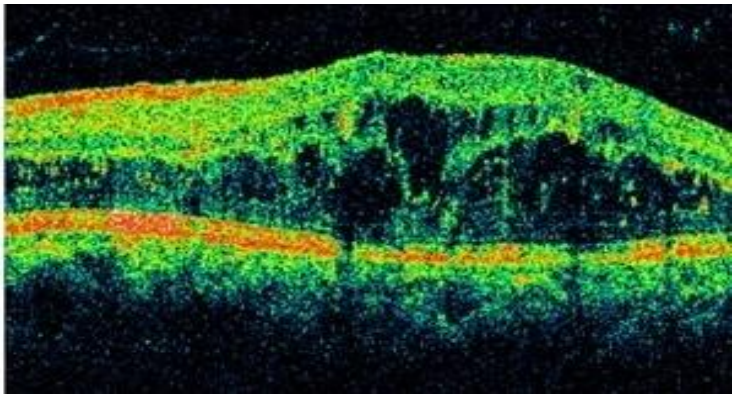


OCT

NORMAL OCT PICTURE



OCT PICTURE OF CSME



- Documentation of the various lesion
- Deciding about the management
- Followup

XI . Optical coherence tomography (OCT).

It is a non invasive non contact procedure, mainly done in case of macular edema to assess the severity and progression of disease**.**

XII. Heidelberg Retina Tomograph (HRT)

XIII.Retinal Thickness Analyzer (RTA)

MANAGEMENT

Various treatment options available for DR includes

1. Medical management
2. Lasers
3. Surgical

Medical management- includes

- Inhibitors of VEGF
- Aspirin
- Antioxidants
- Protein kinase C inhibitors
- Somatostatin
- Cyclooxygenase -2 inhibitors
- Statins
- Aldose reductase inhibitor
- Enzymatic vitreolysis

Treatment depends largely on the stage of presentation of DR :

EARLY NON PROLIFERATIVE DIABETIC RETINOPATHY

Mild/moderate NPDR –

- No treatment
- In these stages further progression can be slowed by good glycemic control.
- Control associated risk factors

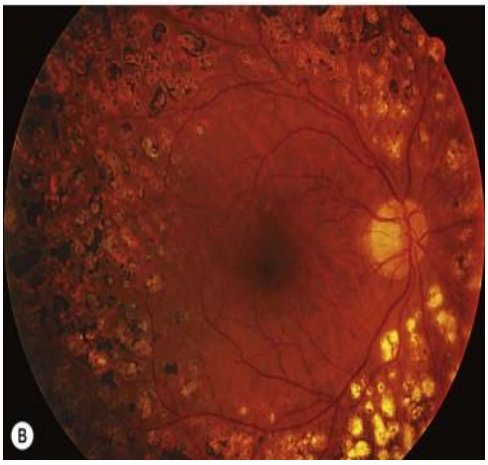
Advanced diabetic retinopathy

Laser Photocoagulation for Diabetic Retinopathy

The rationale behind using Lasers is that

- PRP destroyes the hypoxic retina, so no angiogenic factors are produced , thus reducing the rate of neovascularisation.
- PRP also allows increased oxygen diffusion from the choroid eliminating hypoxic status and thus reduces the production of angiogenic factors and neovascularisation.
- Lasers uses heat to destroy or seal an abnormal blood vessel which leaks.

POST PRP FUNDUS



- It can be done as an OP procedure under topical /local anaesthesia.

LASERS

Various lasers used are

- Argon green
- Argon blue green
- Krypton red
- Frequency doubled Ndyag laser
- Micropulse diode laser.

LASER TREATMENT FOR CSME:

1.Focal laser :

- It is given 500 to 1000 micrometers from the centre of the macula.
- Spot size – 50 to 100 micrometers
- Exposure time – 0.1 sec

2.Grid laser :

- It is given 500 micrometers from the centre of the macula and 500 micrometers from the temporal margin of optic disc.
- Spot size – 100 microns
- Exposure time – 0.1 sec
- Light intensity – minimal.

TREATMENT OF OTHER FORMS OF MACULOPATHY :

1. LASERS

- Argon green
- Argon blue green
- Krypton red
- Frequency doubled Nd-yag
- Micropulse diode laser.

2. ANTI VEGF AGENTS:

- Bevacizumab
- Ranibizumab
- Pegaptinib

3. STEROID

Triamcinolone given as intravitreal injections-

4. PARS PLANA VITRECTOMY

TREATMENT OF PROLIFERATIVE DIABETIC RETINOPATHY

Panretinal photocoagulation

Panretinal photocoagulation (PRP) implies application of laser burns throughout the retina except macular area. It can be given either through slit lamp,IDO or an EndoProbe.

PROCEDURE :

Intensity – moderate intensity

Spot size – 200 to 500 microns placed 1 spot size apart.

Start in a circumferential fashion at 500 µm from the disc and 2 DD from the fovea thus avoiding the central retina and continue peripherally.

It can be given in 2 to 3 sessions and a total of 1200-1600 burns can be given.

In cases where macular oedema and PDR coexist, laser treatment for the macular oedema are performed first, then for PDR, the PRP is spread over 3 to 4 sessions.

ANTI –VEGF AGENTS

Anti-VEGF agents like bevacizumab/ranibizumab/pegaptanib can be administered via an intra-vitreal injection, under sterile conditions under topical anaesthesia.

PARS PLANA VITRECTOMY

It is indicated in

- Severe persistent vitreous haemorrhage
- Premacular subhyaloid haemorrhage
- tractional retinal detachment,

- combined tractional and rhegmatogenous retinal detachment.

CRYOTHERAPY

- The rationale behind using cryotherapy is that it creates an aseptic chorioretinal adhesion so that oxygen supply to the retina is increased.

- It is mainly used in presence of opaque media where lasers cannot be used

Complications of Diabetic Retinopathy

1. Retinal Detachment

Separation of RPE and neuro sensory retina causes retinal detachment.

Condensation and contraction of the vitreous due to haemorrhage and fibrosis leads to tractional retinal detachment in cases of PDR. Ultimately it can result in combined rhegmatogenous and tractional retinal detachment.

2. Rubeosis iridis and rubeotic glaucoma

In PDR severe retinal ischemia results in neovascularisation of iris causing neovascular glaucoma. Later this can cause fibrovascular proliferation at the angle causing closure of the angle resulting in angle closure glaucoma.

3. Cataract

- Juvenile diabetic patients are prone to the development of snow-flake cataract.
- Senile cataract occurs earlier in diabetic patients.
- In patients with uncontrolled diabetes and fluctuating blood sugar levels, changes in fluid electrolyte balance can lead to osmotic reversible cataract.

4. Glaucoma

Glaucoma occurs due to raised intraocular pressure and it can be either primary or secondary. Primary open angle glaucoma with loss of visual field occurs commonly in diabetic patients. Secondary glaucoma particularly rubeotic glaucoma develops in patients with PDR.

5. Other ocular pathology in diabetes

- corneal epitheliopathy
- delayed epithelial wound healing following ocular surgery.

Suggested Timetables For Detailed Ophthalmologic Examination of Diabetic Patients

Age of Onset of DM	Recommended Time of First Eye Examination	Routine Minimum Follow-up
0 – 30	Within five years of Diagnosis	Annually
31 or Older	Upon Diagnosis	Annually
Pregnancy	Before conception or Early in first trimester	Every 3 months or at the discretion of the ophthalmologist

(preferred Practice Patterns Committee, AAO 2003)

Suggested Timetables For Follow-up in Diabetic Retinopathy Patients

Retinal Abnormality	Suggested Follow-up
Normal or rare micro aneurysms	Annually
Mild NPDR	Every 9 months
Moderate NPDR	Every 6 months
Severe NPDR	Every2- 4 Months
CSME	Every 2-4months (Consider laser therapy)
PDR	Every 2-3months (Consider laser therapy)

(preferred Practice Patterns Committee, AAO 2003)

PART-II

AIM OF THE STUDY

The aim of my study was to find the incidence of retinopathy changes, in newly detected DM type II. 200 eyes of 100 patients attending the ophthalmology outpatient department of the Stanley Medical College, Hospital, Chennai between the period from July 2010 to November 2012 were studied.

Special emphasis was laid in studying the following aspects;

- Age
- Sex incidence
- Presence of DR
- Staging of DR
- Presentation of complications
- Associated co morbid conditions

The results thus obtained were analysed and tabulated.

Inclusion criteria:

1. Age group between 30-60 years.
2. Both male and female patients

Exclusion criteria:

1. Type I diabetes mellitus
2. Chronic renal failure
3. Pregnant women
4. Opaque media

Materials and Methods

From July 2010 to November 2012 100 patients who were diagnosed as new case of DM type II was taken up in this study.

Diagonostic Crieteria

- Fasting blood sugar>126mg/dl
- 2 hrs post prandial blood sugar >200mg / dl.

Importance of fundus evaluation was explained to the patients. Evaluation procedure was explained in patients' own language and an informed consent was obtained

After obtaining consent, history was elicited and systemic examination done.

The following evaluation was done:

1. Relevant ocular history
2. Best corrected visual acuity
3. Slit lamp examination for anterior segment
4. Posterior segment evaluation done using indirect ophthalmoscope,slit lamp biomicroscopy with 90D and diabetic retinopathy /maculopathy was graded using ETDRS system.
5. Intraocular pressure measured with Goldmann applanation tonometer.

6. Gonioscopy

7. Visual field analysis.In selected cases octopus automated perimetry is done.

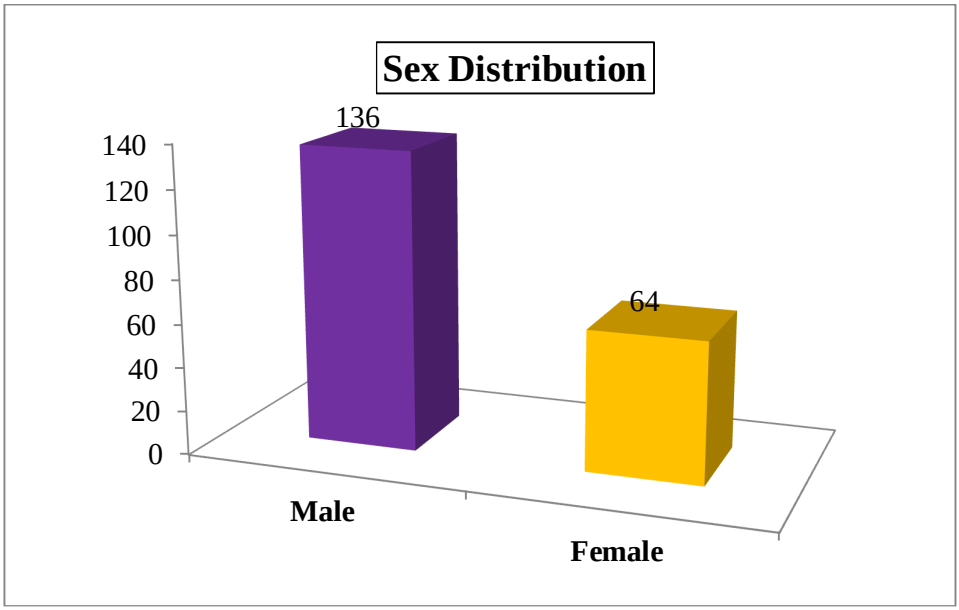
8. Fundus fluorescein angiography for selective cases.

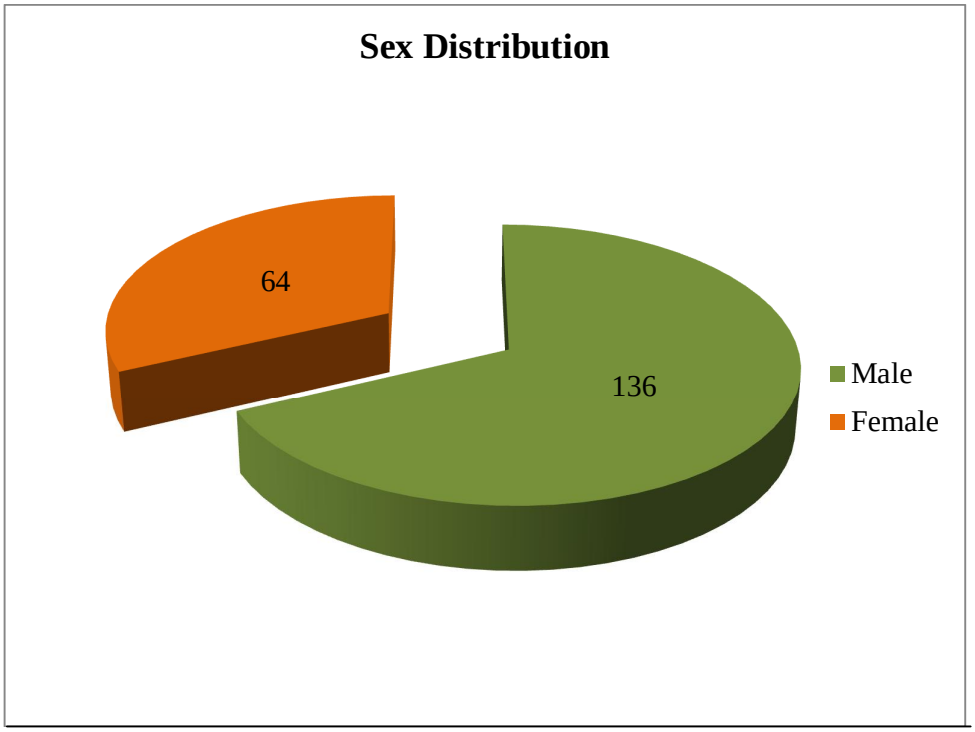
OBSERVATION

SEX DISTRIBUTION

Sex	No. of patients	Percentage
Male	136	68%
Female	64	32%

The ratio of male to female patients in our study was 2.1:1



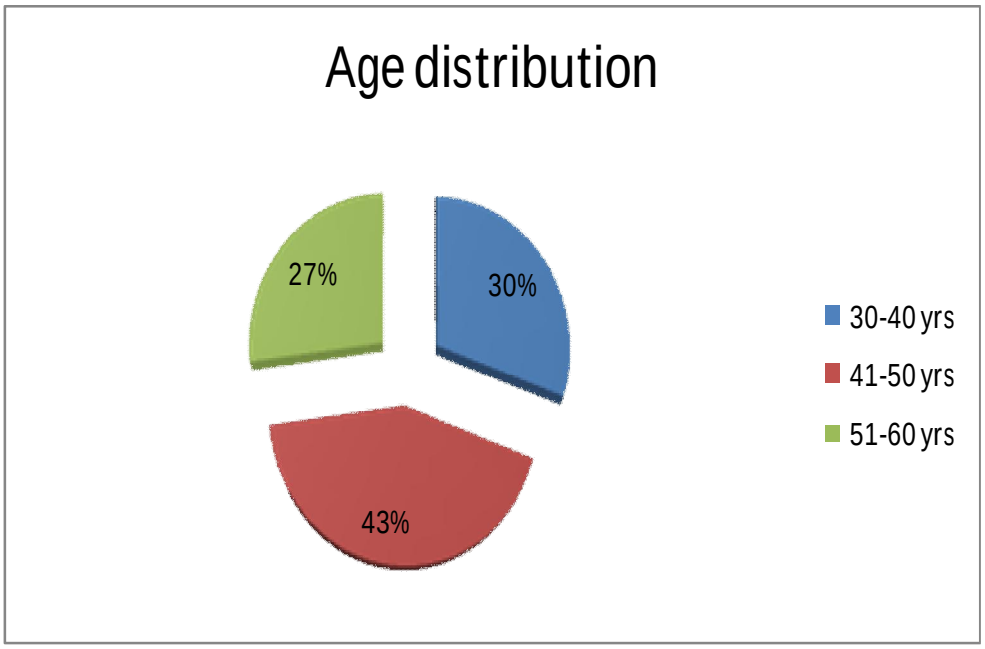

Sex Distribution
64
136
Male
Female

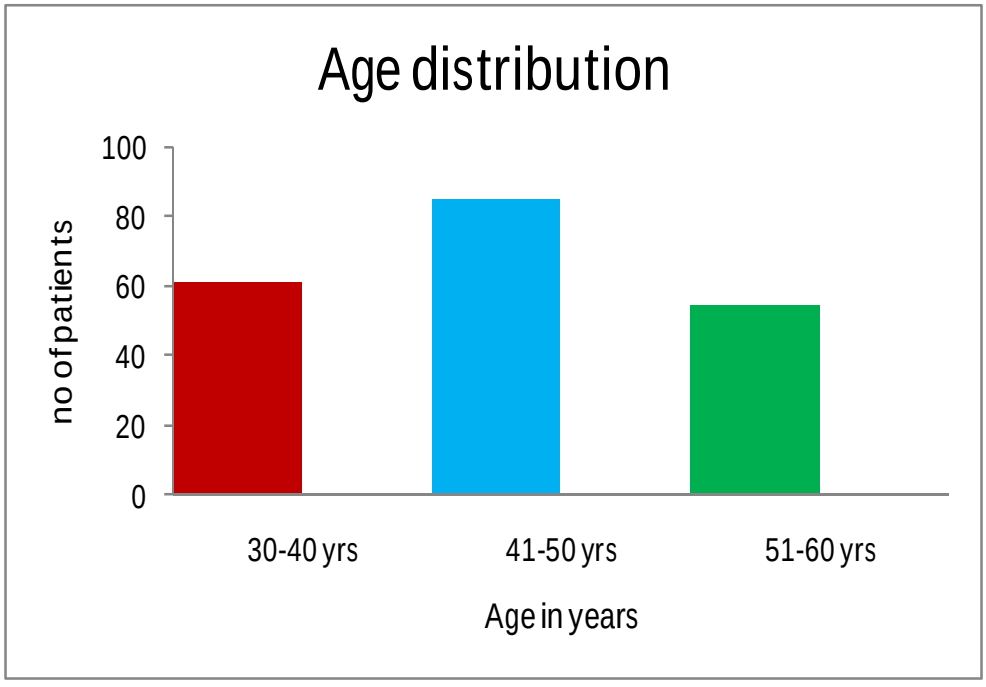
AGE DISTRIBUTION

Age	No. of patients	Percentage
30-40 yrs	61	30.5%
41-50 yrs	85	42.5%
51-60 yrs	54	27%

In this study, patients in the age group of 40 to 50 years were predominantly affected(42.5%).

Least incidence was seen in patients in the age group between 50 to 60 years, because they had undergone previous blood sugar evaluations for various purposes, hence their diabetic status detected earlier.

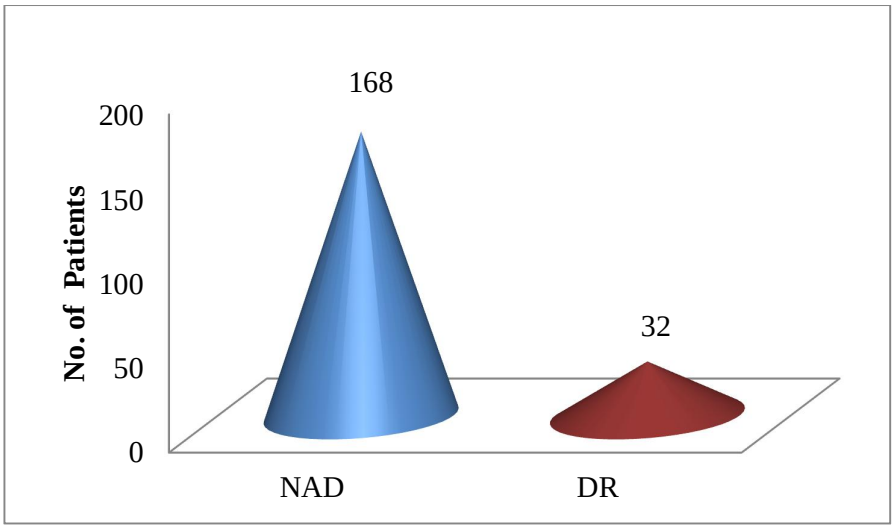

Age distribution
27%
30%
43%
30-40 yrs
41-50 yrs
51-60 yrs

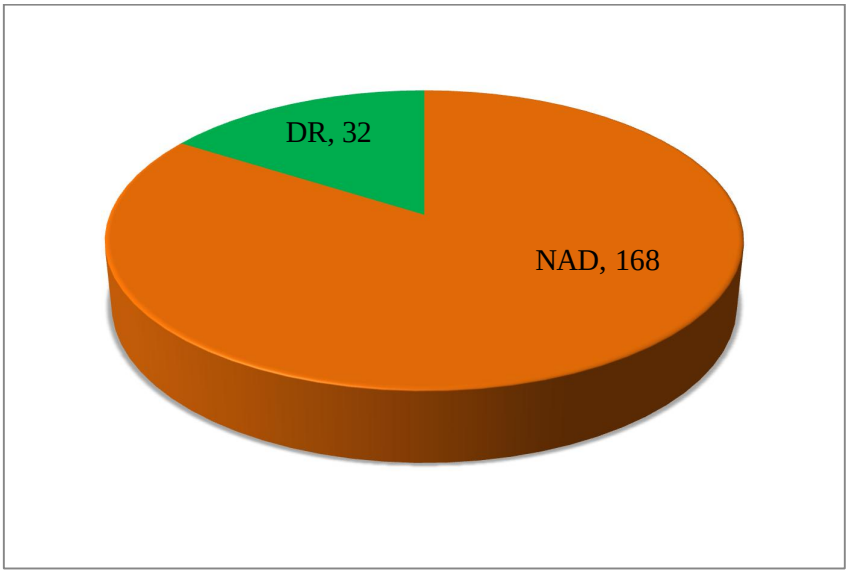

Age distribution
100
80
60
40
20
0
no of patients
30-40 yrs
41-50 yrs
51-60 yrs
Age in years

PREVALANCE OF DIABETIC RETINOPATHY

	No. of Patients	%
DR	32	16
NAD	168	84

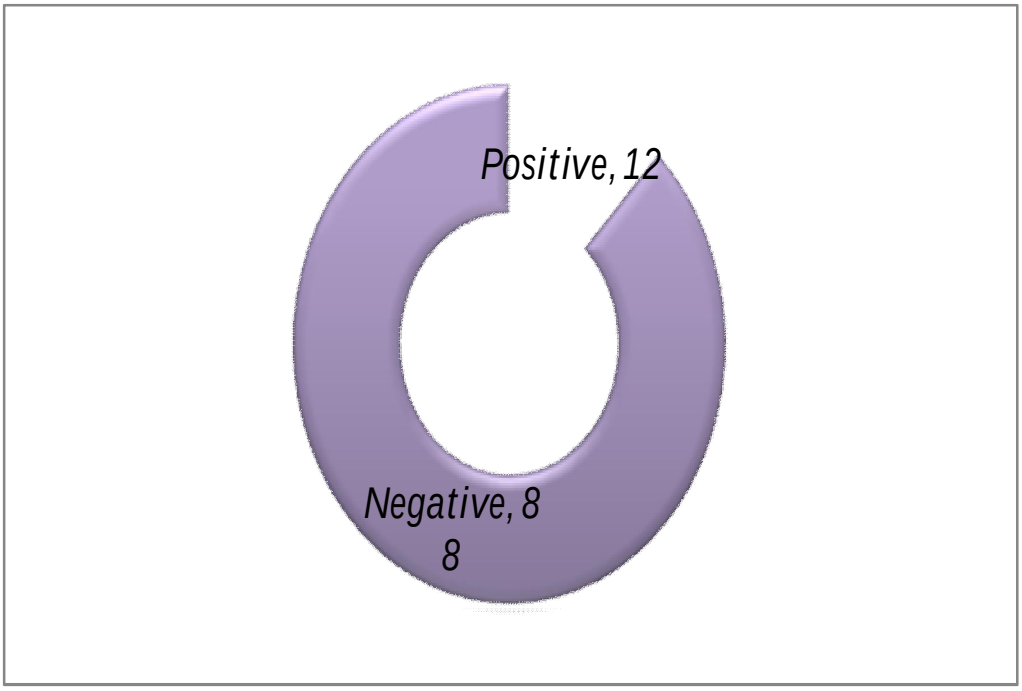
Among the 200 patients evaluated 32 were diagnosed to have DR





FAMILY HISTORY OF DIABETES MELLITUS

Family History	No. of patients	Percentage
Positive	12	12
Negative	88	88

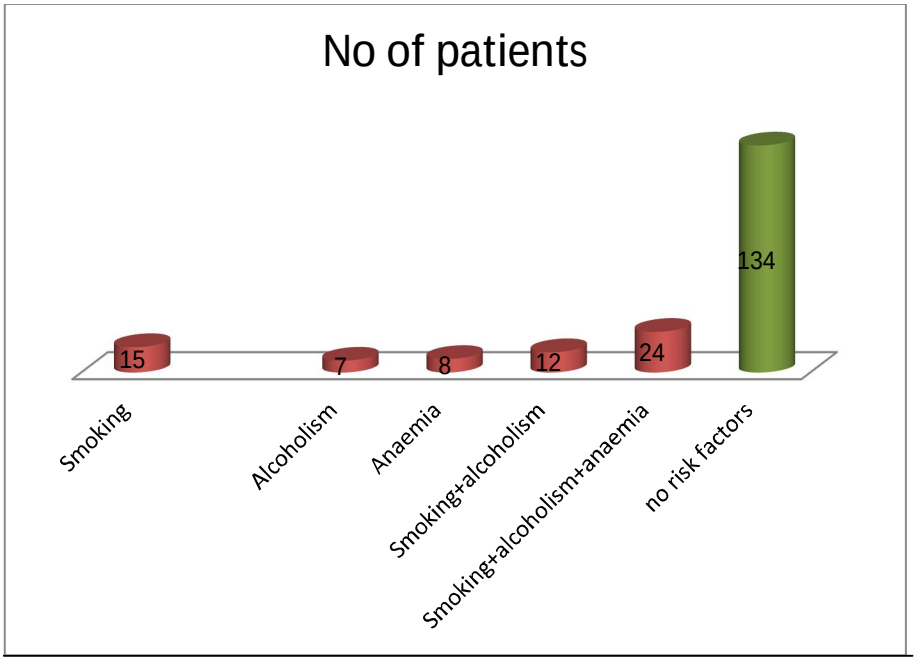


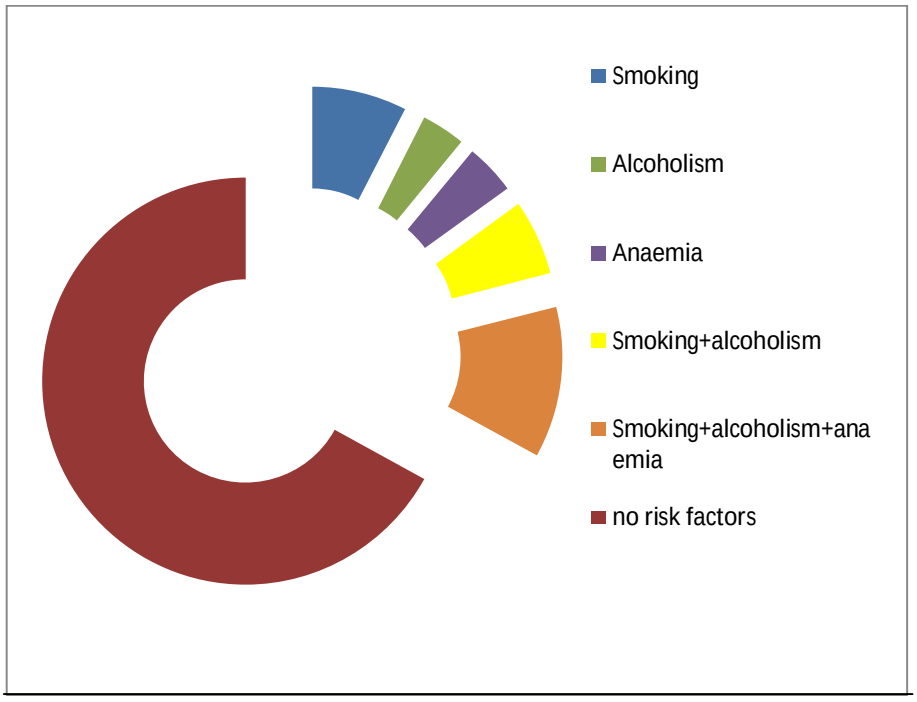
ASSOCIATED RISK FACTORS FOR THE DEVELOPMENT OF DIABETIC RETINOPATHY

Associated risk factors	No of patients	Percentage
Smoking	15	7.5%
Alcoholism	7	3.5%
Anaemia	8	4%
Smoking+alcoholism	12	6%
Smoking+alcoholism+anaemia	24	12%
no risk factors	134	67%

Various risk factors involved in the development of retinopathy includes smoking, alcohol and anaemia.

24 patients had all the three and they had various stages of DR including PDR.


No of patients
15
7
8
12
24
134
Smoking
Alcoholism
Anaemia
Smoking+alcoholism
Smoking+alcoholism+anaemia
no risk factors

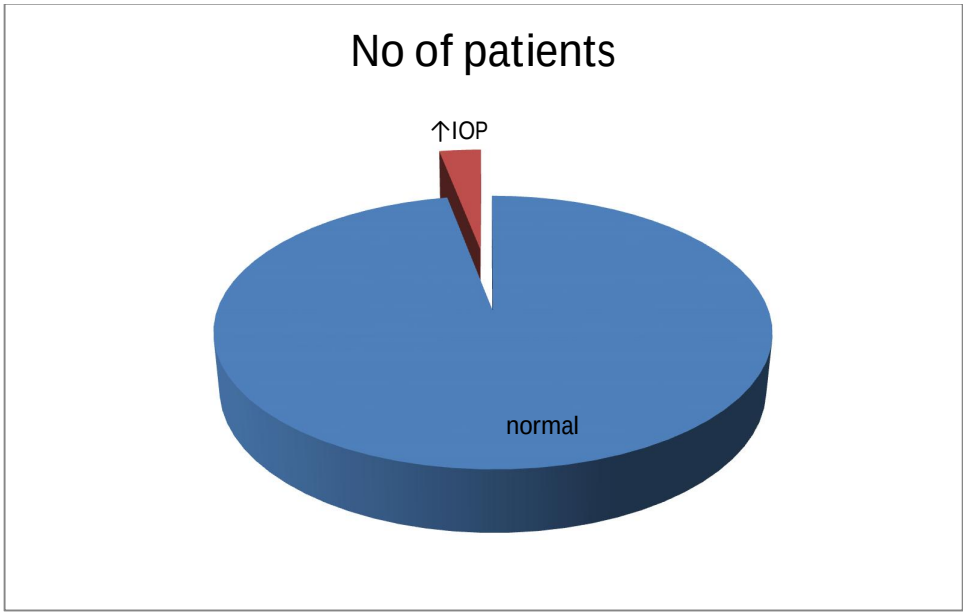

Smoking
Alcoholism
Anaemia
Smoking+alcoholism
Smoking+alcoholism+anaemia
no risk factors

INCIDENCE OF POAG

IOP	No of patients	Percentage
Normal IOP	194	97%
↑IOP	6	3%

Among the 200 patients, 6 of them had raised intraocular pressure above the normal level.

On gonioscopic examination with Goldman single mirror lens the angles were open.

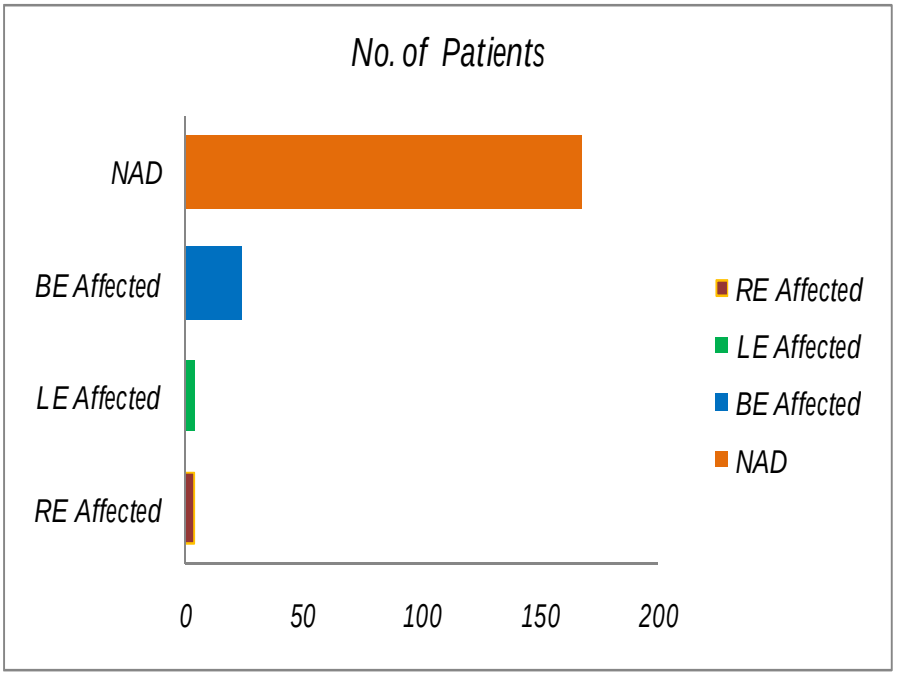


LATERALITY

Laterality	No. of patients	Percentage
RE Affected	4	2%
LE Affected	4	2%
BE Affected	24	12%
NAD	168	84%

In this study 12% of patients had bilateral DR, though the severity was asymmetrical in some patients.

4% of patients had unilateral involvement.



V/A ON PRESENTATION

V/A on presentation	No. of eyes	Percentage
6/6	184	46%
6/9 - 6/18	174	43.5%
6/24 - 6/60	42	10.5%
<6/60	-	-
HM	-	-
CFCF	-	-

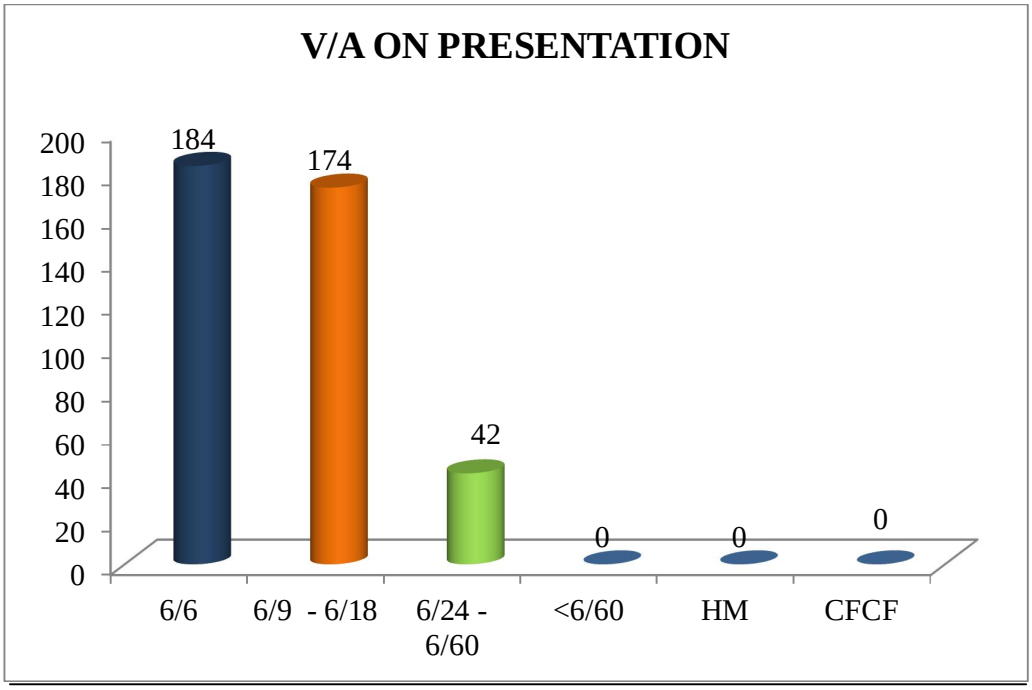
On evaluating the visual acuity of patients on first presentation majority of them (46%)had visual acuity of 6/6.

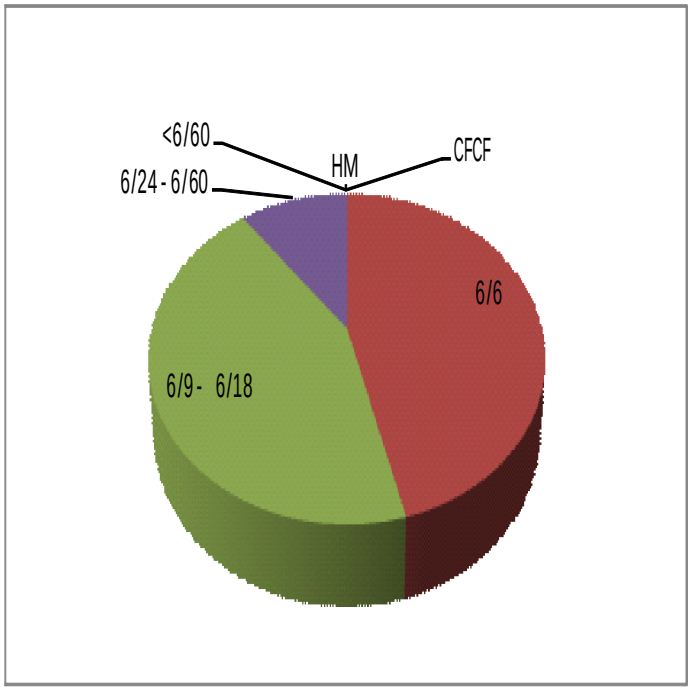
43.5% of patients had visual acuity between 6/9 and 6/18.

10.5% patients had visual acuity between 6/24 and 6/60.

The reduction in vision is due to various other causes which includes

- Cataract


V/A ON PRESENTATION
200
180
160
140
120
100
80
60
40
20
0
184
174
42
0
0
0
6/6
6/9 - 6/18
6/24 - 6/60
<6/60
HM
CFCF


<6/60
HM
CFCF
6/24 - 6/60
6/6
6/9 - 6/18

- Pseudophakia with PCO
- ARMD changes
- Other retinal pathologies
- Refractive error

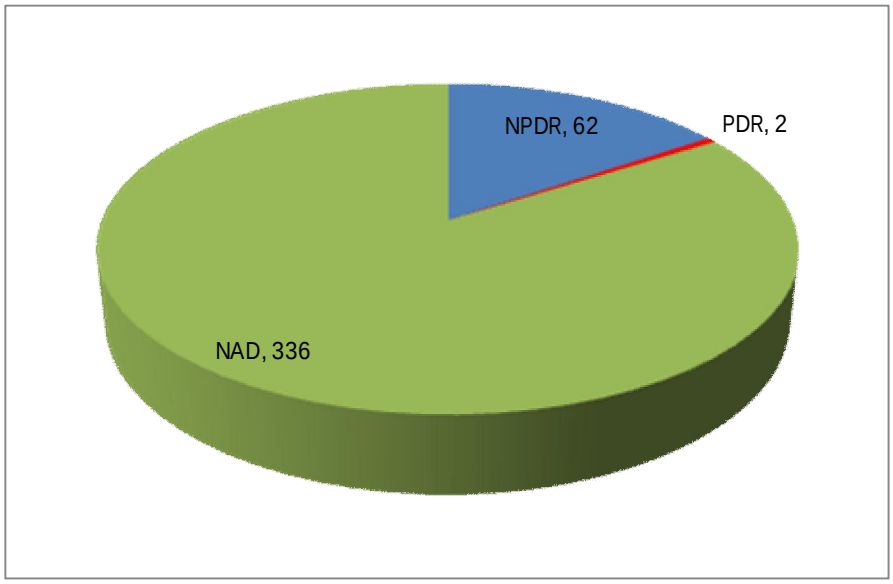
TYPE OF RETINOPATHY

Type of Retinopathy	No. of eyes	Percentage
NPDR	62	15.5%
PDR	2	0.5%
NAD	336	84%

- In our study 62 eyes had non proliferative diabetic retinopathy.
- 2 patients among the 400 had proliferative diabetic retinopathy

  One patient had NVD

  Another patient had NVD with vitreous haemorrage.



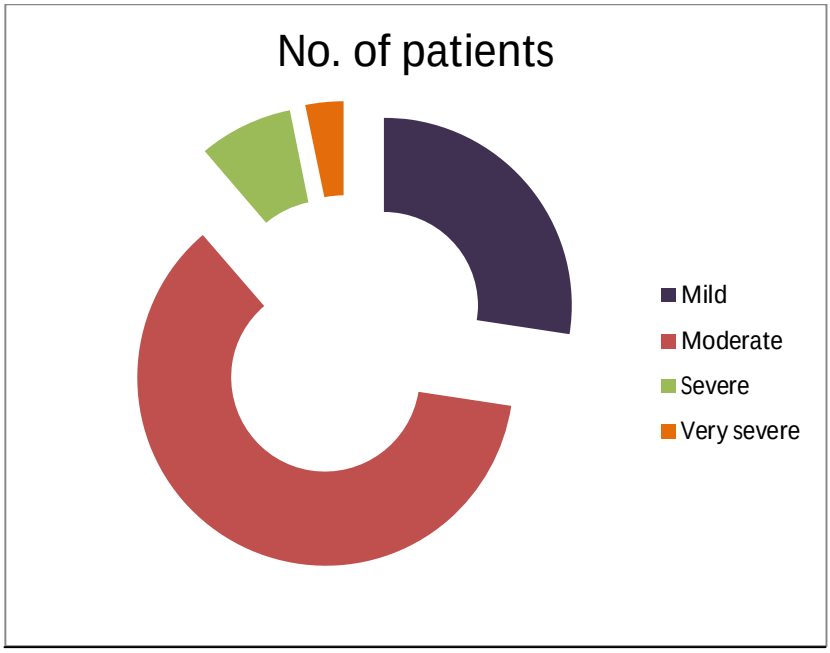
GRADING OF NPDR

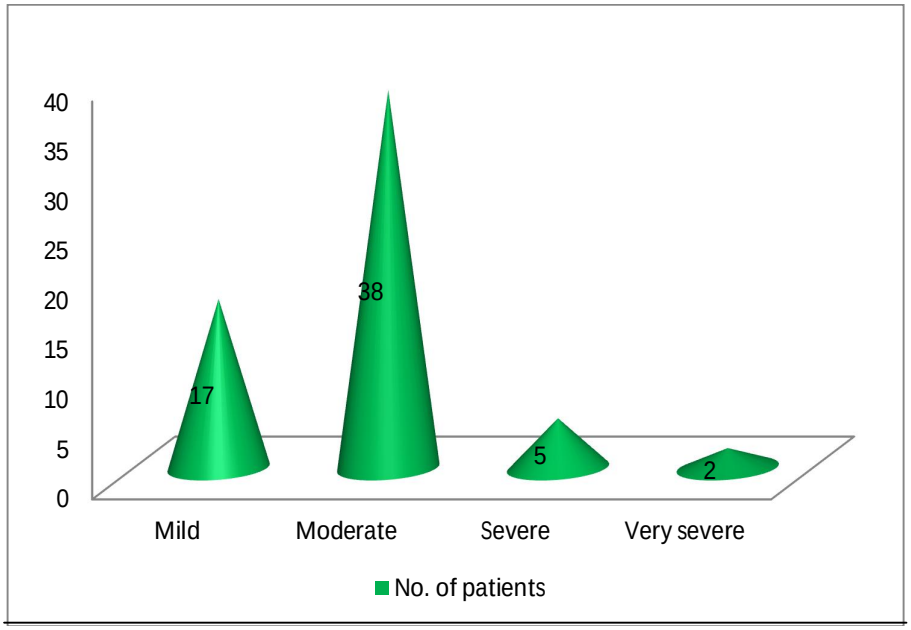
Grading of NPDR	No. of patients	Percentage
Mild	17	27.4%
Moderate	38	61.3%
Severe	5	8.1%
Very severe	2	3.2%

Majority of the patients fall into the category of moderate NPDR.

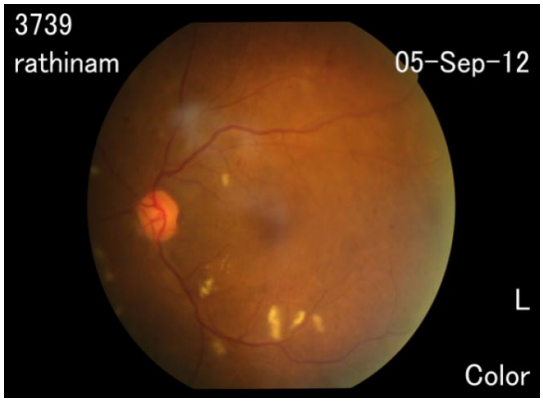
27.4% of the patients had mild NPDR

Severe and very severe NPDR were also seen in very few patients.

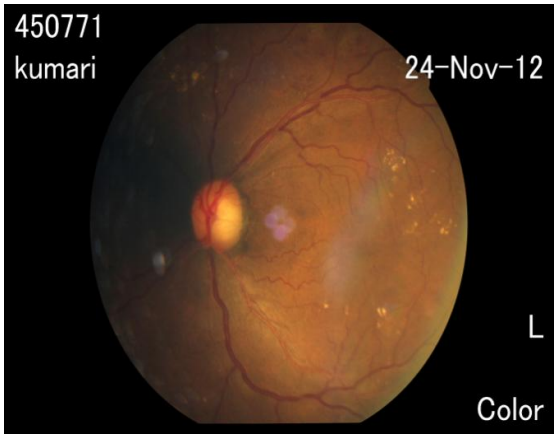

No. of patients
Mild
Moderate
Severe
Very severe


40
35
30
25
20
15
10
5
0
17
38
5
2
Mild
Moderate
Severe
Very severe
No. of patients

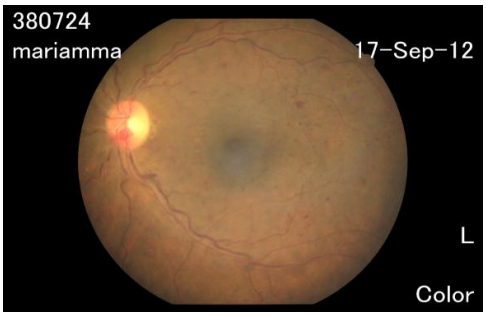
MILD NONPROLIFERATIVE DIABETIC RETINOPATHY XL--1



MODERATE NONPROLIFERATIVE DIABETIC RETINOPATHY XL--169



SEVERE NPDR XL-28



PDR WITH VH XL--152

COMPLICATIONS

Complications	No. of patients	Percentage
VH	1	0.5%
RD	-	-
Iris neovascularisation	-	-

Of the diabetic retinopathy patients one patient had vitreous haemorrage, is one of the sight threatening complication seen in initial presentation.

Comparison of glycosylated hemoglobin level in patients with and without DR

Age group	Levels of glycosylated hemoglobin in patients with DR	Levels of glycosylated hemoglobin in patients without DR
30-40 years	7.2-8.0%	4.4-6.2%
41-50 years	7.8-9.4%	4.1-6.2%
51-60 years	6.9-9.9%	5.3-6.7%

The HbA1C levels of the patients with DR are higher than those of the patients without retinopathy changes.

According to ADA HbA1C levels more than 6.8% is considered as poor glycaemic control.

So this indicates that patients might have had poor glycaemic control prior to the development of DR.

DISCUSSION

The aim of our study was to find the incidence of retinopathy changes, in newly detected DM type II. 200 eyes of 100 patients attending the ophthalmology outpatient department of the Stanley Medical College, Hospital, Chennai were studied. Our observation were compared with various other studies about diabetic retinopathy.

Various studies performed in India as found that there is an increased prevalence of DR in type II DM in the past 30 yrs

DR prevalence in newly diagnosed subjects in my study is16%

But it is comparatively lower than the prevalence data found in Europe study which is 20% to 35% [38,39]

The ratio of male to female patients in this study was 2.1:1 while it was 1.5:1 in the Wisconian epidemiological study [40]of DR 1984 and 1.8:1 in the Oman study[41] In our study DR is more common in men

.

Further studies such as UKPDS study[42], the Hyderabad study[43], and a study of Pima Indians[44] also have concluded that DR is common among males.

DR prevalence according to United Kingdom Prospective Diabetes study (UKPDS), at the time of initial diagnosis was 35% but the study subjects in UKPDS [42]study are older than study subjects in this study.

In our study 12% of patients had bilateral involvement, though the severity was asymmetrical in some patients.4% of patients had unilateral involvement.

Indirect ophthalmoscopy has been used as a standard technique in our study. The sensitivity and specificity of indirect ophthalmoscopy for detecting any retinopathy was 82% and 95% respectively. (Dandona et al)

The diabetes-associated changes in the trabeculum is related to decreased aqueous outflow. In the Wisconsin Epidemiologic study of

Diabetic Retinopathy, approximately 8% of people with diabetes in the older age group had intraocular pressures greater than 21 mmHg. But only 3% of nondiabetics in the older age group had IOP greater than 21 mmHg.[45]

There may be an effect of diabetes on the optic nerve. One can postulate that the optic nerve of people with diabetes may be more susceptible to destructive effects of intraocular pressure. This may exacerbate an increased risk of glaucoma that accompanies aging.[45]

Rema et al reported that 1.7-fold increase in risk of DR in subjects with 2% increase in HbA1C.

The normal range for the hemoglobin A1c is between 4% and 5.6%. Hemoglobin A1c levels between 5.7% and 6.4% indicate increased risk of diabetes, and levels of 6.5% or higher indicate diabetes. In our study patients of DR had HbA1C level more than 7%,thus indicates poor glycaemic control previously.[46]

The important risk factor for diabetes is the blood HbA1c levels. Wong et al reported DR risk increases 1-21-fold for every 1% increase of HbA1C. These observations were confirmed by our study and have showed that a raised blood HbA1c concentration was independently associated with an increased DR prevalence.

SUMMARY

- 200 eyes of 100 patients were studied during the period from July 2010 to November 2012
- The predominantly affected group were between 40 to 50 years of age(42.5%)
- The male:female ratio was 2.1:1
- A majority of patients had visual acuity between 6/9 and 6/18 on first presentation(43.5%)
- Of them 12% of patients had positive family history
- 33% of the patients were exposed to risk factors like smoking, alcohol and anaemia.
- 12% 0f the patients had bilateral diabetic retinopathy
- Of the 200 eyes evaluated 62 eyes had NPDR and 2 had PDR while 336 had no features of diabetic retinopathy.
- Among the NPDR majority (38 eyes) fell under moderate NPDR .

- Of the 100 patients 1 had VH at the time of persentation

- 3% of the patients had POAG during the initial presentation.

- Levels of glycosylated hemoglobin level (HbA1c) were more than 7mmol/l in patients with DR when compared to patients without DR. This indicates patients would have had unnoticed DM for a longer duration.

CONCLUSION

- ✓ Incidence of DR is common among middle aged working population.

- ✓ Both eyes were usually affected ,though asymmetrically.

- ✓ Detailed fundus examination and FFA in selected cases are important in diagnosing the type and severity of diabetic retinopathy.

- ✓ **In conclusion DR associated with vision threatening complications can be made out even at the time of diagnosis of diabetes mellitus type II.**

- ✓ **So regular ophthalmological evaluation is necessary to detect fundus changes in early stages in new cases of DM type-2**

- ✓ **Early diagnosis and treatment of DR helps to stabilize the visual acuity and prevent further loss.**

- ✓ **Diagnosis of complications of DR at the earliest helps to control progression of PDR.**

- ✓ **Increased awareness of the ophthalmological complication in diabetes is necessary in the society to prevent unnecessary visual morbidity in diabetic patients.**

ANNEXURES

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PROFORMA

INCIDENCE OF RETINOPATHY CHANGES IN PATIENTS WITH NEWLY DETECTED DIABETES MELLITUS

– a hospital based study

Hospital no. :

Name :

Age :

Sex :

Occupation :

Address :

Ocular complaints :

Diabetic status

Type :

Treatment details :

Family history :

Associated simultaneous

Systemic illness :

Associated Risk factors :

OCULAR EXAMINATION RE LE

Vision

v/n With pinhole

BCVA

Eyelids and lashes

Extraocular movements

Slit lamp examination

Conjunctiva

Cornea

Anterior chamber

Iris

Pupil

Lens

IOP

Visual field

Gonioscopy

Fundus

a.Direct ophthalmoscopy

1.media

2.disc

Colour

Shape

Size

Margins

Cup

3.vessels

arteries

Veins

4.Macula

Foveal reflex

Edema

1. Background of retina

b.Indirect ophthalmoscopy

Media

Disc

Macula

Periphery of retina

c.slit lamp examination+90D

INVESTIGATIONS

Bloodsugar

Fasting Post prandial

urine : Albumin

Sugar

Deposits

Blood hemoglobin

Glycosylated hemoglobin

Blood urea

Serum creatinine

KEY TO MASTER CHART

DM	-	Diabetes Mellitus
VA	-	Visual acuity
HM	-	Hand movements
CFCF	-	Counting fingers close to face
NPDR	-	Non-Proliferative diabetic retinopathy
PDR	-	Proliferative diabetic retinopathy
MILD NPDR	-	Mild Non-Proliferative diabetic retinopathy
Mod NPDR	-	Moderate Non-Proliferative diabetic retinopathy
Sev NPDR	-	Severe Non-Proliferative diabetic retinopathy
VS NPDR	-	Very Severe Non-Proliferative diabetic retinopathy
BCVA	-	Best corrected visual acuity
IOP	-	Intra ocular pressure
M	-	Male
F	-	Female
A	-	anemia
AL	-	Alcoholism
S	-	Smoking
FBS	-	Fasting Blood Sugar
PPBS	-	Post Prandial Blood Sugar
HBA1C	-	Glyco sylated Hb

RE	-	Right Eye
LE	-	Left Eye
PRP	-	Pan-retinal photocoagulation
I	-	Insulin
O	-	Oral hypoglycemic agents
Comp.	-	Complications
VH	-	Vitreous hemorrhage
RD	-	Retinal detachment
CRAO	-	Central retinal Artery occlusion
Hosp No.	-	Hospital Number

SNO	NAME	AGE	SEX	Hospital No.	V/A ON PRESENTATION		BCVA		FAMILY HISTROY	ASSOCIATED RISK FACTORS	BLOOD SUGAR		HBA1C %	IOP MMHg	TREATMENT OF DM	DIAGNOSIS		MACULOPATHY		COMPLICATIONS
					RE	LE	RE	LE			FBS	PPBS				RE	LE	RE	LE	
1	Rathinam	55	M	171986	6/18	6/12	6/9	6/9	-	S+AL+A	166	273	8.3	12	O	-	Mild NPDR	-	-	-
2	Sulochana	33	F	298764	6/12	6/6	6/6	6/6	-	-	179	232	4.5	14	O	-	-	-	-	-
3	Boopalan	41	M	193279	6/9	6/9	6/6	6/6	-	-	181	297	4.7	11	O	-	-	-	-	-
4	Ranganathan	44	M	397641	6/6	6/6	6/6	6/6	-	-	176	320	5.4	10	O	-	-	-	-	-
5	Soundappan	38	M	316348	6/12	6/6	6/6	6/6		S+AL	187	234	5.7	14	O	-	-	-	-	-
6	Ramesh	43	M	493817	6/24	6/12	6/12	6/12	+	S	182	271	5.2	16	O	-	-	-	-	-
7	Albert	43	M	293798	6/12	6/24	6/6	6/12	-	AL	176	274	5.3	17	O	-	-	-	-	-
8	Rani	45	F	1843217	6/6	6/6	6/6	6/6	-	-	157	257	4.6	15	O	-	-	-	-	-
9	Manickam	32	M	304678	6/6	6/6	6/6	6/6	-	-	156	280	4.7	12	O	-	-	-	-	-
10	Marimuttu	58	M	329781	6/36	6/60	6/24	6/12	-	S+AL+A	210	326	8.8	16	O	MOd NPDR	MOd NPDR	-	-	-
11	Baby	49	F	193671	6/9	6/18	6/6	6/6	-	-	186	271	5.7	18	O	-	-	-	-	-
12	Panneerselvam	31	M	274136	6/12	6/6	6/6	6/6	-	S+AL	173	312	6.2	13	O	-	-	-	-	-
13	Babu	47	M	381376	6/12	6/12	6/6	6/6	-	AL	186	321	5.6	12	O	-	-	-	-	-
14	Shanthi	51	F	281979	6/18	6/18	6/18	6/9		-	271	391	7.9	13	I+O	-	Mild NPDR	-	-	-
15	Periasamy	48	M	314625	6/24	6/36	6/6	6/12	+	S+AL+A	181	392	8.2	23	O	MOd NPDR	MOd NPDR	-	-	-
16	Angamuttu	46	M	1311271	6/6	6/6	6/6	6/6	-	S+AL	189	272	4.6	14	O	-	-	-	-	-
17	Harikrishnan	48	M	179648	6/9	6/6	6/6	6/6	-	S	168	211	5.7	12	O	-	-	-	-	-
18	Arumugam	35	M	134679	6/12	6/12	6/6	6/6	-	AL	176	267	5.7	17	O	-	-	-	-	-
19	Shanthi	33	F	117911	6/12	6/12	6/6	6/6	-	-	210	382	5.8	16	O	-	-	-	-	-
20	Kumar	53	M	139798	6/18	6/36	6/12	6/18	-	S+AL+A	182	293	8.5	24	O	MOd NPDR	MOd NPDR	-	-	-
21	Suresh	33	M	396178	6/18	6/6	6/6	6/6	-	-	192	317	5.3	13	O	-	-	-	-	-
22	Kumaresan	42	M	346819	6/6	6/12	6/6	6/6	-	-	182	265	4.9	12	O	-	-	-	-	-
23	Sundari	51	F	224221	6/9	6/6	6/6	6/6	-	-	210	352	4.3	10	O	-	-	-	-	-
24	Vasanthakumar	35	M	297618	6/6	6/9	6/6	6/6	-	-	171	351	5.9	12	O	-	-	-	-	-
25	Dhanakotti	43	M	391622	6/36	6/60	6/24	6/24	-	S+AL+A	187	324	9.3	28	O	Sev NPDR	MOd NPDR	-	-	-
26	Peter	51	M	317986	6/18	6/12	6/9	6/9	-	AL	171	259	6.3	16	O	-	-	-	-	-
27	Gomathy	45	F	334678	6/36	6/36	6/9	6/9	-	A	199	376	8.3	18	O	Mod NPDR	Mod NPDR	-	-	-
28	Mariammal	39	F	331789	6/36	6/24	6/12	6/9	-	A	232	397	9.1	12	O	Sev NPDR	MOd NPDR	-	-	-
29	Purusothaman	44	M	131146	6/12	6/6	6/6	6/6	-	-	176	267	6.2	13	O	-	-	-	-	-
30	Prabaharan	42	M	291744	6/6	6/6	6/6	6/6	-	S	186	245	5.6	12	O	-	-	-	-	-
31	Doulath	44	F	146891	6/6	6/6	6/6	6/6	-	-	171	286	4.5	15	O	-	-	-	-	-
32	Karthick	44	M	246894	6/12	6/12	6/6	6/6	-	-	186	290	5.8	12	O	-	-	-	-	-
33	Aruldurai	43	M	241316	6/12	6/6	6/6	6/6	-	-	162	311	4.6	14	O	-	-	-	-	-
34	Shanker	39	M	413746	6/18	6/12	6/6	6/6	-	-	177	287	4.8	14	O	-	-	-	-	-
35	Mani	45	M	241763	6/24	6/18	6/9	6/6	-	-	210	326	5.3	13	O	-	-	-	-	-
36	Hafesa	42	F	196913	6/24	6/24	6/12	6/12	-	-	186	271	4.6	13	O	-	-	-	-	-
37	Karthikeyan	39	M	148919	6/12	6/6	6/6	6/6	-	-	173	265	5.7	11	O	-	-	-	-	-
38	Settumani	51	F	393716	6/24	6/18	6/6	6/12	-	S+AL+A	186	345	5.7	16	O	-	Mild NPDR	-	-	-
39	Rathinam	35	M	137819	6/6	6/18	6/6	6/6	-	-	210	298	4.9	16	I+O	-	-	-	-	-
40	Paavendhan	46	M	234532	6/12	6/36	6/9	6/12	+	A	181	290	5,8	17	O	-	-	-	-	-
41	Ayesha	53	F	347619	6/6	6/6	6/6	6/6	-	-	189	272	5.3	16	O	-	-	-	-	-
42	Mokamed	43	M	413761	6/6	6/6	6/6	6/6	-	AL	168	321	5.7	16	O	-	-	-	-	-
43	Ambiga	34	F	341318	6/12	6/12	6/6	6/6	-	-	201	289	4.7	18	O	-	-	-	-	-
44	Duraisamy	51	M	343719	6/12	6/12	6/12	6/9	-	-	210	382	4.6	13	O	-	-	-	-	-
45	Raman	41	M	331719	6/12	6/18	6/6	6/12	+	S+AL+A	233	389	8.3	12	O	Mild NPDR	Mild NPDR	-	-	-
46	Paranjothi	42	M	346798	6/18	6/6	6/6	6/6	-	-	192	317	5.5	13	O	-	-	-	-	-
47	Rekha	54	F	194624	6/6	6/6	6/6	6/6	-	-	182	271	6.2	12	O	-	-	-	-	-
48	Rajamani	44	M	317323	6/9	6/6	6/6	6/6	-	-	210	352	5.7	12	O	-	-	-	-	-
49	Baskhar	46	M	197211	6/6	6/18	6/6	6/6	-	-	171	351	6.2	15	O	-	-	-	-	-
50	Vijayalakhsmi	32	F	193798	6/6	6/6	6/6	6/6	-	-	165	218	5.8	17	0	-	-	-	-	-

SNO	NAME	AGE	SEX	Hospital No.	V/A ON PRESENTATION		BCVA		FAMILY HISTROY	ASSOCIATED RISK FACTORS	BLOOD SUGAR		HB A1C %	IOP MMHg	TREATMENT OF DM	DIAGNOSIS		MACULOPATHY		COMPLICATIONS
					RE	LE	RE	LE			F	PP				RE	LE	RE	LE	
51	Vinoth	51	M	467198	6/6	6/6	6/6	6/6	-	S	198	273	6	12	O	-	-	-	-	-
52	Ammarng	31	M	314462	6/12	6/6	6/6	6/6	-	-	179	301	5.8	14	O	-	-	-	-	-
53	Paramanadhan	48	M	343627	6/8	6/9	6/6	6/6	-	-	181	252	4.8	13	O	-	-	-	-	-
54	Narajanan	51	M	481769	6/24	6/24	6/6	6/18	-	S+AL+A	176	320	9	11	O	MOD NPDR	MOD NPDR	-	-	-
55	Indhumathi	56	F	346178	6/12	6/12	6/6	6/6	-	-	176	298	5.2	18	O	-	-	-	-	-
56	Francis	37	M	481796	6/6	6/6	6/6	6/6	-	AL	182	271	5.8	16	O	-	-	-	-	-
57	Priya	41	F	461789	6/6	6/6	6/6	6/6	-		126	274	4.9	13	O	-	-	-	-	-
58	Amalya	43	F	294672	6/6	6/6	6/6	6/6	-	-	157	257	4.7	12	O	-	-	-	-	-
59	Jayakumar	32	M	413798	6/6	6/6	6/6	6/6	-	-	191	280	5.9	12	O	-	-	-	-	-
60	mohamed basha	54	M	331122	6/18	6/24	6/9	6/12	-	S+AL+A	210	326	7.3	17	O	MOd NPDR	Mild NPDR	-	-	-
61	Antony	41	M	346917	6/6	6/6	6/6	6/6	-	-	186	271	4.7	15	O	-	-	-	-	-
62	Gangadharan	43	M	176922	6/12	6/6	6/6	6/6	-	-	173	312	5.7	15	O	-	-	-	-	-
63	Kannan	34	M	176413	6/12	6/9	6/6	6/6	-	S	186	321	6.2	14	O	-	-	-	-	-
64	Sarala	59	F	343516	6/60	6/60	6/18	6/24	-	S+AL+A	271	391	8.1	12	I+O	Mild NPDR	MOD NPDR	-	-	-
65	Babu	45	M	313768	6/12	6/36	6/61	6/12	+	-	181	392	6	17	O	-	-	-	-	-
66	Joyal	47	M	345136	6/6	6/6	6/6	6/6	-	AL	189	272	6.2	17	O	-	-	-	-	-
67	Shanmugam	36	M	293176	6/9	6/6	6/9	6/6	-	S	176	321	5.2	15	O	-	-	-	-	-
68	Kamatchi	43	F	379198	6/12	6/12	6/6	6/6	-	-	209	327	5.9	24	O	-	-	-	-	-
69	Backiaraj	49	M	413748	6/12	6/12	6/6	6/6	-	-	179	256	5.7	12	O	-	-	-	-	-
70	Karuppusamy	37	M	413398	6/6	6/6	6/6	6/6	-	-	182	293	4.9	17	O	-	-	-	-	-
71	Karpagam	56	F	371642	6/18	6/12	6/12	6/6	-	-	192	317	4.2	14	O	-	-	-	-	-
72	Senthilkuar	42	M	241796	6/6	6/6	6/6	6/6	-	-	182	271	5.9	12	O	-	-	-	-	-
73	Santhosten	39	M	314464	6/9	6/6	6/6	6/6	-	-	210	352	4.8	12	O	-	-	-	-	-
74	Abirami	44	F	221318	6/6	6/24	6/6	6/6	-	-	171	351	4.5	17	O	-	-	-	-	-
75	Bharathi	42	M	413299	6/18	6/6	6/9	6/6	-	-	187	231	6.1	15	O	-	-	-	-	-
76	Velmurugan	42	M	416738	6/6	6/6	6/6	6/6	-	S	121	273	5.9	16	O	-	-	-	-	-
77	Duraisamy	32	M	393176	6/12	6/6	6/6	6/6	-	-	179	301	5.7	14	O	-	-	-	-	-
78	Ranganathan	43	M	391798	6/18	6/9	6/9	6/6	-	S+AL+A	181	252	.6.2	13	O	MOD NPDR	-	-	-	-
79	Shanmugam	54	M	221468	6/6	6/6	6/6	6/6	-	-	176	320	5.7	14	O	-	-	-	-	-
80	Chandrakumar	45	M	416978	6/12	6/12	6/6	6/6	-	S+AL	216	321	5.3	12	O	-	-	-	-	-
81	Roopa	47	F	3910608	6/24	6/6	6/12	6/6	-	-	182	271	4.6	15	O	-	-	-	-	-
82	Srinivasan	34	M	381368	6/9	6/6	6/6	6/6	-	S	126	274	5.7	14	O	-	-	-	-	-
83	Murali	53	M	241736	6/6	6/6	6/6	6/6	-	-	157	257	6.1	17	O	-	-	-	-	-
84	Muthukumar	48	M	468321	6/6	6/6	6/6	6/6	-	S+AL	191	280	5.8	15	O	-	-	-	-	-
85	Ruckmani	35	F	134591	6/18	6/6	6/9	6/6	-	-	210	326	4.9	12	O	-	-	-	-	-
86	Munusamy	52	M	241398	6/6	6/6	6/6	6/6	-	-	186	271	4.8	12	O	-	-	-	-	-
87	Ambaranath	31	M	460312	6/12	6/6	6/6	6/6	-	-	173	312	5.7	14	O	-	-	-	-	-
88	Annamal	48	F	221468	6/24	6/24	6/9	6/12	-	-	186	321	6.4	13	O	-	-	-	-	-
89	Ramu	42	M	440801	6/6	6/12	6/6	6/6	-	S+AL+A	271	391	8.3	16	I+O	-	Mild NPDR	-	-	-
90	Vasantha	39	F	135791	6/12	6/6	6/6	6/6		-	181	392	6.2	14	O	-	-	-	-	-
91	Narayan	52	M	217698	6/36	6/18	6/36	6/12	-	S	189	272	5.7	12	O	-	-	-	-	-
92	Ruckmani	32	F	327168	6/6	6/6	6/6	6/6	-	-	252	194	6.2	13	O	-	-	-	-	-
93	Selvarangam	51	M	281719	6/60	6/36	6/12	6/18	-	S+AL+A	281	401	7.4	16	O+I	MOD NPDR	SEV NPDR	-	-	-
94	Gonndan	45	M	394672	6/12	6/12	6/6	6/6	-	S+AL	210	382	4.9	12	O	-	-	-	-	-
95	Gajalanathan	34	M	161982	6/6	6/6	6/6	6/6	-	-	182	293	5.7	13	O	-	-	-	-	-
96	sathya	54	F	143172	6/18	6/6	6/6	6/6	-	-	192	317	4.3	17	O	-	-	-	-	-
97	Mathew	47	M	169321	6/6	6/12	6/6	6/6	-	-	182	271	5.4	13	O	-	-	-	-	-
98	Ilayaraja	54	M	297861	6/9	6/6	6/6	6/6	-	S	210	352	6.3	15	O	-	-	-	-	-
99	Shajagan	49	M	314678	6/6	6/18	6/6	6/6	-	-	171	351	5.8	16	O	-	-	-	-	-
100	Rangan	35	M	413268	6/6	6/6	6/6	6/6	-	-	176	235	5.5	15	O	-	-	-	-	-

SNO	NAME	AGE	SEX	Hospital No.	V/A ON PRESENTATION		BCVA		FAMILY HISTROY	ASSOCIATED RISK FACTORS	BLOOD SUGAR		HB A1C %	IOP MMHg	TREATMENT OF DM	DIAGNOSIS		MACULOPATHY		COMPLICATIONS
					RE	LE	RE	LE			F	PP				RE	LE	RE	LE	
101	Sugam	37	M	346897	6/6	6/6	6/6	6/6	-	AL	121	273	6.4	12	O	-	-	-	-	-
102	poomalai	46	M	194642	6/12	6/24	6/6	6/12	-	S+AL+A	179	301	8.2	17	O	MOD NPDR	MOD NPDR	-	-	-
103	Murugan	35	M	317233	6/8	6/9	6/6	6/6	-	-	181	252	6.1	11	O	-	-	-	-	-
104	muttu	38	M	197112	6/6	6/6	6/6	6/6	-	-	176	320	5.6	10	O	-	-	-	-	-
105	laksmi	45	F	467981	6/12	6/12	6/6	6/6	-	-	216	181	6	13	O	-	-	-	-	-
106	prabha	49	F	314462	6/24	6/6	6/12	6/6	-	-41066	182	271	5.7	16	O	-	-	-	-	-
107	kumar	51	M	313276	6/3	6/6	6/6	6/6	-	S	146	274	8	12	O	-	-	-	-	-
108	Kumari	34	F	481697	6/6	6/6	6/6	6/6	-	-	157	285	5	17	O	-	-	-	-	-
109	Sugavanam	38	M	461897	6/6	6/6	6/6	6/6	-	-	191	280	5.5	14	O	-	-	-	-	-
110	Arya	44	F	461894	6/18	6/6	6/9	6/6	-	A	210	326	4.7	12	O	-	-	-	-	-
111	Yoshwa	47	M	294726	6/6	6/6	6/6	6/6	-	-	186	271	5.4	12	O	-	-	-	-	-
112	Mohamed	36	M	413789	6/12	6/6	6/6	6/6	-	-	173	312	6.2	14	O	-	-	-	-	-
113	vasanthan	50	M	331212	6/12	6/12	6/12	6/9	-	S	186	321	5.7	12	O	-	-	-	-	-
114	Vimala	45	F	563248	6/9	6/12	6/6	6/6	-	-	271	391	8	15	I+O	Mild NPDR	-	-	-	-
115	Biman	54	M	179486	6/12	6/36	6/9	6/12	+	-	181	392	5	13	I+O	-	-	-	-	-
116	Samy	59	M	134196	6/24	6/18	6/12	6/12	-	S	176	272	7.3	12	O	Mild NPDR	MOD NPDR	-	-	-
117	Umadevi	60	F	119711	6/24	6/18	6/12	6/12	-	-	189	270	5.8	10	O	-	-	-	-	-
118	Banu	57	F	139981	6/12	6/12	6/6	6/6	-	S+AL+A	281	401	7.9	13	O+I	Mild NPDR	MOD NPDR	-	-	-
119	Nisha	43	F	396213	6/12	6/12	6/6	6/6	-	-	210	382	6.2	12	O	-	-	-	-	-
120	Sampath	51	M	224223	6/6	6/60	6/36	6/6	-	-	182	293	5.7	13	O	-	-	-	-	-
121	Kumaran	32	M	297867	6/18	6/6	6/6	6/6	-	-	192	317	6.2	11	O	-	-	-	-	-
122	Muskan	37	M	334891	6/6	6/12	6/6	6/6	-	-	182	271	5.9	13	O	-	-	-	-	-
123	sathyan	54	M	131156	6/9	6/6	6/6	6/6	-	-	210	352	5.3	13	O	-	-	-	-	-
124	Pavadairayan	53	M	291766	6/6	6/18	6/6	6/6	-	-	171	351	4.7	14	O	-	-	-	-	-
125	Eswar	39	M	171879	6/6	6/6	6/6	6/6	-	-	187	233	6.2	11	o	-	-	-	-	-
126	Muneeswari	44	F	298647	6/6	6/9	6/6	6/6	-	A	121	273	5.7	10	O	-	-	-	-	-
127	kulandai	48	M	193771	6/24	6/24	6/6	6/6	-	-	179	301	6.3	14	O	-	-	S	-	-
128	manonmani	55	F	397146	6/18	6/9	6/9	6/6	-	-	181	252	5.6	12	O	-	-	-	-	-
129	Thangaraj	32	M	316483	6/6	6/6	6/6	6/6	-	S	176	320	5.3	12	O	-	-	-	-	-
130	Samikkanu	32	M	493176	6/12	6/12	6/6	6/6	-	-	167	212	5.6	13	O	-	-	-	-	-
131	Thomas	34	M	293908	6/24	6/6	6/12	6/6	-	S	182	271	5.7	17	O	-	-	-	-	-
132	Md.ibraheem	37	M	184123	6/12	6/6	6/6	6/6	-	S+AL+A	189	274	7.1	13	O	Mild NPDR	-	-	-	-
133	Saraswathy	45	F	304781	6/6	6/6	6/6	6/6	-	-	157	257	6.1	12	O	-	-	-	-	-
134	Ayyavu	35	M	329678	6/6	6/6	6/6	6/6	-	-	191	280	5.6	10	O	-	-	-	-	-
135	Mari	39	M	274381	6/12	6/6	6/9	6/6	-	-	210	326	4.6	13	O	-	-	-	-	-
136	Eswari	54	F	381234	6/18	6/24	6/12	6/12	-	-	186	271	6.1	11	O	-	-	-	-	-
137	Ponnan	57	M	281999	6/60	6/60	6/36	6/36	-	S+AL+A	254	412	8.3	26	O+I	Sev NPDR	V SEV NPDR	-	-	-
138	Elango	43	M	314111	6/12	6/24	6/6	6/6	-	S	186	321	5.6	12	O	-	-	-	-	-
139	Prabaharan	44	M	416321	6/12	6/18	6/6	6/6	-	-	271	391	4.7	13	I+O	-	-	-	-	-
140	Kaliappan	51	M	393178	6/12	6/36	6/12	6/12	+	S+AL+A	181	392	7.3	14	O	MOD NPDR	MOD NPDR	-	-	-
141	Kalyani	57	F	416415	6/6	6/6	6/6	6/6	-	-	189	272	6.1	12	O	-	-	-	-	-
142	Antony	43	M	221546	6/9	6/6	6/6	6/6	-	S	252	354	5.8	11	O	-	-	-	-	-
143	Govindasamy	42	M	391768	6/12	6/12	6/6	6/6	-	-	281	401	6.2	10	O	-	-	-	-	-
144	Santhi	54	F	381441	6/12	6/12	6/6	6/6	-	-	210	382	5.7	12	O	-	-	-	-	-
145	Banu bagam	42	F	241778	6/6	6/6	6/6	6/6	-	A	182	293	6	11	O	-	-	-	-	-
146	Ismail	33	M	132663	6/18	6/6	6/6	6/6	-	-	192	317	6.2	13	O	-	-	-	-	-
147	Rose mary	45	F	460991	6/6	6/12	6/6	6/6	-	-	182	271	4.9	15	O	-	-	-	-	-
148	Mumtaz	55	F	135896	6/12	6/9	6/12	6/6	-	-	210	352	6	13	O	-	-	-	-	-
149	Saravanan	59	M	346231	6/36	6/18	6/6	6/6	-	-	171	351	5.3	16	O	-	-	-	-	-
150	Nandakumar	57	M	176090	6/24	6/18	6/6	6/9	-	S+AL+A	187	289	8.5	12	O	MOD NPDR	MOD NPDR	-	-	-

SNO	NAME	AGE	SEX	Hospital No.	V/A ON PRESENTATION		BCVA		FAMILY HISTROY	ASSOCIATED RISK FACTORS	BLOOD SUGAR		HB A1C %	IOP MMHg	TREATMENT OF DM	DIAGNOSIS		MACULOPATHY		COMPLICATIONS
					RE	LE	RE	LE			F	PP				RE	LE	RE	LE	
151	Perumal	55	M	413422	6/12	6/9	6/6	6/6	-	S	121	273	5.7	`4	O	-	-	-	-	-
152	Meenatchi	57	F	224401	6/36	6/36	6/18	6/12	-	S+AL+A	243	402	9	11	O+I	PDR	V SEV NPDR	-	VH	-
153	Athi	58	M	371980	6/8	6/9	6/6	6/6	-	-	181	252	5,1	13	O	-	-	-	-	-
154	Thambidurai	51	M	293376	6/12	6/18	6/9	6/12	-	S+AL+A	176	320	9.9	16	O	Mild NPDR	MOD NPDR	-	-	-
155	Appasamy	35	M	413370	6/12	6/12	6/6	6/6	#VALUE!	-	176	231	6.2	12	O	-	-	-	-	-
156	Narayanan	36	M	345056	6/24	6/24	6/12	6/6	+	S	182	271	4.7	12	O	-	-	-	-	-
157	Muttu naicker	33	M	314646	6/6	6/9	6/6	6/6	-	S	126	274	5.7	14	O	-	-	-	-	-
158	Asaithambi	30	M	217167	6/6	6/6	6/6	6/6	-	-	157	257	4.8	11	O	-	-	-	-	-
159	Chinnusamy	52	M	327897	6/36	6/24	6/6	6/6	-	-	191	280	5.2	12	O	-	-	-	-	-
160	Anguraj	45	M	169056	6/18	6/6	6/9	6/6	-	-	210	326	5	14	O	-	-	-	-	-
161	Brintha	43	F	413551	6/6	6/6	6/6	6/6	-	-	186	271	5.3	12	O	-	-	-	-	-
162	Kalaiarasan	49	M	314118	6/12	6/6	6/6	6/6	-	-	173	312	5.2	12	O	-	-	-	-	-
163	Lila	46	F	314736	6/12	6/18	6/6	6/6P	-	S	186	321	5	11	O	-	-	-	-	-
164	Devikarani	48	F	413363	6/6	6/24	6/6	6/24	-	A	271	391	8.6	15	I+O	Mild NPDR	Mod NPDR	-	-	-
165	Subramani	44	M	224890	6/12	6/36	6/18	6/12	+	S+AL+A	181	392	9	16	O+I	MOD NPDR	MOD NPDR	-	-	-
166	Siva	47	M	413098	6/9	6/6	6/6	6/6	-	S	189	272	5.7	12	O	-	-	-	-	-
167	Mohan	30	M	101546	6/9	6/6	6/6	6/6	-	S	176	243	6.2	15	O	-	-	-	-	-
168	Arumugam	36	M	297432	6/6	6/6	6/6	6/6	-	S	169	234	5.6	17	O	-	-	-	-	-
169	Kumari	57	F	281734	6/24	6/36	6/12	6/18	-	-	189	382	7.4	13	O	MOD NPDR	Mild NPDR	-	-	-
170	Kaleesswari	42	F	314110	6/6	6/6	6/6	6/6	-	-	182	293	6.6	15	O+I	-	-	-	-	-
171	Steepan	34	M	373764	6/18	6/6	6/6	6/6	-	-	187	244	5.8	16	O	-	-	-	-	-
172	Indu	35	F	478493	6/6	6/12	6/6	6/6	-	-	182	271	4.5	13	O	-	-	-	-	-
173	Anandan	41	M	263756	6/9	6/6	6/6	6/6	-	S+AL+A	210	352	7.9	13	O+I	MOD NPDR	MOD NPDR	-	-	-
174	Kaliammal	44	F	108765	6/6	6/18	6/6	6/6	-	-	171	351	5.P0	11	O	-	-	-	-	-
175	Ramanan	45	M	276548	6/18	6/12p	6/6	6/6	-	-	176	243	4.9	16	O	-	-	-	-	-
176	Pavalakodi	39	F	345743	6/9	6/12	6/6	6/6			121	273	5.7	15	O	-	-	-	-	-
177	Backiam	47	F	432598	6/24	6/12	6/6	6/6	-	-	179	301	8.3	12	O	Mild NPDR	Mild NPDR	-	-	-
178	Pappathy	49	F	112657	6/18	6/9	6/6	6/6	-	-	181	252	6.3	13	O	-	-	-	-	-
179	Nazeera	39	F	143268	6/6	6/6	6/6	6/6	-	-	176	241	5.9	13	O	-	-	-	-	-
180	vivekandan	37	M	232122	6/6	6/6	6/6	6/6	-	-	176	233	6.1	18	O	-	-	-	-	-
181	Moorthy	52	M	134543	6/24	6/6	6/12	6/6	+	S	211	398	7.4	14	O	MOD NPDR	PDR	-	-	-
182	Manikandan	36	M	432564	6/18	6/12	6/6	6/6	-	S+AL	126	274	5	13	O	-	-	-	-	-
183	Sankaran	39	M	234323	6/6	6/6	6/6	6/6	-	-	157	257	5.6	11	O	-	-	-	-	-
184	Rangan	42	M	345434	6/6	6/6	6/6	6/6	-	-	191	280	5.6	12	O	-	-	-	-	-
185	Ramu	31	M	111123	6/18	6/6	6/6	6/6	-	S+AL+A	210	326	8.5	15	O	Mild NPDR	Mild NPDR	-	-	-
186	Vaithiyalingam	42	M	234323	6/6	6/6	6/6	6/6	-	-	186	271	4	12	O	-	-	-	-	-
187	Madurai veeran	47	M	237654	6/12	6/9	6/6	6/6	-	-	173	293	4.4	17	O	-	-	-	-	-
188	Pushpa	36	F	345362	6/12	6/6	6/6	6/6	-	-	186	321	5.6	13	O	-	-	-	-	-
189	Radha	37	F	342568	6/6	6/12	6/6	6/6	-	A	271	391	7.9	13	I+O	Mild NPDR	MOD NPDR	-	-	-
190	Kannayeram	40	M	110023	6/24	6/36	6/12	6/12	+	S+AL+A	181	392	8.8	26	O+I	Sev NPDR	MOD NPDR	-	-	-
191	Rani	49	M	118790	6/60	6/24	6/6	6/6	-	S	189	272	5.5	11	O	-	-	-	-	-
192	Selvam	58	M	436271	6/9	6/6	6/6	6/6	-	S	252	194	4.7	10	O	-	-	-	-	-
193	Senthilkuar	57	M	364758	6/12	6/12	6/6	6/6	-	S	281	401	4.8	15	O	-	-	-	-	-
194	Ravi	60	M	234323	6/12	6/12	6/6	6/6	-	-	198	382	5.3	15	O	-	-	-	-	-
195	Kulandaisamy	42	M	112321	6/6	6/9	6/6	6/6	-	S+AL	182	293	6	17	O	-	-	-	-	-
196	Narayanasamy	45	M	342564	6/18	6/6	6/6	6/6	-	S+AL	192	317	5.2	12	O	-	-	-	-	-
197	Raman	53	M	543627	6/12	6/12	6/6	6/6	-	S+AL	187	271	5.3	15	O	-	-	-	-	-
198	Boopalan	35	M	234376	6/9	6/6	6/6	6/6	-	-	175	245	4.6	12	O	-	-	-	-	-
199	Arasappan	42	M	112232	6/6	6/18	6/6	6/6	-	S	171	245	5.6	10	O	-	-	-	-	-
200	Kalian	41	M	324563	6/12	6/12	6/9	6/12	-	S+AL+A	186	343	8.3	13	O	MOD NPDR	MOD NPDR	-	-	-