"LIPID PROFILE IN DIABETIC RETINOPATHY IN TYPE 2 DIABETIC MELLITUS"

By

Dr. AMITABH GUPTA

Dissertation submitted to the

B.L.D.E. UNIVERSITY,VIJAYAPUR, KARNATAKA

In partial fulfillment of the requirements for the degree of

MASTERS OF SURGERY

In

OPHTHALMOLOGY

Under the guidance of

Dr. SUNIL G.BIRADAR$_{M.S.}$

PROFESSOR

DEPARTMENT OF OPHTHALMOLOGY

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

& RESEARCH CENTRE, VIJAYAPUR,

KARNATAKA

2017

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

DECLARATION BY THE CANDIDATE

I, **Dr. AMITABH GUPTA,** hereby declare that this dissertationentitled **"LIPID PROFILE IN DIABETIC RETINOPATHY IN TYPE 2 DIABETIC MELLITUS"**is a bonafide andgenuine research work carried out by me under the guidance of**Dr.SUNIL G.BIRADAR**, Professor , Department of Ophthalmology,B.L.D.E.U's Shri B. M. Patil Medical College Hospital and ResearchCentre,Vijayapur.

Date: **Dr. AMITABH GUPTA**

Place: Vijayapur Post Graduate Student,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical

College, Hospital & Research Centre,

Vijayapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE,VIJAYAPUR

CERTIFICATE BY THE GUIDE

This to certify that the dissertation entitled **"LIPID PROFILE IN DIABETIC RETINOPATHY IN TYPE 2 DIABETIC MELLITUS"** is a bonafide research work done by **Dr. AMITABH GUPTA**, under my overall supervision and guidance, inpartial fulfillment of the requirements for the degree of M.S. inOphthalmology.

Date: **Dr.SUNIL G.BIRADAR**M.S.

Place: Vijayapur Professor ,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College,

Hospital & Research Centre,Vijayapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR

ENDORSEMENT BY THE HEAD OF DEPARTMENT

This to certify that the dissertation entitled **"LIPID PROFILE IN DIABETIC RETINOPATHY IN TYPE 2 DIABETIC MELLITUS"** is a bonafide research work done by **Dr. AMITABH GUPTA.** under the guidance of **Dr.SUNIL G.BIRADAR .**M.S., Professor , Department of Ophthalmology at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre,Vijayapur.

Date: **Dr. VALLABHA.K** M.S, DOMS.

Place: VijayapurProfessor & Head,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College,

ospital& Research Centre, Vijayapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR

ENDORSEMENT BY THE PRINCIPAL

This to certify that the dissertation entitled **"LIPID PROFILE IN DIABETIC RETINOPATHY IN TYPE 2 DIABETIC MELLITUS"** is a bonafide research work done by **Dr. AMITABH GUPTA** under the guidance of **Dr. SUNIL G.BIRADAR**M.S. Professor , Department of Ophthalmology at B.L.D.E.U's,Shri B. M. Patil Medical College Hospital and Research Centre,Vijayapur.

Date**:** **DR. S.P. Guggarigoudar**M.S.

Place:Vijayapur. Principal,

B.L.D.E.U's

Shri B. M. Patil Medical College,

Hospital & Research Centre,Vijayapur

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the B.L.D.E. UNIVERSITY, VIJAYAPUR,Karnataka shall have the rights to preserve, use and disseminate thisdissertation/thesis in print or electronic format for academic/research purposes.

Date: **Dr. AMITABH GUPTA**

Place: Vijayapur Post Graduate Student,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College,

Hospital & Research Centre,Vijayapur.

ACKNOWLEDGEMENT

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

I express my profound gratitude and sincere thanks to my guide, ***Dr. Sunil. G. Biradar****M.S.,Professor , Department of Ophthalmology, B.L.D.E.U's Shri B. M. PatilMedical College,Vijayapur , for his constant and unfailing support, professional insight,valuable suggestions, motivation and exemplary guidance to carry out and complete thisdissertation. I am deeply grateful to him for providing me necessary facilities and excellentsupervision to complete this work.*

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

My sincere thanks to our Medical Superintendent ***Dr. VIJAYKUMAR*** *for his support and inspiration.*

*I am deeply indebted and grateful to my professors****Dr. Vallabha K.*** ***M.S., DOMS.,****Professor & Head, and* ***Dr. M. H. Patil****M.S.,* *Department of Ophthalmology, B.L.D.E.U's Shri B. M. PatilMedical College,Vijayapur , who with their valuable suggestions and constant guidancesupported me throughout the preparation of this dissertation work.*

*My thanks to****Dr. Raghavendra.Ijeri****M.S.,* *Associate Professorr,****Dr. Jyoti.R.C,*** *Department of Ophthalmology, B.L.D.E.U'sShri B. M. Patil Medical College,Vijayapur , for their valuable suggestions and encouragementwhich have definitely helped me improve my research work.*

*I acknowledge my gratitude to****, Dr. Vijay and Dr. Priyanka,*** *Postgraduate colleagues, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College,Vijayapur, for his support, advice and help in data collection.*

I also thank all my seniors ***Dr.Shrawan****,****Dr. Shilpa,Dr.Darshan****,****Dr.Gautam, DrHarsha and Dr. Neeta,*** *my juniors* ***Dr.Vinit, Dr. Dishita and Dr. Prashant****for theirco-operation during the preparation of this dissertation.*

I thank ***Mr.Mohd.Shannawaz****, Statistician for his masterly guidance and statisticalanalysis. I sincerely acknowledge the support and kindness shown towards me*

by all the staffof Central Library, Shri B. M. Patil Medical College,Vijayapur , at all times.

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

It is with great pleasure I express my sincere gratitude to ***Dr .Tapeshwar Prasad****and****Smt. Sushila Gupta****for their constantencouragement, inspiration and sacrifices.*

*My heartly thanks to my wife****Dr. Rashmi.Gupta****, for her encouragement, support and sacrifices .*

I am grateful to Preeti internet cafe,Vijayapur for their timely and fantastic printing work.

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

Date- ***Dr. AMITABH GUPTA***

Place :Vijayapur

LIST OF ABBREVIATIONS

CSME	-	Clinicallly significant macular edema
CWS	-	Cotton Wool spot
DM	-	Diabetes Mellitus
DR	-	Diabetic Retinopathy
DME	-	Diabetic Macular Edema
NPDR	-	Non proliferative Diabetic Retinopathy
DRS	-	Diabetic Retinopathy Study
ETDRS	-	Early Treatment Diabetic Retinopathy Study
H/Ma	-	Hemorrhage/microaneurysm
IOP	-	Intra ocular pressure
IRMA	-	Intra retinal microvascularabnormalities
IDDM	-	Insulin dependent diabetes mellitus
NIDDM	-	Non insulin dependent diabetes mellitus
NPDR	-	Non proliferative diabetic retinopathy
NVD	-	New Vessels on disc
NVE	-	New vessels elsewhere in the retina.
OCT	-	Optical Coherence Tomography
PDR	-	Proliferative diabetic retinopathy
PRP	-	Pan Retinal Photocoagulation
RD	-	Retinal detachment
VEGF	-	Vascular Endothelial Growth Factor
WESDR	-	Wisconsin Epidemiologic Study of Diabetic Retinopathy

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

ABSTRACT

Background and Objective:

Diabetic retinopathy (DR) remains a leading cause of visual disability and blindness. It is a major microvascular complication of diabetes and is frequently accompanied by lipid exudation. Dyslipidemia leads to development of hard exudates and Clinically Significant Macular Edema (CSME). These, in turn, interfere with vision. The elevated lipid levels are associated with endothelial dysfunction which appears to play an important role in the pathogenesis of Diabetic Retinopathy, particularly in relation to breakdown of blood-retinal barrier. The current study was undertaken to determine the association of serum lipid profile with diabetic retinopathy and its severity. In the present study, 164 patients having type II diabetes mellitus of age group ranging from 30 to 80 years were studied. The patients were categorized with respect to the presence or absence of diabetic retinopathy. In the group having retinopathy, patients were subcategorized depending on the severity / grade of retinopathy and presence or absence of CSME. All the three groups had a near equal sex distribution with only a slight male predominance. A significant correlation was found between the patient age and diabetic retinopathy. In the present study, the duration since diagnosis of diabetes (diabetic age) ranged from 5 - 25 years. It is found that patients with retinopathy significantly had a longer mean duration of diabetes as compared to those diabetics without retinopathy. Most of the diabetics included in the study had poor glycemic control suggested raised FBS and PPBS levels.

The present study showed statistically significant correlation between diabetic retinopathy and raised total cholesterol level . Hypercholesterolemia was significantly

associated with the occurrence of diabetic retinopathy and CSME. Mean serum total cholesterol concentrations were higher in subjects with severe NPDR, very severe NPDR and PDR as compared with subjects without DR. No correlation was found between diabetic retinopathy and visual acuity. Thus, this study reinforces the observation that there is a strong association between dyslipidemia and diabetic retinopathy including CSME.

AIM AND OBJECTIVE OF THE STUDY:

To study the relationship between diabetic retinopathy in type 2 diabetic mellitus and serum lipid levels.

METHODS:

All type 2 diabetic mellitus patients with diabetic retinopathy attending outpatient department of Ophthalmology at B.L.D.E. University's ShriB.M.Patil Medical College Hospital and Research Centre, Bijapur Karnataka from December 2014 - May 2016 fulfilling the inclusion and exclusion criteria.

RESULTS:

Diabetic retinopathy and its severity was dependent on the control of diabetes and dyslipidemia among the patients. Dyslipidemia played a role in the pathogenesis of diabetic retinopathy. The total cholesterol level of patients with different diabetic retinopathies were high($p=0.001$). The LDL value were also significantly high in subjects($p=<0.001$). triglyceride level showed a rise with the severity of diabetic retinopathy($p=0.004$).Diabetes and dyslipidemia predispose to the pathogenesis and progression of diabetic retinopathy.

INTERPRETATION AND CONCLUSION:

There was significant correlation between total cholesterol, triglyceride, and low density lipoprotein , and severity of diabetic retinopathy.

Key Words : Diabetes Mellitus, Diabetic Retinopathy, Lipid Profile.

INTRODUCTION

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defect in insulin secretion, insulin action or both. It is a single most important disease which can affect nearly every organ system of the body.[1]

Diabetes is the commonest metabolic abnormality in the humans.[2] Type 2 diabetes is the commonest form of diabetes constituting nearly 90% of the diabetic population.[3] India with the largest number of diabetic subjects earned the title Diabetes capital of the world.

Diabetic retinopathy is a very common, potentially preventable, long-term, microvascular complication of Diabetes Mellitus and a leading cause of visual disability and blindness.[4]It is considered the hallmark of generalized microangiopathy occurring in a diabetic patient. In India the prevalence of diabetic retinopathy in general population is 3.5%, and the prevalence of diabetic retinopathy in the population with diabetes mellitus was 18.0%. In a population-based study in South India, diabetic retinopathy was detected in 1.78% of the diabetic patients screened.[5-6]

While there are multiple risk factors which have been associated with the development and progression of diabetic retinopathy, the duration of the disease and the age of the patient are said to be the strongest predictors. Other risk factors like hypertension, pregnancy, blood glucose level control and presence of nephropathy are shown to have a strong association. Dyslipidemia, microalbuminuria, BMI and smoking are some of the factors whose role as predictors of diabetic retinopathy is not well established.[7-9]

Diabetic retinopathy is frequently accompanied by lipid exudation.[10]Elevated serum lipid levels are associated with an increased risk of retinal hard exudate in persons with diabetic retinopathy. Although retinal hard exudate usually accompanies diabetic macular edema, increasing amounts of exudate appear to be independently associated with an increased risk of visual impairment.[11]The elevated lipid levels are also associated with endothelial dysfunction, which appears to play an important role in the pathogenesis of diabetic retinopathy, particularly in relation to the breakdown of blood-retinal barrier.

The association between serum lipid levels and diabetic retinopathy has been investigated in few studies. Some studies show a positive relationship between serum cholesterol and low-density lipoprotein levels and retinal hard exudation. Other studies show serum triglyceride levels as being important in the progression of retinopathy. Certain other studies show no relationship between serum lipid levels and diabetic retinopathy.[12]

The current study was undertaken to determine the association of serum lipid profile with diabetic retinopathy and its severity. The conflicting reports in the literature regarding the association between serum lipid levels and diabetic retinopathy and the paucity of studies relative to the existing case load warranted this study.

AIM AND OBJECTIVES

To study the relationship between diabetic retinopathy in type 2 diabetic mellitus and serum lipid levels.

REVIEW OF LITERATURE

- Cetin EN. et al. studied the association of serum lipid levels with diabetic retinopathy. They found that no significant association between serum lipids and the severity ofdiabetic retinopathy.[13]

- According to Report 22 of Early Treatment Diabetic Retinopathy Study (ETDRS) the serum lipid profile measured in the 2709 patients enrolled showed that patients with elevated total serum cholesterol levels or serum low-density lipoprotein cholesterol levels at baseline were twice as likely to have retinal hard exudates as patients with normal levels. These patients were also at higher risk of developing hard exudate during the course of the study. The risk of losing visual acuity was associated with the extent of hard exudate even after adjusting for the extent of macular edema.[14]

- Gupta et al. studied 350 subjects with type 2 diabetes who were randomly selected for the study during the period of January 2003 to December 2003. The eye examination was scheduled in the first visit protocol, along with other screening of macro and microangiopathy. Diabetic retinopathy was detected in 119/350 cases [34%]. Diabetics with raised LDL-C (>130mg%) showed higher prevalence of Diabetic retinopathy (38%) compared to others.(28.3%) (p=0.05).[15]

- Al-Bdour et al. studied risk factors associated with diabetic retinopathy among diabetic patients. A total of 986 patients with diabetes mellitus were assessed with detailed relevant history, complete medical and ophthalmic evaluation. Out of the 1961 eyes examined, 64.1 percent had one form of diabetic retinopathy; 54.8 percent had nonproliferative diabetic retinopathy (NPDR), 9.3 percent had proliferative diabetic retinopathy (PDR) and 30.8 percent had maculopathy.The study showed a positive relation between diabetic retinopathy and hypercholesterolemia in which it was significantly associated with the development of maculopathy (p=0.04), but not NPDR(0.192) or PDR(0.364).[16]

- Ucgun et al. studied the lipid profile in 54 patients with NPDR, 27 patients with exudative diabetic macular edema and 27 patients without exudative diabetic

macular edema and found that total serum cholesterol and LDL levels were elevated in patients with macular edema and high hard exudates. Triglyceride, HDL, VLDL levels were not different between the two groups.[17]

- Sharma P K. et al. studied the lipid profile in diabetics with and without retinopathy. They found that Diabetics showed significantly raised levels of total and LDL cholesterol, (257.0±68.88 mg% 173.5±67.7mg% respectively) compared to controls matched with age, sex and socioeconomic status. No significant difference was observed in these parameters in diabetics with and without retinopathy.[18]

- Association of elevated serum lipids with increased risk of progression to high risk Proliferative Diabetic Retinopathy(PDR) as well as their association with increased hard exudates and decreased visual acuity should provide additional motivation for lowering the frequently elevated lipid levels of diabetic patients.[19]

- In a study done by Iiechie AA.et al. no significant association between serum lipids with diabetic retinopathy in type 2 diabetic mellitus patients.[20]

DIABETES MELLITUS

Diabetes mellitus (DM) refers to a group of common metabolic disorders thatshare the phenotype of hyperglycemia.

It is defined as a group of metabolic diseases that are characterized byhyperglycemia resulting from defects in insulin secretion, insulin action or both.

Diabetes may present with symptoms suggestive of hyperglycemia such aspolyuria, polydipsia, polyphagia, and weight loss or may present with one of the acute or chronic complications or may be detected incidentally in hospitalized patients.

Diabetes may be seen intermittently only as in pregnancy.

CLASSIFICATION[21]

Diabetes mellitus is the most prevalent metabolic and non-communicable disorderin the world. There has been a lack of uniformity in manifestation, complications,management and genetics. This had led to epidemiological agencies to put forthvarieties of classifications for this syndrome of chronic hyperglycemia.

I.Type 1 diabetes (beta cell destruction, usually leading to absolute insulindeficiency)

a) Immune mediated

b) Idiopathic

II. Type 2 diabetes (may range predominantly insulin resistance with relativeinsulin deficiency to a predominantly insulin secretory defect with insulinresistance)

III. Other specific types of diabetes

A. Genetic defects of beta cell function characterized by mutations in :

1. Hepatocyte nuclear transcription factor (HNF)4alpha (MODY 1)

2. Glucokinase (MODY 2)

3. HNF-1 alpha (MODY 3)

4. Insulin promoter factor-1(IPF-1: MODY 4)

5. HNF-1beta (MODY 5)

6. Neuro D1 (MODY 6)

7. Mitochondrial DNA

8. Subunits of ATP sensitive potassium channel

9. Proinsulin or insulin

B. Genetic defects in Insulin action

1. Type A insulin resistance

2. Leprechaunism

3. Rabson-Mendenall syndrome

4. Lipodystrophy syndromes

C. Diseases of the exocrine pancreas- pancreatitis, pancreatectomy, neoplasia,cystic fibrosis, haemochromatosis, fibrocalculouspancreatopathymutations in carboxyl ester lipase.

D.Endocrinopathies – acromegaly, cushing's syndrome, glucagonoma,pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma.

E. Drug or chemical induced- glucocorticoids, pentamidine, nicotinic acid,Beta adrenergic agonists, thiazides, alpha interferons, protease inhibitors,anti psychotics (Atypicals and others)

F. Infections- congenital rubella, cytomegalovirus, coxsackie virus

G. Uncommon forms of immune mediated diabetes – "Stiff person"syndrome, anti-insulin receptor antibodies.

H. Other genetic syndromes sometimes associated with diabetes- Wolfram's

syndrome, Down's syndrome, Klinefelter's syndrome, Turner's syndrome,Friedreich's ataxia, Huntington's chorea, Laurence –Moon Biedlsyndrome, mytonic dystrophy, PraderWilli syndrome

IV. Gestational diabetes Mellitus (GDM)

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS:

The International Expert Committee with members appointed by the AmericanDiabetes Association (ADA), the European Association for the study of Diabetes andthe International Diabetes Federation has issued diagnostic criteria for Diabetesmellitus based on the following premises:

1) The Fasting Plasma Glucose, the response to an oral glucose challenge(OGTT-oral glucose tolerance test) and HbA1c differ among individuals and
2) Diabetes mellitus is defined as the level of glycemia at which diabetes

   specificcomplications occur rather than on deviations from a population based mean.

Criteria for the diagnosis of Diabetes Mellitus:[21]

1) A random blood glucose concentration> 200mg/dl (11.1 mmol/l)accompanied by classic symptoms of diabetes mellitus (polyuria, polydipsia,weight loss). **(**Random is defined as without regard to time since last meal.)

OR

2) Fasting plasma glucose (FPG) > 126 mg/dl (7.0 mmol/l) (Fasting is defined asno caloric intake for at least 8 hours).

OR

3) HbA1C > 6.5%

OR

4) Two-hour plasma glucose > 200mg/dl (11.1mmol/l) during an oral glucosetolerance test. (The test should be performed using a glucose load containingthe equivalent of 75gm anhydrous glucose dissolved in water, notrecommended in routine use.)

EPIDEMIOLOGY

The worldwide prevalence of diabetes mellitus has risen dramatically over thepast two decades, from an estimated 30 million cases in 1985 to 285 million in 2010.Based on current trend, the International Diabetes Federation projects that 438 millionindividuals will have diabetes by the year 2030.[22]

Although the prevalence of both type 1 and type 2 diabetes mellitus is

increasing worldwide, the prevalence of type 2 diabetes mellitus is rising much morerapidly because of increasing obesity, reduced activity levels as countries becomemore industrialized and the aging of population.

Type 2 diabetes is the predominant form of diabetes worldwide, accountingfor 90% of cases globally. An epidemic of Type 2 diabetes mellitus is underway inboth developed and developing countries, although brunt of the disorder is feltdisproportionately in Non-European populations. This is primarily due to rapidtransition occurring in these countries as a consequence of urbanization,industrialization and globalization.

The epidemiology of diabetes in India, the second largest country with apopulation of over 1 billion is of prime importance as the prevalence of diabetes isgrowing rapidly not only in urban area but also in rural areas.

In 2000, India (31.7 million) topped the world with the highest number ofpeople with diabetes mellitus followed by China (20.8 million) with the United States(17.7 million) in second and third place respectively.

According to the recent WHO report, India has the highest number of peoplewith diabetes in the world, with an estimated 32 million in 2000 which is set toincrease to a staggering 80 million by 2030.

DIABETIC RETINOPATHY

EPIDEMIOLOGY

Diabetic retinopathy is among the most common ophthalmic complication ofdiabetes mellitus.[23] Studies have reported that the prevalence of diabetic retinopathy inIndia varies from 20-31%. The estimates of the prevalence of diabetic retinopathy inIndia suggests that there may be nearly 5.6 million people with diabetic retinopathy inIndia .

Pathogenesis of DR

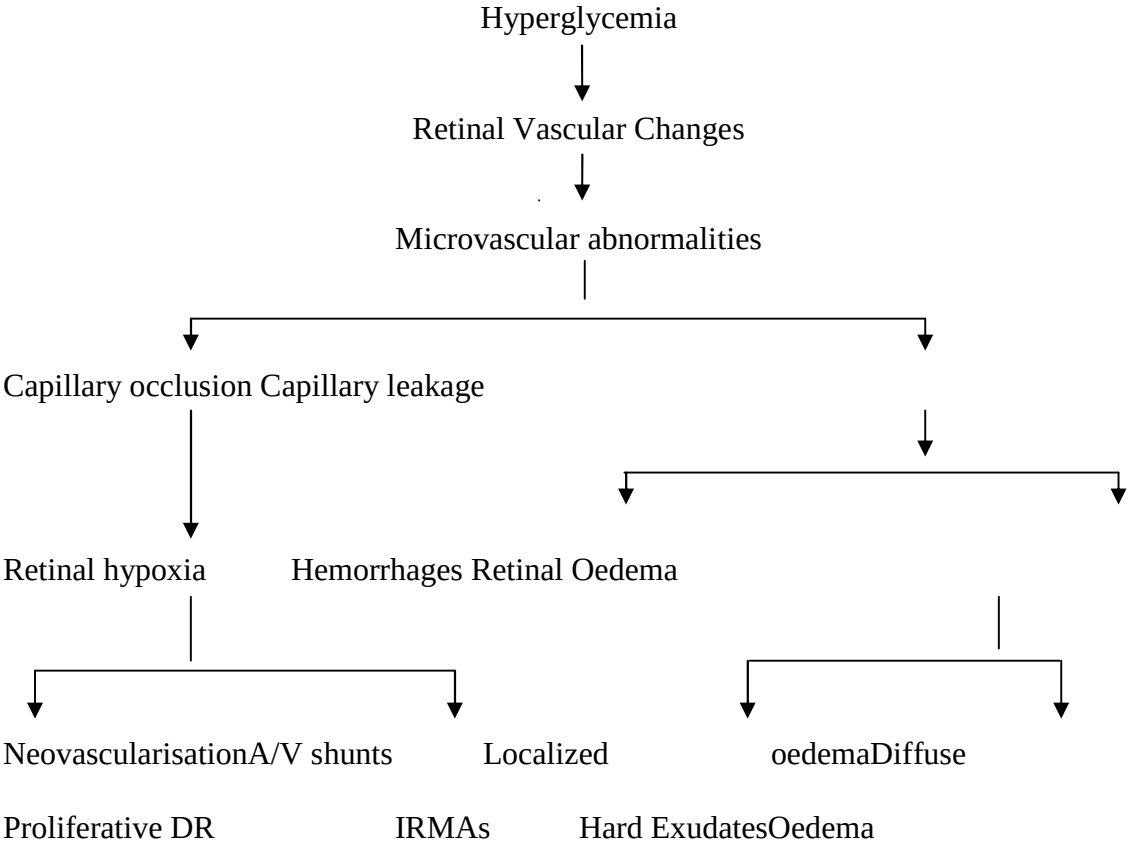


Table No. 1: Pathogenesis of Diabetic Retinopathy

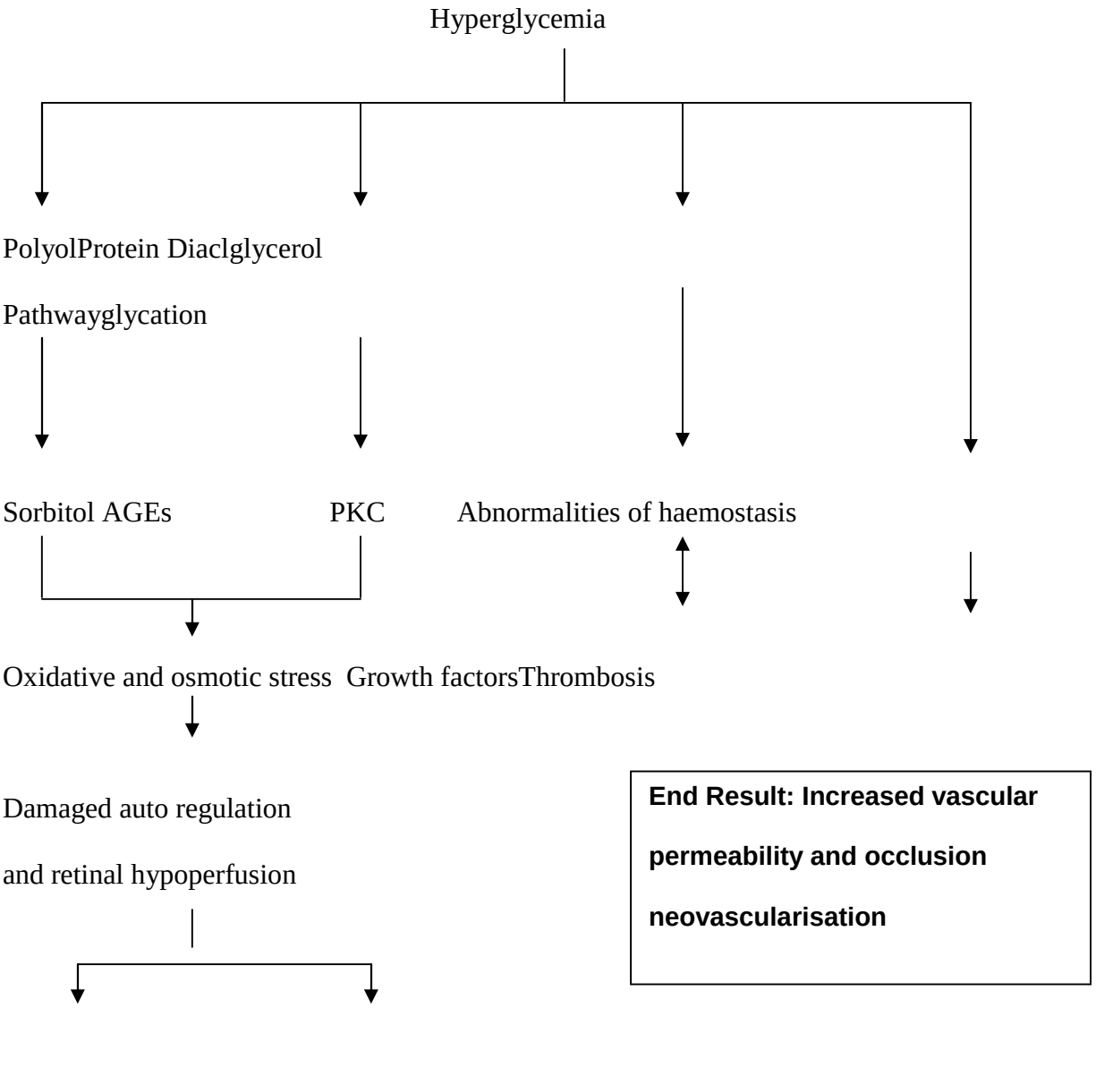


Table No. 2 : Effect of Hyperglycemia on pathogenesis of DR

CLASSIFICATIONS OF RETINOPATHY:

Many classifications have been proposed for diabetic retinopathy. Some of these are:

I. Hirschberg's Classification :

In 1980 Hirschberg [24]recognised three following types

1. Inflammatory – characterised by spots and haemorrhages

2. Haemorrhagic

3. Pigmentary

II. Ballantyne and Michaelson's Classification:

Ballantyne and Michaelson[25]divided diabetic retinopathy into 5 stages

1. Microlesions –comprised of microaneurysms

2. Macrolesions –dot and blot haemorrhages

3. Vascular changes-Gross changes in veins

4. Destructive changes-intraocular haemorrhages, retinitis proliferans, RD,Glaucoma

5. Mixed forms- diabetic changes associated with arteriosclerosis and /or hypertension.

III. Scott's Classification :[26]

Stage 1a - capillary microaneurysms

Stage 1b - phlebosclerosis; loops; coils; or knots in distended veins

Stage 11a - punctuate haemorrhages with or without discrete flecks of exudates

Stage 11b - larger round or blot haemorrhages, with confluent exudates

Stage 111a - more numerous haemorrhages and exudates

Stage 111b - haemorrhage into the vitreous

Stage 1V - Retinitis proliferans, retinal detachment, gross degenerative

changes.

IV. Alaert's and Slosse'sClassification :

They suggested a classification [27] which allows the fact that in some cases there ispreponderance of vascular whereas in others exudative lesions are seen, although in mostcases they occur side by side.

V. Lee's Classification :[28]

1. Angiopathy type-venous dilation; microaneurysms, haemorrhage&

neovascularisation

2. Exudative type

3. Proliferative retinopathy

4. Vitreous haemorrhage

VI. Duke Elder Classification :[24]

1. Pre retinopathic stage- characterised by uniform distension and turgescenceofVeins
2. Simple diabetic retinopathy- characterised by microaneurysms, exudates,haemorrhage and venous changes.
3. Proliferative diabetic retinopathy- formation of new vessels in the retina andproliferation into the vitreous

VII. Peyman'sClassification :[29]

1. Non proliferative/simple diabetic retinopathy- characterisedby microaneurysms,dot and blot haemorrhages ,exudates.

2. Proliferative/malignanant diabetic retinopathy-characterised by new vessel andglial tissue proliferation.

VIII. Clinical classification of diabetic retinopathy-Kanski :[30]

1. Background diabetic retinopathy
2. Diabetic maculopathy- focal; diffuse; ischaemic
3. Pre proliferative diabetic retinopathy
4. Proliferative diabetic retinopathy
5. Advanced diabetic eye disease

- persistent vitreous haemorrhage
- retinal detachment
- opaque membrane formation
- neovascular glaucoma

IX. Modern ETDRS :

The ETDRS in the 1980s developed a staging system which is recognisedas the gold standard for clinical trials. The ETDRS classification of DR[31]is based on the grading of fundus photography of seven stereoscopic fields and the severity of the retinopathy is determined after assessment of these photographs. Each ETDRS grade can predict the prognosis for developing sight-threatening retinopathy. In particular IRMA and venous beading have been identified as high risk for the development of PDR, and cotton wool spots are low risk. The ETDRS grading scheme is too complex to be easily used in clinical practice. Therefore an adaptation of the ETDRS classification is now widely

used. This classification system translates features identified by ETDRS as high and low risk into clinical practice. The level of retinopathy represents the risk of developing sight threatening DR and is used to plan follow-up and treatment.

Levels of Retinopathy:

NPDR

A. Mild NPDR

At least one microaneurysmFig. No. 1

Definition not met for B, C, D, E, F

Fig No. 1 showing Mild NPDR

B. Moderate NPDR

≥ Standard photograph Fig. No. 2

SE (CWS), VB, and IRMAs definitely present

Definition not met for C, D, E and F

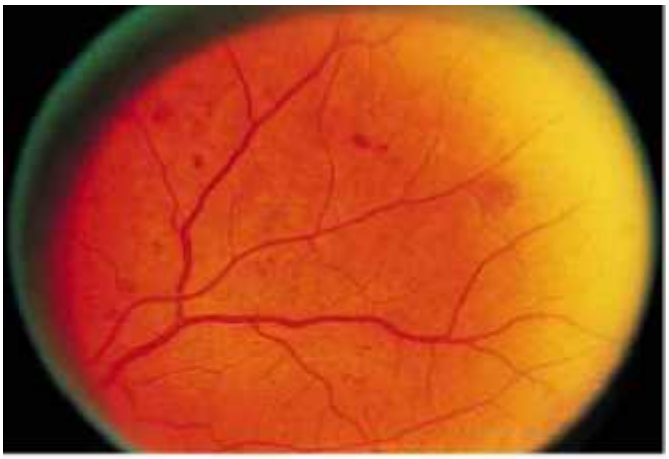


Fig No. 2 showing Moderate NPDR

C. Severe NPDR

H/Ma ≥ standard photograph Fig. No. 2 in all four quadrants

VB in two or more quadrants (Fig. No. 3)

IRMA ≥ standard photograph (Fig No. 4) in at least one quadrant

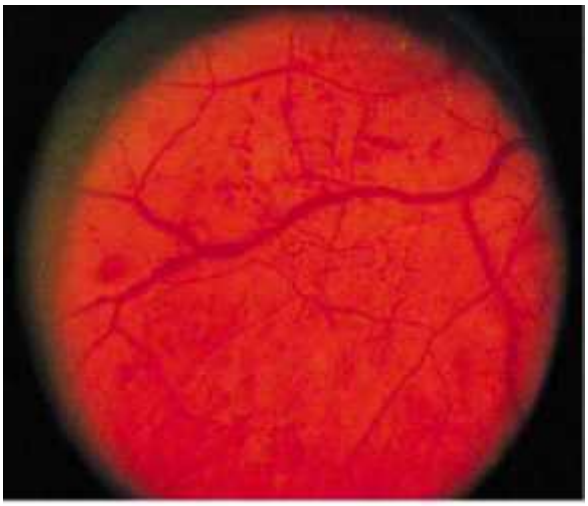


Fig No. 3: Severe NPDR

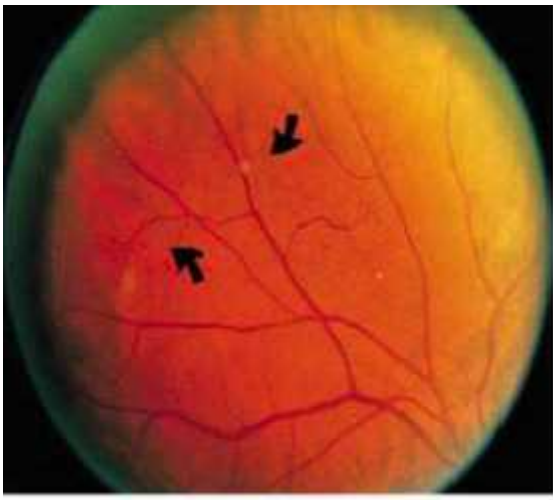


Fig No .4 : VB in > 2Quadrants

D. Very Severe NPDR

Any two or more of C(Fig. No. 5)

Definition not met for E, F.

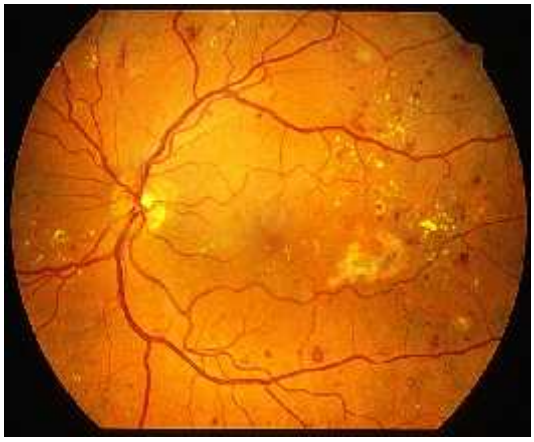


Fig No. 5 showing Very SevereNPDR

PDR

(Composed of: [1] NVD or NVE, [2] preretinal or vitreous haemorrhage,

[3]fibrous tissue proliferation)

E. Early PDR- New vessels

Definition not met for F

F. High-risk PDR

NVD ≥1/3-1/2 disc area **(**Fig. No. 6) or

NVD and vitreous or preretinalhaemorrhage or

NVE ≥1/2 disc area and preretinal or vitreous haemorrhage

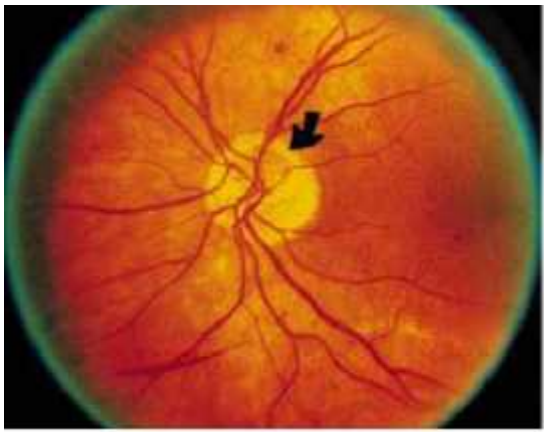


Fig No. 6: High risk PDR

X. International Clinical Diabetic Retinopathy Disease Severity Scale:[32]

Disease Severity Level	Clinical features
No apparent Retinopathy	No abnormalities
Mild NPDR	Microaneurysm only
Moderate NPDR	More than just microaneurysms, but less than severe NPDR
Severe NPDR	Any of the following: >20 intra-retinal hemorrhages in each 4 quadrants definite venous beading in > 2 quadrants and no signs of PDR
Very severe NPDR	All of the following: >20 intra-retinal hemorrhages in each of 4 Quadrants definite venous beading in > 2 quadrants prominent IRMA in > 1 quadrant
PDR	One or more of the following: neovasularisation vitreous/pre-retinal hemorrhage

Table No. 3: International Clinical Diabetic Retinopathy Disease Severity Scale

CSME :

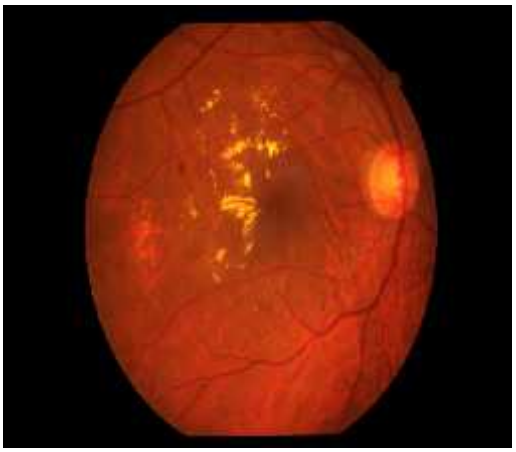


Fig No. 7: Showing CSME

1. Thickening of the retina located ≤500 μm from the centre of the macula (Fig. No. 7).or
2. Hard exudate with thickening of the adjacent retina located ≤500 μm from thecentre of the macula or
3. A zone of retinal thickening, 1 disc area or larger in size located ≤1 disc diameter from the centre of the macula

Diabetic macular oedema: divided into two broad levels:

Macular oedema apparently absent- no apparent retinal thickening or hard exudates in the posterior pole and Macular oedema apparently present- some retinal thickening or hard exudates in the posterior pole.

The below table shows the classification system for retinopathy and maculopathy; adapted from Scottish DRS grading scheme.

Retinopathy	**Description**
RO	No DR
R1 mild	Background DR The presence of > 1 of the following: dot hemorrhages microaneurysms hard exudates cotton wool spots superficial/flame shaped hemorrhages
R2 (Observable)	Background DR-observable >4 blot haemorrhages in 1 hemifield only
R3 (referable)	Background DR-referable Any of the following: > 4 blot hemorrhages in both superior & inferior fieldsvenous bleeding IRMA
R 4 (proliferative)	PDR Any of the following: active new vessels vitreous hemorrhage refer ophthalmology
Maculopathy	**Description**
MO	No blot haemorrhages/hard exudates <2 disc diameters of the centre of fovea
M1 (observable)	Blot hemorrhages /hard exudates within a radius of >1 but <2disc diameters of centre of fovea
M2 (referable)	Blot haemorrhages /hard exudates within a radius of < 1disc diameter of the centre of fovea

Table No. 4: Classification system for retinopathy and maculopathy.

Lesions of diabetic retinopathy:

Micro aneurysms: Originally noted by Mackenzie and Nettleship - Seen as red, round intraretinal lesions. Appear to derive from retinal capillaries and found in vicinity of occluded capillaries. Microaneurysms are clinically visible only when they are above 30 microns and the upper limit is 125 microns. More microaneurysms are seen on fundus angiography than clinically. On angiography they appear as hyperfluorescent dots.

Generally they fill during early venous stage and some may leak dye to the surrounding retina.

Intraretinalhaemorrhages: This appears secondary to ruptured microaneurysms,capillaries and venules. The shape of the intraretinalhaemorrhage depends on thelocation within the retinal layer. Dot and blot haemorrhage – located in deeper layers of retina (between outer plexiform and inner nuclear layer of the retina) Flame shaped haemorrhages – located in the superficial nerve fibre layer Intraretinalhaemorrhages resolve in 6-12 week period.

Hard exudates: These are collection of lipid adjacent to microvascular leakage.Intraretinalexuates appears as creamy – yellow flakes, flecks or dots. They may bearranged in individual streaks, in clusters, or in large circinate rings (if so almost always surround leaking microaneurysms or areas of capillary non perfusion). The hard exudates have an affinity for the posterior pole, particularly the macula.[33]

Soft exudates: Also known as cotton wool spots, it is seen secondary to arteriolarclosure. It occurs in more advanced NPDR. These are small infarcts of nerve fibrelayer.These appear as fluffy white/yellow white striations of nerve fibre layer. Cotton wool spots generally resolve in 2-3 months.

Venous beading and loops

Represent focal areas of venous dilatation. Other venous abnormalities include loops and reduplication of venous segments. The degree of venous beading in ocular fundus images has been shown to be a more powerful predictor of conversion to proliferative diabetic retinopathy than any other type of retinal abnormality. Further, the

degree of venous beading has been shown to be well correlated with disease progression. Reduplication of veins is the least common retinal abnormality but is of the most important prognostic significance. These changes are associated with capillary non perfusion and retinal ischemia.[34]

Foveal Avascular Zone Abnormalities: Normally FAZ is approximately 350 to 750 microns in diameter. Abnormalities seen in the FAZ are irregular intercapillary spaces. Decrease vision without macular oedema warrants a suspicious outlook towards the FAZ.

Intraretinalmicrovascular abnormalities: Intraretinalmicrovascular abnormalities (IRMA) represent either new vessel growth or remodelling of pre-existing vessels through endothelial cell proliferation within the retinal tissues to act as shunts through areas of nonperfusion. They are irregular, segmental dilatation of the capillary bed running between the arteriole and venule adjacent to capillary non perfusion area. They stain on fluorescein angiography, but usually do not leak fluorescein. These eyes are at high risk of developing PDR.[35]

Proliferative lesions: Proliferative diabetic retinopathy .As a response to retinalischemia there is development of extra retinal fibrovascular proliferation. For thediagnosis of PDR the following lesions are necessary: presence of newly formed blood vessel and/or presence of fibrous tissue arising from retina or optic disc and extending along the inner surface of the retina or into the vitreous cavity. These immature vessels have a propensity to bleed. They also cause development of firm adhesion between retina and vitreous body, and when they contract can cause tractional retinal detachment.[35]

Neovacularisation of disc (NVD): It is seen as fine vessels bridging across physiological cup. NVD has a tendency to follow temporal arcade. Grows between the internal limiting membrane of the retina and posterior hyaloids face of the vitreous and adheres to it. As NVD matures fibrous tissue grows to accompany it. With time the fibrovascular bridge may form linking the superior and inferior temporal arcade.[35]

Fig No. 8: Neovacularisation of disc

New vessels arising from elsewhere (NVE): Seen as wheel like networks with vessels radiating like spokes from centre and circumferential vessel bounding the periphery. When vitreous haemorrhage does occur it assumes a classic boat like configuration.

High risk characteristics:

NVD -1/4th TO 1/3rd in extent

NVD with pre-retinal haemorrhage

NVE with vitreous haemorrhage

Advanced diabetic eye:

Advanced diabetic eye disease is the end stage of proliferative retinopathy defined by any of the following potentially blinding complication of proliferative diabetic retinopathy.[36]

Severe vitreous haemorrhage

New vessels in the iris

Neovascular glaucoma

Tractional retinal detachment

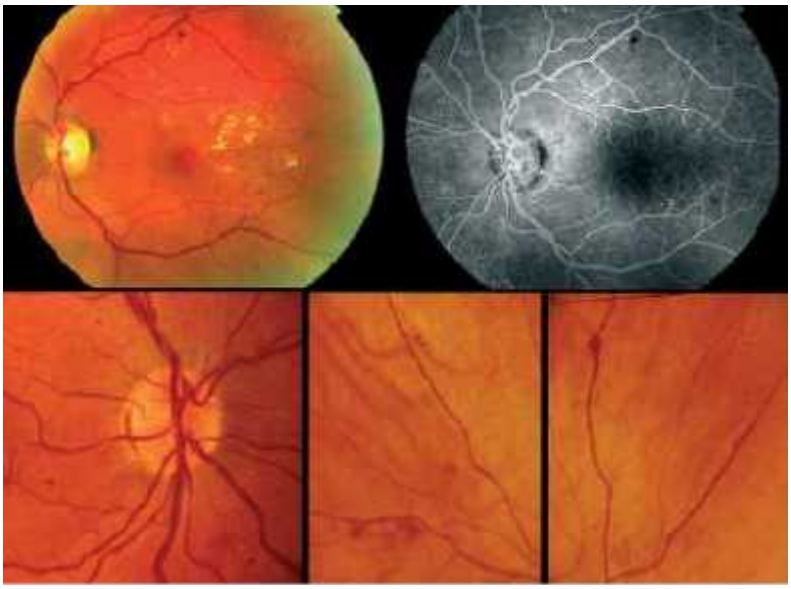


Fig No. 9: Non-proliferative diabetic retinopathy Cardinal signs are retinal microaneurysms,haemorrhages, and hard exudates ; and intraretinalmicrovascular abnormalities; venousbeading ; and venous loop formation.

Preretinopathy	**Mild NPDR**	**Moderate NPDR**	**PDR**	**Advanced DED**
Abnormalities of:	Microaneurysms	Venous bleeding	NVE	NVI
Electrophysiology	Intra-retinal Hemorrhages	Venous loop	NVD	NVG
Psychophysics	Cotton wool spots	Cotton wool spots	Fibrovascular proliferation	Vitreous Hemorrhage
Retinal blood Flow	Venous dilatation	Cluster hemorrhages		TRD
Vascular Permeability	Exudates	IRMA		

Table No.5: Progression of diabetic retinopathy

Glycemic Control:

Glycemic control has a strong influence on many indices of diabetic retinopathysuch as prevalence of retinopathy, incidence of retinopathy, progression of retinopathy, need for focal and scatter photocoagulation, and loss of visual acuity.[37] The influence ofglycemic control is apparent in both type 1 and type 2 diabetes. The concept of a laboratory cutpoint for blood glucose normality is vague. however, 5–9.8% of patients over age 40 in developed countries have typical lesions of diabetic retinopathy even though they do not meet criteria for diabetes.[38]Retinopathy consistent with diabetic retinopathy can develop in certain patients whose blood glucoses range in the normal range for the population; in adults over the age of 49, the 5-year incidence of such an event is 10%. This may in part reflect increased genetic susceptibility to the effects of hyperglycemia. Responsiveness to treatments for manifestations of diabetic retinopathymay also depend on glycemic control. Failure of DME to respond to focal/grid laser photocoagulation has been associated with higher glycosylated hemoglobin.[39]

INVESTIGATIONS for a case of diabetic Retinopathy:

1. Regular:

Technique	**Advantages**	**Disadvantages**	**Recommendatations**
Direct Ophthalmoscopy	-Mobile -Inexpensive	-Requires pupil dilatation -Small field -Low sensitivity -Less effective than slit- amp biomicroscopy	-Optional for Screening -Pupils must be dilated
Indirect Ophthalmoscopy	-Mobile -Large field -Relatively Inexpensive	-Requires pupil dilatation -small abnormalities maybe missed -Less effective than Slitlampbiomicroscopy	-Optional for Screening -Pupils must be Dilated
	-Large field	-Requires pupil	-Required for

Slit-Lamp Biomicroscopy		dilatation -Immobile -Requires special lenses	ophthalmologic examination
Non Mydriatic retinal photography	-Large field -non-medical staff canbe trained to use -can be digitally stored	-Relatively inexpensive -dark space required	Recommended for screening

Table No. 6: Investigations available for Diabetic Retinopathy

2. Fluorescein angiography:

Fluorescein angiography ranks as one of the crucial diagnostic aids for detecting ocular pathophysiologic mechanisms. Chao and Flocks provided the earliest description of fluorcscein angiography in 1958 and it was introduced into clinical use in 1861 by Novotny and Alvis.[40] The interaction of pthalic acid anhydride and resorcinol results in the formation of sodium fluorescein (C20 H10 05 Na2). It has a low molecular weight. It is highly soluble in water. It absorbs light maximally between 480 to 510nm wavelength whilst its emission spectrum peaks at 530nm. Toxic reaction to fluorescein is rare. These mainly include nausea, vomitting, urticaira, skin rashes; hypotension and shock.

The various phases of fluorescein angiography normally observed are as follows.

1. Choroidal phase

- 8 to 10 seconds after injection of dye in antecubtal vein. (arm to retina time)
- Seen as a diffuse fluorescent flush
- Patchy filling may be seen

2. Retinal arterial phase

- 8.5 to 11.0 seconds after injection of dye in antecubital vein.

- Temporal artery fills somewhat earlier than the nasal one and the upper artery slightlybefore the lower one.

3. Capillary phase

- Around the disc are seen the radial peri-papillary capillaries.
- Macula shows the typical capillary pattern with the central avascular Zone (500microns in diameter).
- No special pattern can be recognised in the middle and far fundus periphery.

4. Venous phase

- Earliest venous filing occurs at the posterior pole.

5. Recirculation phase

- Begins within the first minute after injection.

In diabetic retinopathy, fluorescein angiography is useful for early detectionof neovascularization, areas of capillary non perfusion, intraretinalmicrovascularabnormalities, microaneurysms etc. It is also done before the retina is subjected tophotocoagulation.

3. OCT

The development of imaging equipment is allowing new features of diabeticmacular oedema to be identified, which may have implications for treatment in thefuture.[41] OCT has identified macular pathologies that cannot be observed by clinical examination and therefore have not been part of prognosis and treatment planning in the past.

TREATMENT OF DIABETIC RETINOPATHY

I. Medical line of treatment

II. Photocoagulation

III. Surgical line of treatment

I. Medical line of treatment

Control of diabetes by oral hypoglycaemic drugs, conventional insulin treatmentor continuous insulin pumps play an important role.Clofibrate has been claimed to beuseful in clearing retinal hard exudates.[42] Calcium dobesylate has been shown to have a beneficial effect on abnormal retinal capillary permeability.

Agent	**Molecules tried**
Antiplatelet/anti-coagulants	Aspirin Ticlopidine
Aldolasereductase inhibitors	Ponalrestat Tolrestat
GH suppressors	Octreotide
Anti-angiogenesis	Curcumin
ACE inhibitors	Candesartan Perindopril
PKC-beta inhibitors	Ruboxistaurin

Table No. 7: Potential molecules for the management of diabetic retinopathy

II (i) Panretinal Photocoagulation (PRP)

a. Pretreatment Discussion with Patients:

Patients usually need numerous follow-up visits and may require

supplementary laser treatment.

- PRP reduces the risk of visual loss and blindness.
- Although laser treatment is effective, some patients may still develop vitreoushemorrhage. The hemorrhage is caused by the diabetes and not by the laser; itmay mean the patient needs more laser treatment.
- Laser treatment often reduces peripheral and night vision; treatment maymoderately reduce central vision. This short-term side effect is compensated

bythe significant long-term reduction in severe vision loss and blindness inlasertreatedpatients.

b. Lenses for PRP:

The three-mirror Goldmann contact lens has a central opening for treating the posterior pole, and side mirrors for treating the mid peripheral and peripheraretina. Disadvantages: small field of view, which requires continual manipulation of the lens to complete treatment. Spot size is set at 500μm.

Newer wide-angle contact lenses are often used. Although the image is inverted, there is a large field of view allowing for many burns with the field while easily maintaining orientation to the disc and macula. The optics of these wide-angle lenses will affect the laser spot size on the retina. Wide-angle indirect ophthalmoscopy lenses provide an inverted image, but show a large field of view and a magnification of the spot in the retina. Scatter treatment can be applied to a large area of retina in a single image, and it is easy to visualize the disk and the macula.

c. Technique for PRP:

i. The pupil should be fully dilated and topical anesthesia is used. Retrobulbarsubtenons anesthesia to reduce pain and decrease eye motion can be employed asnecessary.

ii. The most common wavelengths used are Argon green, blue green (generallyavoided currently), and 532 green laser, using the slit-lamp delivery system. Incase of hazy media, Krypton red or diode red laser (814 nm) can be used.

Slitlamp treatment is most commonly done through a contact lens but can also be performed using indirect ophthalmoscopy. For example, when treatment is given under general anesthetic.

iii. Typical initial settings on the Argon laser would be 500 μm spot size, a 0.1second exposure and 250-270 mw power. The power is gradually increased until a

whitish reaction is obtained on the retina. The lesions are placed 1 burn width apart.

iv. A total of 1600-3000 burns are placed in 1 or more sittings, carefully avoidingthemacular area and any areas of tractional elevation of the retina. The burns areplaced 2 to 3 disc diameters away from the center of the macula and 1 discdiameter away from the disc, usually outside the arcades and extendedperipherally up to the equator and beyond.

v. Laser treatment should not be applied over major retinal veins, preretinalhemorrhages, darkly pigmented chorioretinal scars, or within 1 DD (200-300 μm)of center of macula, so as to avoid risk of hemorrhage or large scotomas.

vi. Other considerations:

- Additional photocoagulation is needed if there is evidence of worsening ofproliferative DR.
- Add laser burns in between scars of initial treatment further peripherally and alsoat the posterior pole, sparing the area within 500-1500 μm from the center ofcmacula.
- Favor quadrants with active new vessels or areas with intraretinalmicrovascularabnormalities where scars are more widely spaced and areas of severe ischemianot previously treated, such as the temporal part of the posterior pole.
- Direct treatment of NVE in between scars is possible.

d. Panretinal (Scatter) Photocoagulation Technique Following Diabetic Retinopathy Clinical Research Network (DRCRNet) Consensus.

Panretinal (scatter) photocoagulation initially consists of 1200 to1600 burns (or the equivalent area treated with a multi-spot laser), with a spot size on the retina of approximately 500 μm given over 1 to 3 sittings and completed within eight weeks days of initiation, As shown in the below table No.8.

Burn Characteristic	**Direct/Grid Photocoagulation(Modified ETDRS technique)**	**Mild Macular Grid Photocoagulation technique**
Direct treatment	Directly treat all leaking microaneurysms in areas of retinal thickening 500 to 3000 µm from centre of the macula	Not applicable
Change in MA color with direct treatment	Not required, but atleast a mildgray white burn should beevident beneath all microaneurysms.	Not applicable
Burn size for direct treatment	50-100µm	Not applicable
Burn duration for direct treatment	0.05-0.1 sec	Not applicable
Grid treatment	Applied to all areas with diffuseleakage /non-perfusion withinarea described below forTreatment	Applied to entire are described below for treatment
Area considered for grid treatment	-500-300 µm superiorly, nasally and inferiorly from center of macula. -500-3500 µm temporally from macular center. -No burns are placed < 500µmof disc	-500-300 µm superiorly, nasally and inferiorly from center of macula. -500-3500 µm temporally from macular center. -No burns are placed < 500 µm of disc

Burn size for grid treatment	50-100 µm	50 µm
Burn duration for grid Treatment	0.05-0.1 sec	0.05-0.1 sec
Burn intensity	Barely visible (light gray)	Barely visible (light gray)
Wavelength(grid and focal treatment)	Green to yellow	Green

Table No.8: Modified-EDTRS and the Mild Macular Grid Photocoagulation Techniques

II(ii)Laser photocoagulation

Focal laser

Possible mechanisms of action

- Direct closure of leaking microaneurysms
- Laser induced endovascular thrombosis
- Heat-induced contraction of the vessel wall Procedure – compare vascular landmarks on fluoroscein angiograms are compared to the fundus.
- Fundus is precisely located.
- All fluorescein leaks are directly treated.
- Laser burns are spaced one burn width apart.
- The end point is whitening or darkening of the treated area.
- Laser parameters- 100-200 microns spot size, 0.1 second duration, 50-100 Mw power

II (iii) Grid laser

Photoreceptor destruction increases inner retinal oxygenation resulting invasoconstriction, decreased retinal blood flow and decreased vascular leakage

- Retinal pigment epithelium damage causes retinal capillary and venuleendothelial dysfunction.
- Photocoagulation debridement of dysfunctioning RPE cells may result inenhanced blood retinal barrier
- Grid photocoagulation is performed in areas of thickened retina showing diffuseleakage or capillary dropout.
- Laser parameters- 100-200 micron spot size, 0.1 to 0.5 sec duration, 50-100mwpower, light intensity burns.
- Grid is not done within 500 microns of the macula/disc margin

Pan retinal photocoagulation for proliferative diabetic retinopathy:

It may alter the extracellular modulating factors responsible for control ofintraocular neovascularisation. Destruction of oxygen consuming photoreceptors andRPE may improve inner retinal oxygenation, thus decreasing stimulus forvasoproliferative factors

Procedure – laser spots are placed outside the arcades and extend peripherally up to theequator and beyond

- Laser settings- 300-500micron spot size, 0.1 second, 220-27- mw power.
- Power and duration are adjusted till gray white reaction is achieved
- Burns are placed one burn width apart; a total of 1600-2000 burns are applied in 2-3 sittings starting first with the inferior quadrant, because if vitreoushaemorrhage occurs then the inferior quadrant will be difficult to treat.

Size (on retina)	500 µm
Exposure	0.1sec recommended
Intensity	Mild white(i.e. 2+ to 3+)
Distribution of sessions	Edges 1 burn width apart
Number	1-3
Nasal proximity to disk	No closer than 500 µm
Temporal proximity to center	No closer than 3000µm
Superior/inferior limit	No further posterior than 1 burn within temporal arcades
Extent	Arcades (3000µm from macular center) to atleast the equator
Total number of burns	1200-1600n
Wavelenghth	Green or yellow(red can be used if vitreous hemorrhage)

Table No. 9: The burn characteristics for panretinal photocoagulation

III. Surgical line of treatment

Pituitary ablation by ytrrium 90 implantation or hypophysectomy advocated as a treatment for proliferative retinopathy following a case report by Poulstein in 1953 Kohner and co-workers have advocated pituitary ablation as a treatment of choice for diabetic retinopathy characterised by progressive capillary dilatation, capillary occlusion, cotton wool spot formation and severe proliferative diabetic retinopathy.[43]

(i)Vitrectomyforms a mainstay of the surgical line of proliferative treatment of diabetic retinopathy with its attendant complications.[44]

Indications for vitrectomy in severe diabetic retinopathy

(a) Media opacity

- Non clearing vitreous haemorrhage
- Pre macular haemorrhage

(b) Tractions

- Progressive fibrovascular proliferation
- Tractional retinal detachment involving macula
- Combined rhegmatogenous and tractional retinal detachment
- Macular oedema associated with taut posterior hyaloids

(c) Others

- Recurrent vitreous haemorrhage
- Anterior hyaloidfibrovascular proliferation
- Goals of vitrectomy in proliferative diabetic retinopathy
- To clear the media
- To relieve all tractions on the retina
- To reattach retina where needed
- Closure of retinal breaks if any

The aims of vitrectomy*:*

1. To clear media opacities such as vitreous haemorrhage;
2. To remove proliferative tissue causing tractional forces on the retina;

3. Prevent further neovascularization by laser endophotocoagulation and by removalof vitreous gel so removing the scaffold along which fibrovascular tissue canproliferate;
4. Repair retinal detachment by excising tractional membranes and removingfibrovascular tissue from the surface of the retina.

Surgical technique:

The central core vitreous gel is first removed with the vitrectomy probe. If there isno posterior vitreous separation then this is next elevated in an accessible area withouttight vitreo-retinal adhesion. The optic disc is often a good location for this if no posteriorvitreous separation exists elsewhere. The neovascular membrane complexes are nowdissected away from the retina being careful to ensure that the dissection is in the correctplane and not in a false plane of vitreo-schisis, which can occur in PDR.This can beachieved using an 'en-bloc' dissection then delamination technique using the remainingposterior hyaloids face to exert antero-posterior traction to open up the plane ofdissection . Alternatively via circumcision from the posterior hyaloid face and thensegmentation of neovascular complexes followed by their delamination from the surfaceof the retina. 'En-bloc' dissection and delamination involve the use of horizontal cuttingscissors to cut through vascular 'pegs' joining the membrane complexes to the retina.

Segmentation may involve vertical cutting scissors to divide membranes between pegs inorder to relieve traction between these. Once the traction has been relieved the retina iscarefully inspected for signs of retinal breaks. If identified these are treated with laser to form a surrounding chorioretinal adhesion and then gas or oil is used as tamponade. PRP is completed if deficient using indirect or endolaser and often continued up to the pars plana, which possibly reduces the rate of post vitrectomy bleeding.

Complications:

1. Post-vitrectomy vitreous cavity haemorrhage can occur in some patients.(10-20%)
2. Retinal tears and holes

3. Raised IOP
4. Cataracts can develop following vitrectomy surgery, especially in patients over 60 years.
5. Rubeosis-rare.

Visual prognosis post vitrectomy: Overall, about 70–90% of patients will get animprovement in vision with vitrectomy surgery with the final acuity result depending largelyon the degree of maculopathy. Less than 5% may be made worse.

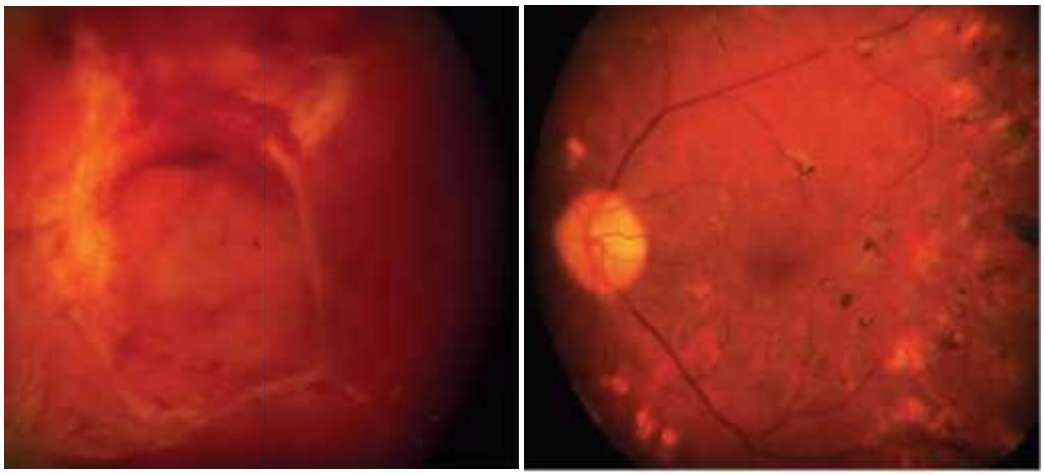


Fig No. 10: (a,b) Pre and post vitrectomy for vitreous haemorrhage and traction retinal detachment. VA postoperatively 6/12.

(ii) Intravitreal drugs:

Intravitreal triamcinolone has been used for macular oedema refractory to laser treatment.[45] Using OCT has shown this treatment to reduce retinal thickness.

Improvement in vision has been recorded in some cases. However, this effect is transient and appears not to be sustained despite repeat injections. A recent randomised clinical trial showed that laser is more effective than intravitreal triamcinolone at preventing visual loss. There are also significant side-effects with intravitreal triamcinolone, with raised intraocular pressure and cataract being very common.

Intravitreal injections of anti-VEGF such as ranibizumab, pegaptanib andbevacizumab are currently being explored as therapies for DR. Several trials have showna beneficial

effect when anti-VEGF agents are used alone or combined with laser formacular oedema.[46]

A recent randomised trial of 854 eyes has shown that ranibizumab combinedwith laser for 'centre involving' diabetic macular oedema (ie DMO that involves thefovea) is more effective than laser alone.[47]At 1 year there was a significantly greatermean increase in visual acuity in eyes treated with ranibizumab and laser, than those whohad only laser. Multiple injections were required, however, and were also necessary inthe second year of the study, but the authors report no systemic side-effects attributable tothe treatment. There is concern that these drugs can, though rarely, affect patientssystemically to increase the risk of stroke and myocardial infarction. Additionally, VEGFmay be necessary in maintaining the function of healthy retina. However, it is very likelythat anti-VEGF therapy in combination with laser has a place in the management of bothPDR and macular oedema.

Severity of Retinopathy	**Presence of CSME**	**Follow-up (Months)**	**Scatter(Panretinal) Photocoagulation**	**Fluorescein Angiography**	**Focal laser**
Normal/ Mild NPDR	No	12	No	No	No
Mild to Mod NPDR	No	6-12	No	No	No
	Yes	2-4	No	Usually	Usually
Severe to very Severe NPDR	No	2-4	Sometimes	Rarely	No
		2-4	Sometimes	Usually	Usually
Non high-risk PDR	No	2-4	Sometimes	Rarely	No
		2-4	Sometimes	Usually	Usually

High risk PDR	No	3-4	Usually	Rarely	No
		3-4	Usually	Usually	Usually
High risk PDR Not amenable to PC	-	1-6	Not possible	Occasionally	Not possible

Table No. 10: Management recommendations for patients with Diabetes retinopathy

The recommended follow up regimen for newly diagnosed diabetic patients is asgiven in table no.11

	IDDM	**NIDDM**
1st Examination	5years after onset if patient's age < 30 yr. At puberty after diagnosis	On diagnosis immediately if patient's age>31 yr.
Follow-up	Annually *	Annually *

* normal at first examination

Table No. 11: Ocular examination and follow up schedule in diabetes subjects

BRIEF ACCOUNT OF THE LIPID CHEMISTRY

The lipids are a heterogenous group of compounds including fats, oils,steroids, waxes and related compounds that are related more by their physical thanchemical properties.

The lipids have following functions.

1. They are important dietary constituents not only because of their high energyvalue, but also because of the fat soluble vitamins and essential fatty acidscontained in the fat of natural foods.
2. Fat is stored in adipose tissue, where it also serves as a thermal insulator in thesubcutaneous tissues and around certain organs.
3. Non polar lipids act as electrical insulators, allowing rapid propagation ofdepolarization waves along myelinated nerves.
4. Combination of lipid and protein (lipoproteins) serve as the means oftransporting lipids in the blood.

The lipids are classified as given below.[48]

A) Simple lipids (esters of fatty acid with various alcohols)

- Fats: esters of fatty acids with glycerol. Oils are fats in liquid state
- Waxes: esters of fatty acids with higher molecular weight monohydric alcohols.

B) Complex lipids: These are esters of fatty acids containing groups in addition to analcohol and a fatty acid groups)

- Phospholipids: lipids containing in addition to fatty acids and an alcohol, aphosphoric acid residue.
- Glycolipids: lipids containing a fatty acid, sphingosine and carbhohydrate.

Other complex lipids: lipids such as sulfolipids and aminolipids. Lipoproteins may also be placed in this category.

C) Derieved lipids: These include fatty acids, glycerol, steroids, other alcohols, fattyaldehydes, hydrocarbons, lipid soluble vitamins and hormones.

FATTY ACIDS

They are present as such in minute concentrations in plasma cells. Fatty acidsoccur mainly as esters in natural fats and oils but do occur in the unesterified form asfree fatty acids, a transport form found in the plasma. Fatty acids that occur in naturalfats are usually straight chain derivatives containing an even number of carbon atoms.

Lipid Chemistry

Fatty acids are of two types

a. Saturated fatty acids - Those which contain no double bonds.

b. Unsaturated fatty acids- Those which contain one or more double bonds.

Saturated fatty acids having 10 or less carbon atoms are called "Lower fatty acids"

e.g. - Acetic acid, butyric acid

Saturated fatty acids having more than 10 carbon atoms areCalled "higher fatty acids". e.g. - Palmitic acid, stearic acid.

Unsaturated fatty acids are classified according to degree of saturation.

1. **Mono unsaturated fatty acids** - These are those fatty acids which contain one

double bond. E.g. Oleic acid.

2. **Polyunsaturated fatty acids** - These are fatty acids which contain more than onedouble bond.

eg. Linoleic acid series

Linolenic acid series

Arachidonic acid series

Essential fatty acids are those which cannot be synthesized in body and mustbe provided in the diet. Lack of these essential fatty acids in diet can produce growthretardation and other deficiency syndromes. Free fatty acids are immediately availableenergy source and provide much of the energy requirements of body. Normal valueranges from 250 - 400 mg / dl.

CHOLESTEROL

Cholesterol is widely distributed in all cells of the body. It is the best knownsteroid because of its association with atherosclerosis and heart disease.

It occurs as a white or faintly yellow, almost odorless, pearly leaflets orgranules. It is insoluble in water. Sparingly soluble in alcohol and soluble in ether,chloroform, hot alcohol, ethyl acetate alcohol and vegetable "oil.

Cholesterol is found in largest amounts in normal human adult brain andnervous tissue of about 20%, in liver - 0.3%, skin - 0.3%, intestinal mucosa 0.2% andcertain endocrine glands namely adrenal cortex contain about 10%.

The normal level of serum total cholesterol in adults varies from 150-250 mg/dl

TRIGLYCERIDES (NEUTRAL FATS)

These molecules are used to provide energy. In the body, stored fat in adiposetissue is the storage form of energy.

Important sites of adipose tissue are subcutaneous tissue around some internalorgans and omentum. Fat under the skin prevent heat loss in winter and the intestinalorgans get support from fat around them. The triglycerides constitute the body's maincaloric reserve. Normal value ranges from 40-150 mg %.

PHOSPHOLIPIDS

Phospholipids are compound lipids. They contain in addition to fatty acids andglycerol one more alcohol or phosphatidic acid, residue nitrogen containing base andother substituents.

Phosphatidic acid is important as an intermediate in the synthesis oftriacylglycerols as well as phosphoglycerols but is not found in any great quantity intissues.

They are classified into 3 groups.

1. **Glycerophopsholipid**- Here glycerol is the alcohol group.

    eg - Lecithin, Cephalin

2. **Phosphoinositides**- Here inositol is the alcohol group.

    eg - phosphatidylinositol

3. **Sphingophospholipids**- Here sphingol is the alcohol group.

    eg - Sphingomyelin

The lecithins are the most abundant phospholipids at the cell membrane and representa large portion of body's store of choline.

Dipalmitoyl lecithin is a very effective surface active agent and a majorconstituent of the surfactant preventing adherence, due to surface tension of the innersurface of lungs.

Phosphatidylethanolamine (cephalin) and phosphatidyl serine differ fromphosphatidylcholineony in that ethanolamine or serine, respectively replaces choline.

Sphingomyelins are found in large quantities in brain and nerve tissue.

On hydrolysis, the sphingomyelins yield a fatty acid, phosphoric acid, cholineand a complex amino alcohol, sphingosine. No glycerol is present. The combinationof sphingosine plus fatty acid is known as ceramide, a structure also found in theglycophospholipids.

GLYCOLIPIDS

Glycolipids are widely distributed in every tissue of the body, particularly innervous tissue such as brain. They occur particularly in the outer leaflet of the plasmamembrane, where they contribute to cell surface carbhohydrates.

The major glycolipids found in animal tissues are glycosphingolipids. Theycontain ceramide and one or more sugars. Galactosylceramide is a majorglycosphinglipid of brain and other nervous tissue, found in relatively low amountselsewhere. It contains a number of characteristic C24 fatty acids, eg, cerebronic acid.

Galactosylceramide can be converted to sulfogalactosylceramide (sulfatide),present in high amounts in myelin. Glucosylceramide is the predominant simpleglycosphingolipid of extraneural tissues, also occurring in the brain in small amounts.

Gangliosides are complex glycosphingolipids derived fromglucosylceramidethat contain in addition tone or more molecules of sialic acid. Neuraminic acid is theprincipal sialic acid found in human tissues. Gangliosides are present in nervoussystems in high concentration.

LIPO PROTEINS OF PLASMA

In plasma, cholesterol and triglycerides form integral component ofmacromolecule complex called lipoprotein which are conjugated proteins. Lipid partis the prosthetic group and lipid free protein are designated as apolipoproteins or apoproteins. Protein separation including electrophoresis and ultracentrifugation showsprogress in lipoprotein chemistry.

Teselius et al in 1941 reported existence of two lipoprotein classes separatedby moving boundary electrophoresis.

In 1954 Gofmen et al separated lipoproteins by ultra centrifugation into fivemajor density classes.

OVERVIEW OF LIPOPROTEINS AND LIPOPROTEIN METABOLISM

Lipoproteins are microemulsions composed of lipids (cholesterol, cholesterylester, triglyceride and phospholipid) and proteins (apoproteins). Their function is totransport non water soluble cholesterol and triglycerides in plasma. Lipoproteins arespherical particles containing a central core of non-polar lipids (primarily triglyceridesand cholesteryl ester) and a surface monolayer of phospholipids and apoproteins.

Lipoproteins have been classified on the basis of their densities during ultracentrifugation into :[49]

1) Chylomicrons

2) Very low density lipoprotein (VLDL)

3) Low density Lipoprotein (LDL)

4) High density lipoprotein

5) Lipoprotein a

Table 12 CLASSIFICATION OF LIPOPROTEINS[49]

Lipoprotein	Source	Diameter (nm)	Density (g/ml)	Composition		Main lipid components
				Protein (%)	Lipid (%)	
Chylomicrons	Intestine	90-1000	<0.95	1-2	98-99	Triacylglycerol
VLDL	Liver	30-90	0.95-1.006	6-8	92-94	Triacylglycerol
LDL	VLDL	20-25	1.019-1.063	21	79	Cholesterol
Lp(a)		25-35	1.040-1.063	36	55	Cholesterol
HDL	Liver, intestine, VLDL chylomicrons					Phospholipids Cholesterol
HDL1		20-25	1.019-1.063	32	68	
HDL2		10-20	1.063-1.125	33	67	
HDL3		5-10	1.125-1.210	57	43	

CHYLOMICRONS

Chylomicrons are the largest of lipoproteins. They measure about 90-1000nmin diameter and they have the least density as compared to other classes oflipoproteins.[49]

These are particles that are primarily triglyceride bearing and are produced bythe intestine after exogenous fat undergoes digestion. These are responsible for thetransport of dietary triglycerides and cholesterol. Dietary triglycerides are hydrolyzedin the gut,

releasing monoglycerides and fatty acids that are then reesterified to formtriglycerides in the intestinal mucosal cell.

These triglycerides are assembled with newly absorbed cholesterol, apoproteinB48 and the A apoproteins. Upon secretion from the enterocyte, these assembledparticles enter the lymphatic circulation and then the bloodstream, where they acquireC apoproteins and apo E by transfer from HDL.

As chylomicrons enter the plasma, the triglycerides are rapidly hydrolyzed bythe enzyme lipoprotein lipase (LPL), which resides on the surface of capillaryendothelial cells. LPL is synthesized primarily in adipose tissue and striated muscle. Itis secreted and transported to the endothelial surface, where it acts on triglyceride richparticles. Its action requires the presence of apo CII on the surface of the lipoprotein,whereas apo CIII inhibits LPL.

As triglyceride is depleted from the chylomicrons, phospholipids and A and Capoproteins are transferred to HDL. The residual chylomicron particle, which has lost80 to 90 % of its triglyceride and is now relatively cholesterol enriched is calledchylomicron remnant.

VERY LOWDENSITY LIPOPROTEINS

VLDLS are synthesized in the endoplasmic reticulum of hepatocytes and arecomposed of endogenous triglyceride derived from plasma free fatty acids,chylomicron remnants and from de novo lipogenesis .[49]

Nascent VLDLs are secreted into the circulation; contain apoB100 and smallamounts of apo C and apo E. After VLDLs enter the circulation, they are metabolizedin the same manner as chylomicrons by the enzyme LPL, with the fatty acids that areliberated following the same fate as those liberated from chylomicrons. Aftersecretion, VLDLs acquire more C and E apoproteins by transfer from HDL. Inaddition, free cholesterol is progressively exchanged to HDL, where it is esterifiedand the cholesteryl ester is returned to VLDL.

As VLDLs become progressively depleted of triglyceride, a portion of thesurface, including cholesterol, apolipoproteins C and E and phospholipids is removedand contributes to nascent HDL particles.

LOW DENSITY LIPOPROTEINS

LDLs are products of the metabolism of VLDL. They have got a diameter of18-25nm. The only apoprotein in LDL is apo B and only one molecule of apo B ispresent per particle of LDL. Clearance of LDL is mediated by a specific receptorpresent on the surface of both liver and peripheral cells. Once it is bound to thereceptor, the lipoprotein is internalized by an endocytic process. The vesicle thenfuses with a lysosome, where enzymes degrade the apoB and hydrolyze thecholesteryl ester to free cholesterol.

The smaller remnants of VLDL are triglyceride depleted, cholesterol richparticles, some of which are isolated in the IDL compartment, although some remainin the VLDL compartment.

VLDL particles are believed to be secreted in a spectrum of sizes with variousdegrees of triglyceride enrichment. The larger VLDL particles appear to be morerapidly cleared and less likely to be converted to LDL. On the other hand, smallerVLDL particles that are richer in cholesterol may be preferentially converted to LDL.

All peripheral cells express the LDL-receptor (LDL-R), and recycle it to thecell surface upon need for cholesterol. Cholesterol is delivered to these cells throughbinding of LDL to LDL-R, which triggers endocytosis (internalization) of bothspecies. When the need for cholesterol is satisfied, the recycling of LDL-R isdiscontinued. Normally, an LDL particle stays in circulation for no more than a fewdays before being consumed by a cholesterol needing cell.

However, under conditions of sustained cholesterol excess, the particle staysin circulation for longer periods of time, and becomes more vulnerable to undesiredmodifications (e.g. oxidation). As high levels of oxidized LDL are commonly foundin atherosclerotic plaques, they are thought to be the major inducer of atherosclerotiClesions. Hence, LDL became known as bad cholesterol.

HIGH DENSITY LIPOPROTEINS

HDLs also are represented by a spectrum of particles of various sizes anddensities. HDLs are 18-25nm in diameter.

HDL is synthesized in the liver and intestine as a nascent, discoid-shapedparticle that contains predominantly apoA-I, and some phospholipids.[49]Upon

maturation, HDL assumes a spherical shape, and the composition of its core lipidsbecomes very similar to that of LDL. However, the relative higher protein content inHDL renders the particle denser and more resistant to undesired modifications.

Unlike LDL, HDL is not recognized by LDL-R, and cannot deliver cholesterolto tissue cells. Instead, it has the ability to remove excess peripheral cholesterol andreturn it to the liver for recycling and excretion. This process, called reversecholesterol transport, is thought to protect against atherosclerosis. Observationalstudies over the last 2 decades have consistently shown strong correlation betweenelevated HDL levels and low incidents of coronary artery disease (CAD). Hence HDLhas been dubbed “good” cholesterol.

LIPOPROTEIN (a)

Lipoprotein (a) or Lp (a) has been established as an independent CAD riskfactor. The structure is similar to that of an LDL molecule linked by a disulphidebridge to apoprotein A.Lp (a) levels range from 1-100mg /dl with the largest number of values below20 mg/dl.

Although Lp (a) is structurally similar to LDL, the former appears to beregulated independently and carries an independent relation to overall coronary risk.If serum levels of both LDL and Lp (a) are elevated the risk of CAD is markedlyincreased.

The mechanism by which high levels of Lp (a) are related to coronaryatherosclerosis is unclear. It has been suggested that because of the structuralsimilarities of Lp (a) to plasminogen, high levels of Lp (a) may inhibit thethrombolytic activity of naturally occurring tissue plasminogen activity.

An alternative explanation for the association between elevated Lp (a) levelsand atherosclerosis in that Lp (a) may somehow alter the LDL mediated delivery ofcholesterol to the atherosclerotic plaque.

CHARACTERISTICS AND MECHANISMS OF DYSLIPIDEMIA IN DIABETES

Dyslipidemia is a relatively common problem in patient with diabetes mellitusas compared to non-diabetics. Patients with diabetes typically have an atherogeniclipid profile characterized by elevated triglycerides, increased LDL, VLDL andcholesterol and decreased HDL.

There are several reasons for this association:

First, insulin plays an important role in the regulation of intermediary lipidmetabolism and fluctuations in the degree of diabetic control thus produce a variableeffect on plasma lipoprotein metabolism.

Secondly, many non-insulin dependent diabetic patients are obese, and obesityleads to the development of hyperlipidemia.

Increased LDL

There is an increased proportion of small dense LDL particles. Total LDLlevels may be modestly increased but are comparable with those of the generalpopulation, although an individual with type 2 diabetes mellitus and a total LDL of100 mg/dl may actually have many more circulating small, dense LDL particles thanan individual with normal insulin sensitivity and the same LDL level.

Hypertriglyceridemia

Triglycerides come from the diet (exogenous) or are newly synthesized by theliver (endogenous) using dietary carbohydrate precursors and re esterified fatty acidsabsorbed from peripheral tissues. Exogenous triglycerides circulate as chylomicrons,while endogenous triglycerides combine with hepatic cholesterol to form VLDLparticles, which are secreted into the circulation by hepatocytes.

Abnormalities of LPL activity, HSL activity, and fatty acid metabolism allcontribute to baseline and often extreme postprandial hypertriglyceridemia associatedwith type 2 diabetes and insulin resistance.

Although fasting LPL levels are typically increased in the setting of obesitybecause of the large number of adipocytes, insulin resistance at the level of the fat cellcauses decreased LPL activity and therefore, an abnormal response of LPL to aglucose load.

Diminished LPL activity leads to an accumulation of atherogenic LDLprecursors, such as VLDL in the circulation. HSL activity is increased in type 2diabetes, which causes increased circulating FFA.

In the setting of insulin resistance, adipocytes take up less circulating FFA.

This situation, called reduced fatty acid trapping, allows excess FFA delivery to theliver, which, in turn, causes increased hepatic secretion of VLDL particles. This ismost pronounced and prolonged after a meal.[50-52]

Decreased HDL

The decreased HDL in NIDDM is mostly reflected in decreases in the HDL2subfraction.[53]

Although it is not completely understood how hepatic lipase acts in theregulation of HDL, it is possible that the lower HDL concentrations in NIDDM mayin part be attributable to higher hepatic lipase activity. Hepatic lipase is elevated inobese female NIDDM subjects and increased in thin male NIDDM individuals, theactivity in the latter group decreasing after normalization of glycemia with insulintherapy.

Reduced HDL leads to diminished clearance of cholesterol from peripheraltissues. The actions of CETP illustrate why hypertriglyceridemia and reduced HDLtypically go hand in hand in patients with type 2 diabetes. In the setting of elevatedcirculating triglycerides, CETP allows an increased influx of VLDL triglycerides intoHDL particles. This occurs as an exchange reaction, with a simultaneous efflux ofcholesteryl ester out of HDL

particles. This process leads to reduced HDL levelsowing to increased clearance of HDL particles.

There is also reduced production of HDL particles in type 2 diabetes owing toabnormal LPL activity, causing decreased conversion of dense, triglyceride- rich HDLto more buoyant particles.

Qualitative Changes in LDL: LDL Particle Size

Aalready mentioned, although the total LDL level may be normal or onlymodestly elevated in patients with type 2 diabetes and obesity there are frequentlyqualitative changes in LDL particles that confer increased risk of CAD.

As individuals proceed from normal insulin sensitivity to insulin resistance,VLDL particles become larger and LDL particles become smaller. Individuals with

type 2 diabetes have been shown to have smaller, denser LDL particles even afteradjusting for elevated triglyceride levels and lower HDL levels.

LDL particles are divided into two subclasses based on particle size andatherogenicity. Pattern A particles are buoyant and pattern B particles are small, denseand more atherogenic. Pattern B molecules are formed by a lipid exchange processsimilar to that that occurs in HDL particles in the setting of elevated triglycerides.Through the actions of CETP, triglyceride from VLDL is exchanged with cholesterylester from LDL. Hydrolysis of the triglyceride-rich LDL particle produces smaller,denser LDL particles.

Pattern B particles are considered more atherogenic than pattern A particlesfor several reasons: they adhere to and penetrate the arterial wall more easily, they aremore toxic to endothelial cells, they exert a procoagulant effect by causing greaterproduction of plasminogen activator inhibitor-1 by endothelial cells, and they areoxidized more easily.

Studies done in the general population and in diabetic populations have shownthat individuals with elevated levels of pattern B particles are at higher risk of CAD.

Glycosylation of LDL

Epidemiological data have demonstrated that higher HbAlC levels are

associated with higher rates of CAD. Severe hyperglycemia may worsen diabeticdyslipidemia via glycosylation of LDL particles. Glycosylated LDL particles arethought to have increased atherogenicity. Advanced glycation end products maymodify LDL particles such that they have reduced affinity for hepatic LDL receptorsand thereby a prolonged half-life.

LDL particles that "live longer" may have a greater likelihood of becoming

oxidized or taken up by macrophages, in turn leading to the formation of foam cells.[54-55]

Increased VLDL:

The most common alteration of lipoproteins in NIDDM is an elevation inVLDL, as reflected by either increased total triglyceride or VLDL triglycerideconcentrations. Abnormalities in both production and clearance of VLDL triglyceridehave been reported in NIDDM. Several studies have observed an overproduction ofVLDL triglyceride. Although there are fewer studies of VLDL Apo B metabolism inNIDDM, results indicate that there is a clearance defect similar to that for VLDLtriglyceride, whereas VLDL Apo B production may be influenced primarily byobesity. Subjects with type 2 diabetes mellitus have a decrease in fractional catabolicrate (FCR) for VLDL Apo B.[56]

Haffneret al[57] found slower clearance of chylomicron Apo B in

hyperlipidemic subjects with NIDDM. The proportional decrease in clearance ofVLDL Apo B was similar to that observed for VLDL triglyceride.[58]

There appear to be changes in the composition of VLDL in NIDDM whichmay either reflect or be the cause of alterations in VLDL metabolism. Several studieshave suggested that diabetics may have a large, triglyceride rich VLDL**.**[59]

There are multiple alterations in VLDL metabolism in non-insulin-dependentdiabetes mellitus. NIDDM appears to induce an overproduction of VLDL triglycerideand to a lesser extent, of VLDL Apo B. FCR for both VLDL triglyceride and Apo Bare lower and are associated with lower activities of LPL.

Finally, there are indications that the VLDL particle in NIDDM has alteredcomposition. The mechanism for the overproduction of VLDL is not clear. The mostlikely explanation is that it is a result of the increased flow of substrates, particularlyglucose and free fatty acids, to the liver.

MATERIALS AND METHODS

SOURCE OF DATA:

All type 2 diabetic mellitus patients with diabetic retinopathy attending outpatient department of Ophthalmology.

METHOD OF COLLECTION OF DATA:

STUDY DURATION- December 2014 - May 2016.

SAMPLE SIZE:.

With 33.9%[60] prevalence of diabetic retinopathy among the type 2 diabetes mellitus patients (type 2 diabetes mellitus prevalence is 12.4%[61]) and margin of error as ± 3%. The minimum sample size is 164.

Total sample size =164

Formula;[62]

$$n = \frac{z^2 p(1-p)}{d^2}$$

Where :

n = sample size

z = z statistic(1.96) for 95 % level of confidence

p = expected prevalence

d = margin of error

STATISTICAL ANALYSIS:

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square (χ^2)/ Freeman-HaltonFisher exact test was employed to determine the significance of differences

between groups for categorical data. The difference of the means of analysis variables was tested by ANOVA. If the p-value was < 0.05, then the results were considered to be significant. Data were analyzed using SPSS software v.23.0.

INCLUSION CRITERIA:

All Patients who has been diagnosed type 2 Diabetes Mellitus with Diabetic retinopathy.

EXCLUSION CRITERIA:

a) Patient with type 1 diabetes mellitus.

b) Patients with hypertension.

c) Patients who have undergone treatment earlier for any form of diabetic eye disease.

d) Diabetes patients on hypolipidemic drugs.

e) Patients retinal disease mimicking diabetic retinopathy.

INVESTIGATIONS:

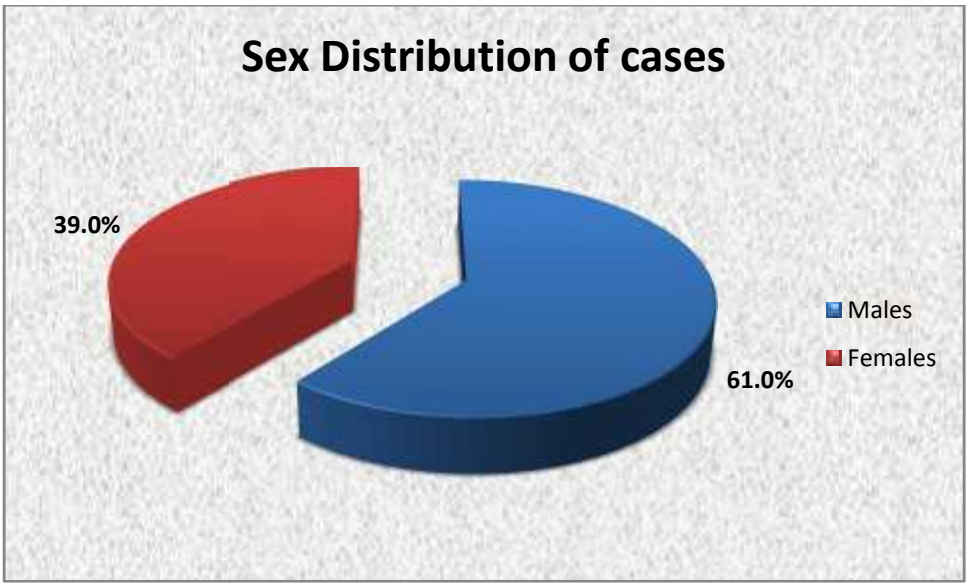
- Blood sugar levels (FBS, PPBS,)
- Lipid profile(Total cholesterol, HDL, LDL, Triglycerides, VLDL)
- Urine Albumin
- HbA_{1C}
- Fundus Fluorescein Angiography if necessary.
- Fundus photograph will be taken wherever feasible.

OBSERVATIONS AND RESULTS

Table No. 13 :Sex Distribution of patients

Sex	N	Percent
Males	100	61.0
Females	64	39.0
Total	164	100

Graph No. 1 : Sex Distribution of patients

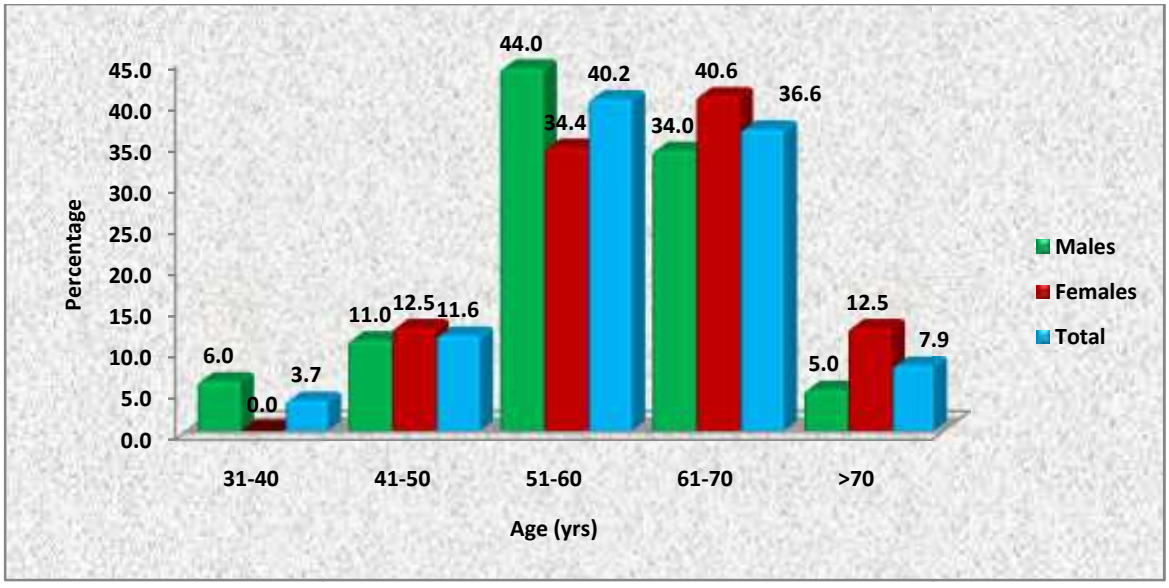


The observation study was conducted on totally 164 consecutive patients of NIDDM. Out of 164, 100 were male and 64 were females.

Table No. 14 : Sex Distribution according to Age of the patients

Age (yrs)	Males		Females		Total		p value
	N	%	N	%	N	%	
31-40	6	6.0	0	0.0	6	3.7	0.09
41-50	11	11.0	8	12.5	19	11.6	
51-60	44	44.0	22	34.4	66	40.2	
61-70	34	34.0	26	40.6	60	36.6	
>70	5	5.0	8	12.5	13	7.9	
Total	100	100.0	64	100.0	164	100.0	

Graph No. 2 : Sex Distribution according to Age of the patients

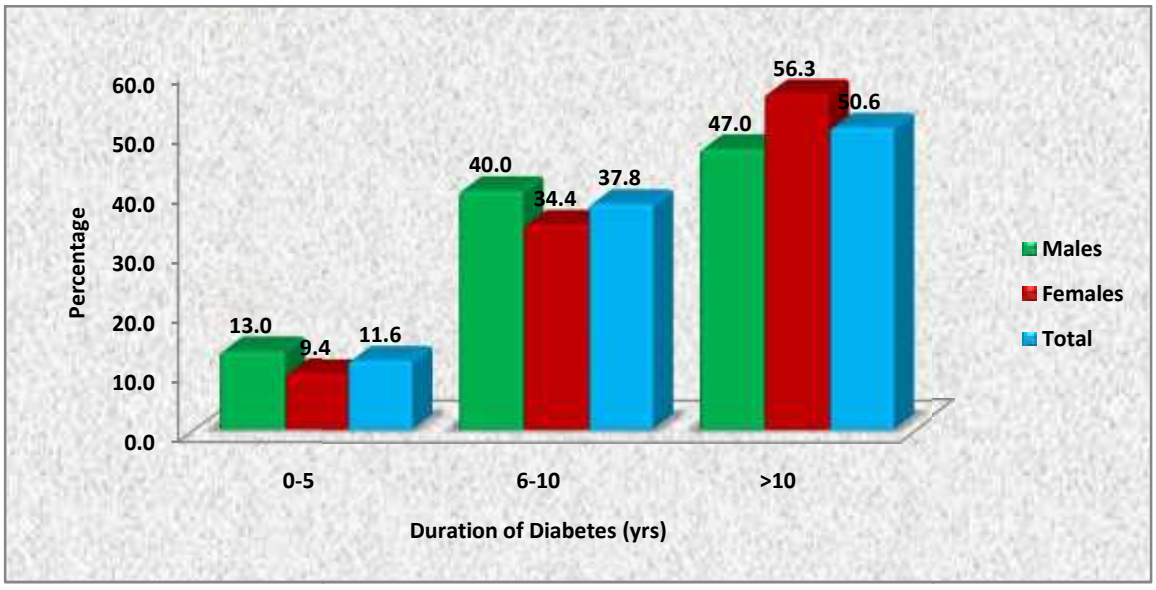


Among both the sexes the age group between 51-60 year had maximum number of patients (40.2%). While between 31-40 years had 3.7%,41-50 had 11.6%, 61-70 had 36.6% and >70 had 7.9%. Sex was not significant different among age groups.

Table No. 15 : Sex Distribution according to Duration of Diabetes

Duration of Diabetes (yrs)	Males		Females		Total		p value
	N	%	N	%	N	%	
0-5	13	13.0	6	9.4	19	11.6	0.489
6-10	40	40.0	22	34.4	62	37.8	
>10	47	47.0	36	56.3	83	50.6	
Total	100	100.0	64	100.0	164	100.0	

Graph No. 3 : Sex Distribution according to Duration of Diabetes

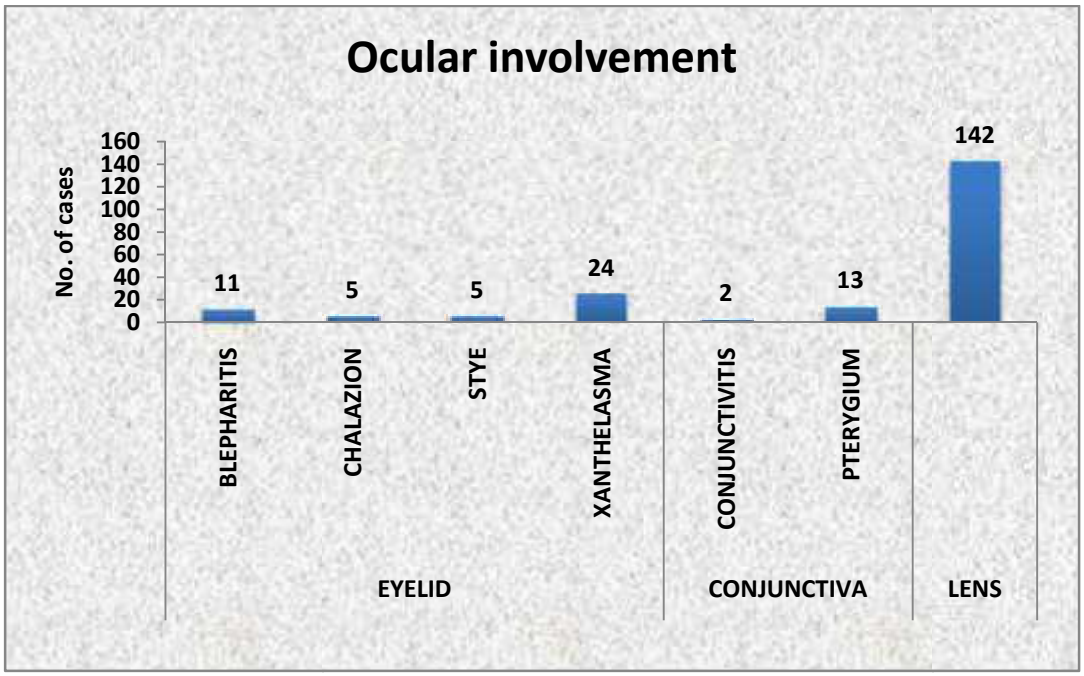


We have accumulated data of duration of diabetes according to the sex distribution . It shows that in both male and female the maximum number of patients had more than 10 years of diabetes (50.6%).Sex was not significant different among duration of diabetes.

Table No. 16 :Ocular involvement among patients

Ocular involvement		N	Percent
EYELID	BLEPHARITIS	11	6.7
	CHALAZION	5	3.0
	STYE	5	3.0
	XANTHELASMA	24	14.6
CONJUNCTIVA	CONJUNCTIVITIS	2	1.2
	PTERYGIUM	13	7.9
LENS		142	86.6
Total		164	100.0

Graph No. 4 :Ocular involvement among patients



We have accumulated data of all the positive ocular involvement, in this study population it shows that most common prevalent ocular involvement is of lens,142 patients (86.6%),45 patients have some eyelid lesions out of which 24 have xanthelasma,11 had blepheritis,5 had stye, and 5 had chalazion.

Table No. 17 :Distribution of severity of diabetic retinopathy and presence of CSME

Grade of Retinopathy	Retinopathy		Retinopathy With CSME	
	N	Percent	N	Percent
Mild NPDR	34	20.7	0	0.0
Moderate NPDR	82	50	4	36.4
Severe NPDR	24	14.6	3	27.3
Very severe NPDR	2	1.2	0	0.0
PDR	22	13.4	4	36.4
Total	164	100	11	100.0

Graph No. 5 : Distribution of severity of diabetic retinopathy

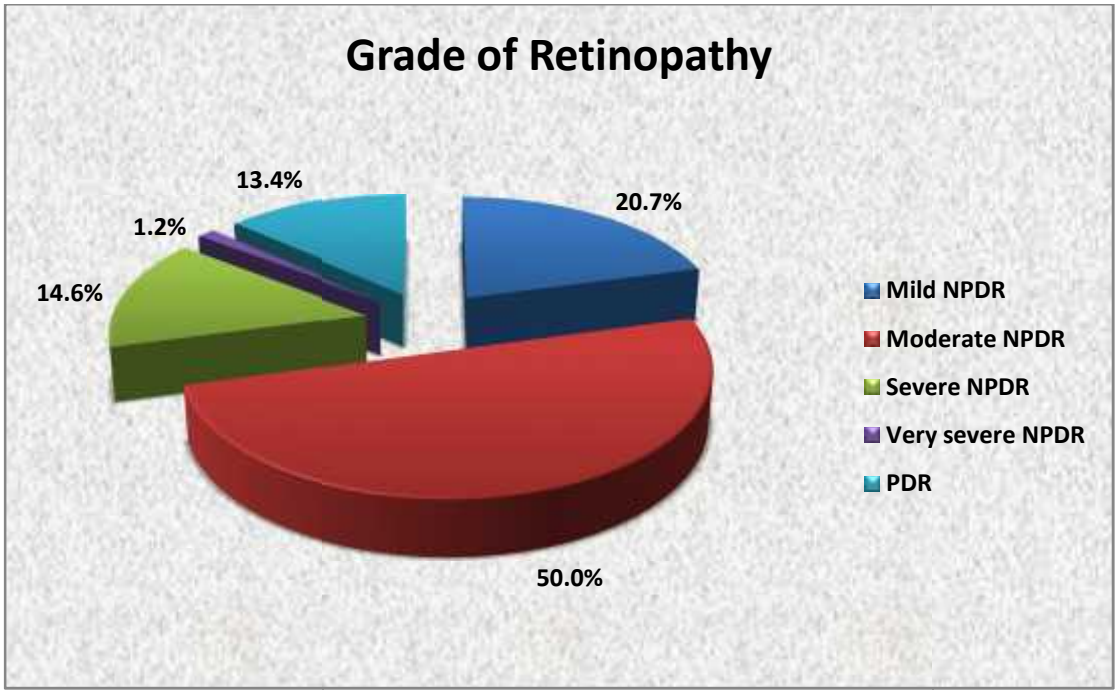
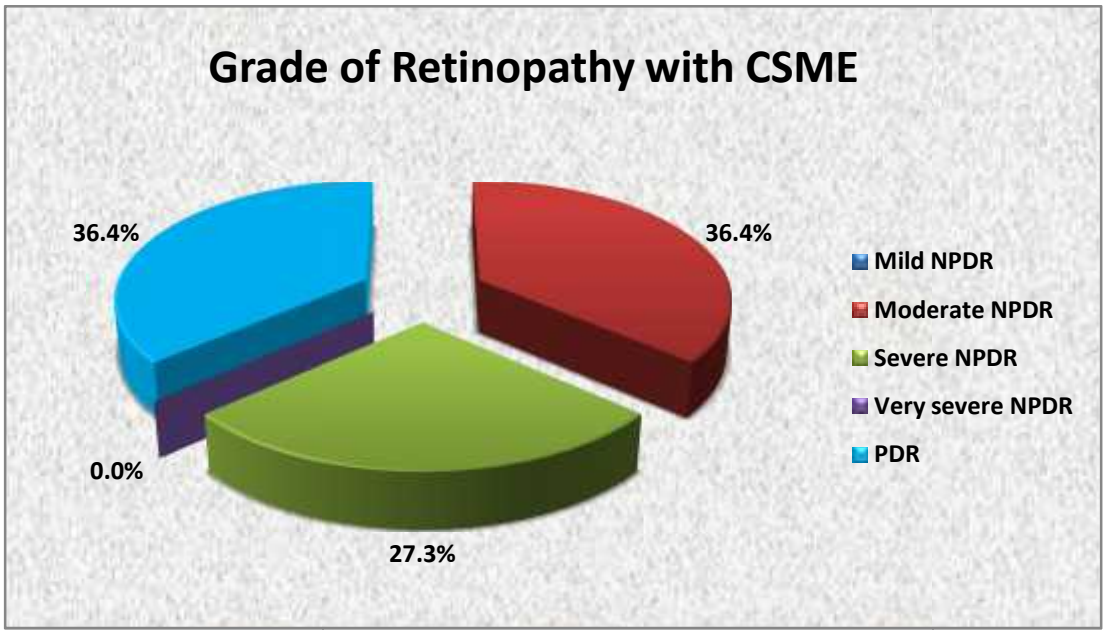


Figure No.6 : Distribution of severity of diabetic retinopathy and presence of CSME

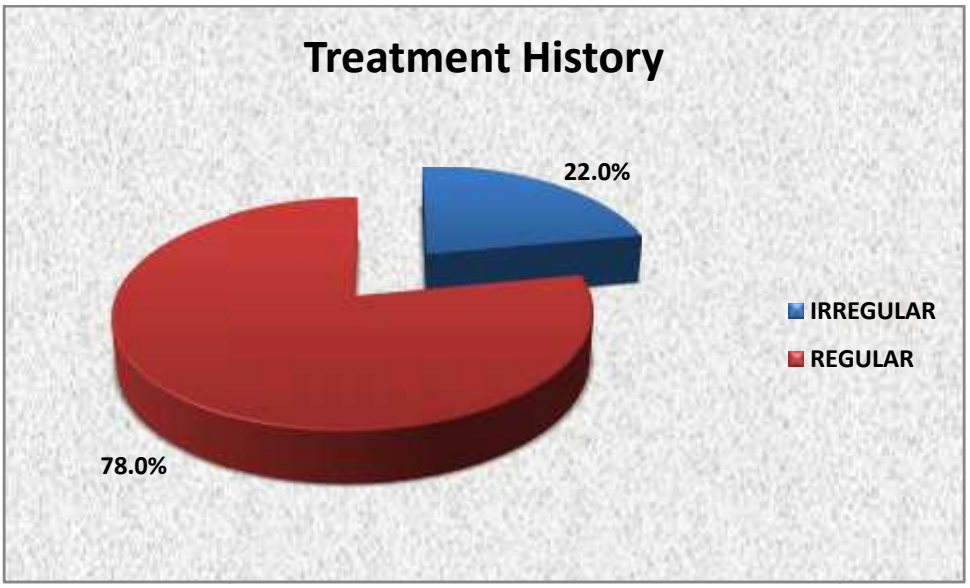


In this study Mild NPDR was present in 34 (20.7%),Moderate NPDR in 82 (50%), Severe NPDR in 24(14.6%),very severe NPDR 2 (1.2%),PDR in 22 (13.4%) of patients. Among these 11 patients had CSME.

Table No.18 :Distribution ofpatients according to Treatment History

Treatment History	N	Percent
IRREGULAR	36	22.0
REGULAR	128	78.0

GraphNo .7 :Distribution ofpatients according to Treatment History



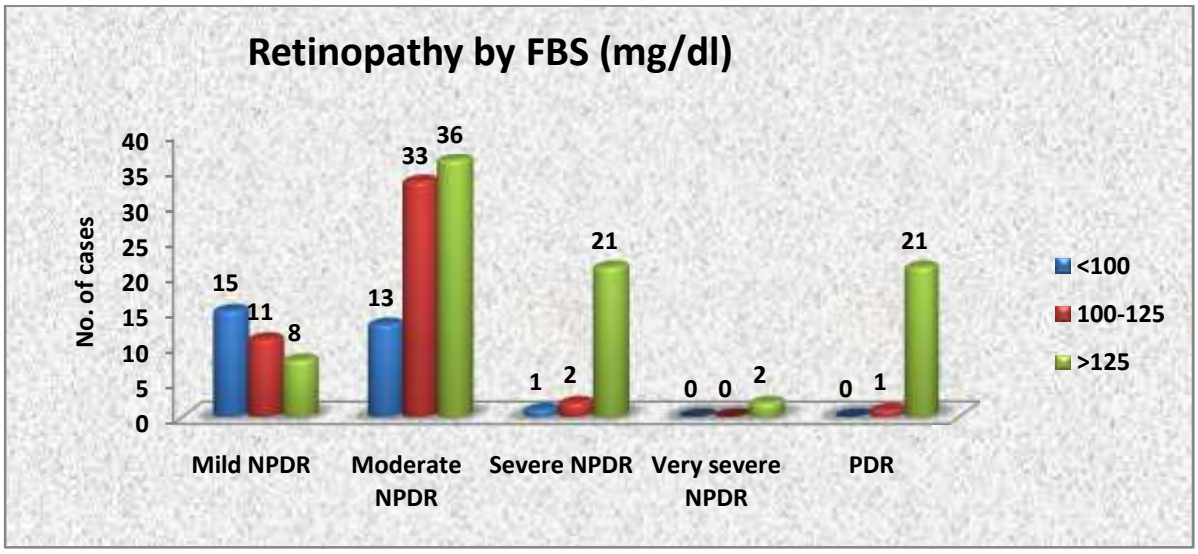
In this study 36 (22%) patients had history of irregular treatment while 128 (78%) had regular treatment.

Table No. 19 :Association of FBS with severity of diabetic retinopathy

FBS (mg/dl)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
<100	15	44.1	13	15.9	1	4.2	0	0.0	0	0.0	<0.001*
100-125	11	32.4	33	40.2	2	8.3	0	0.0	1	4.5	
>125	8	23.5	36	43.9	21	87.5	2	100.0	21	95.5	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No. 8 : Association of FBS with severity of diabetic retinopathy



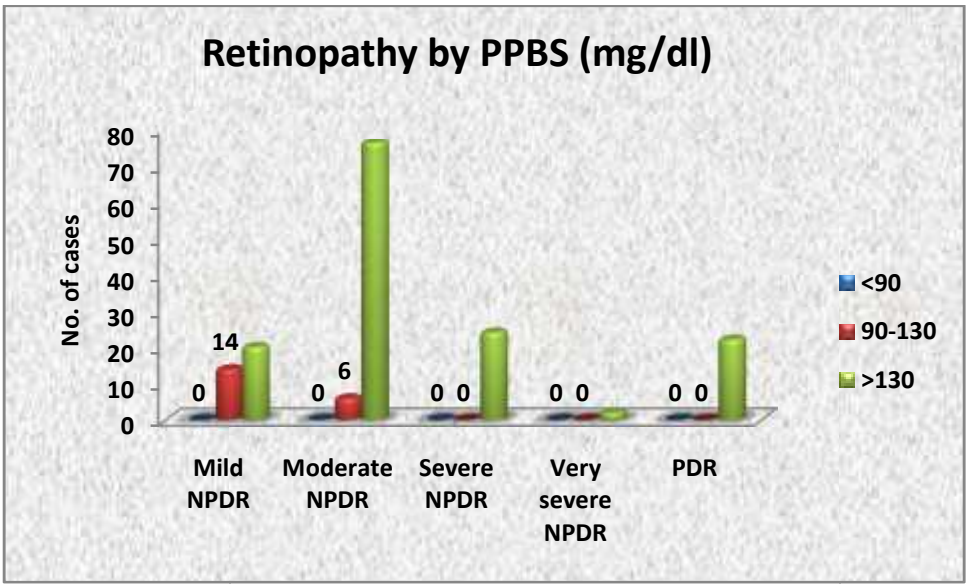
The fasting blood sugar level of patients with different type of retinopathies were examined and found significant with p value of <0.001. The maximum number of Mild NPDR 15 (44.1%) had FBS >100, Moderate NPDR 36 (43.9%) had FBS >125, Severe NPDR 21 (87.5%) had FBS >125 , Very severe NPDR 2 (100%) had >125, and PDR 21 had >125.FBSwas significant different by severity of diabetic retinopathy.

Table No. 20 :Association of PPBS with severity of diabetic retinopathy

PPBS (mg/dl)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
<90	0	0	0	0	0	0.0	0	0	0	0.0	<0.001*
90-130	14	41.2	6	7.3	0	0.0	0	0.0	0	0.0	
>130	20	58.8	76	92.7	24	100.0	2	100.0	22	100.0	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No.9 :Association of PPBS with severity of diabetic retinopathy



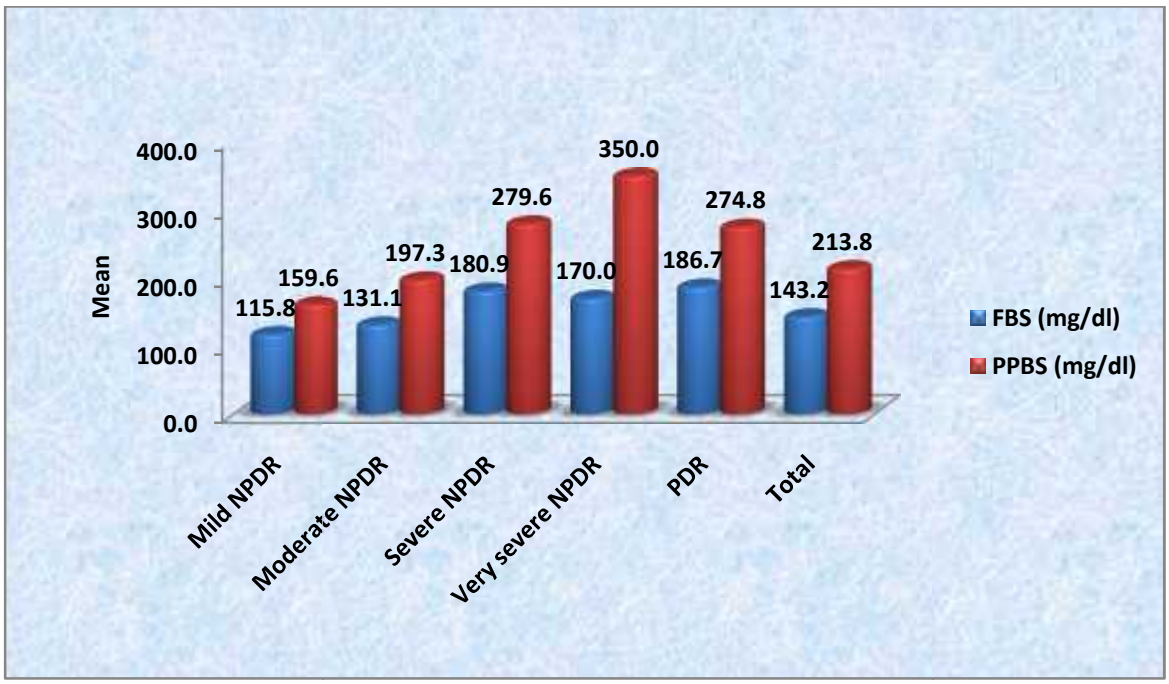
The PPBS value of diabetic retinopathy patients included in the study was examined and found significant with p value of <0.001. Maximum patients with all the different forms of retinopathy had PPBS value >130mg/dl.PPBSwas significant different by severity of diabetic retinopathy.

Table No. 21 :Mean Sugar levels according to severity of diabetic retinopathy

Mean±SD	Mild NPDR	Moderate NPDR	Severe NPDR	Very severe NPDR	PDR	Total	p value
FBS (mg/dl)	115.8±46.7	131.1±48.2	180.9±57.3	170±14.1	186.7±40.2	143.2±54.5	<0.001*
PPBS (mg/dl)	159.6±50.9	197.3±61	279.6±72.6	350±42.4	274.8±59.4	213.8±75.5	<0.001*

Note:*significant at 5% level of significance (p<0.05)

Graph No. 10 : Mean Sugar levels according to severity of diabetic retinopathy

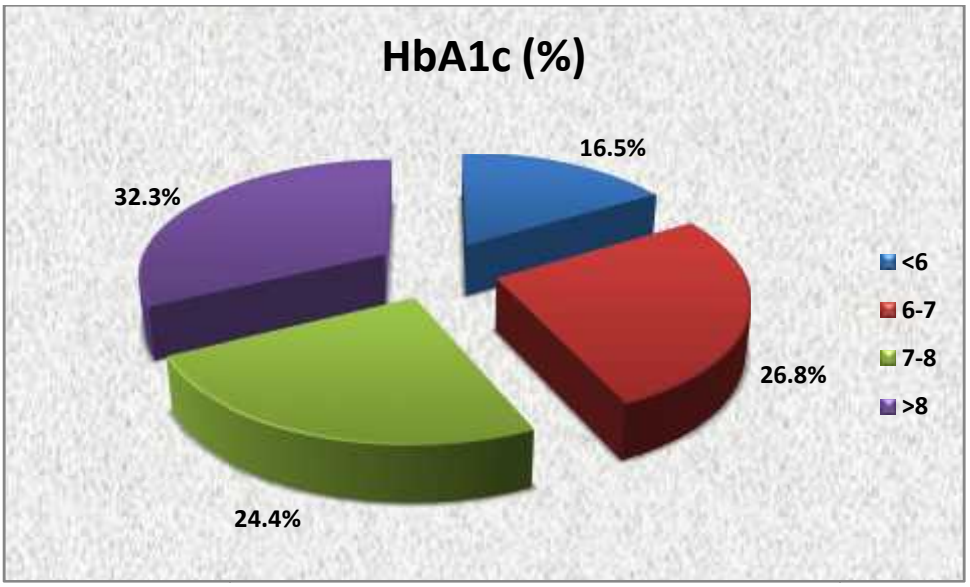


Mean sugar level according to severity of diabetic retinopathy shows a significant relation between the fasting blood sugar level with p= <0.001 and PPBS (p=<0.001).

Table No. 22 :Distribution of patients according to HbA1c

HbA1c (%)	N	Percent
<6	27	16.5
6-7	44	26.8
7-8	40	24.4
>8	53	32.3
Total	164	100

Graph No. 11: Distribution of patients according to HbA1c

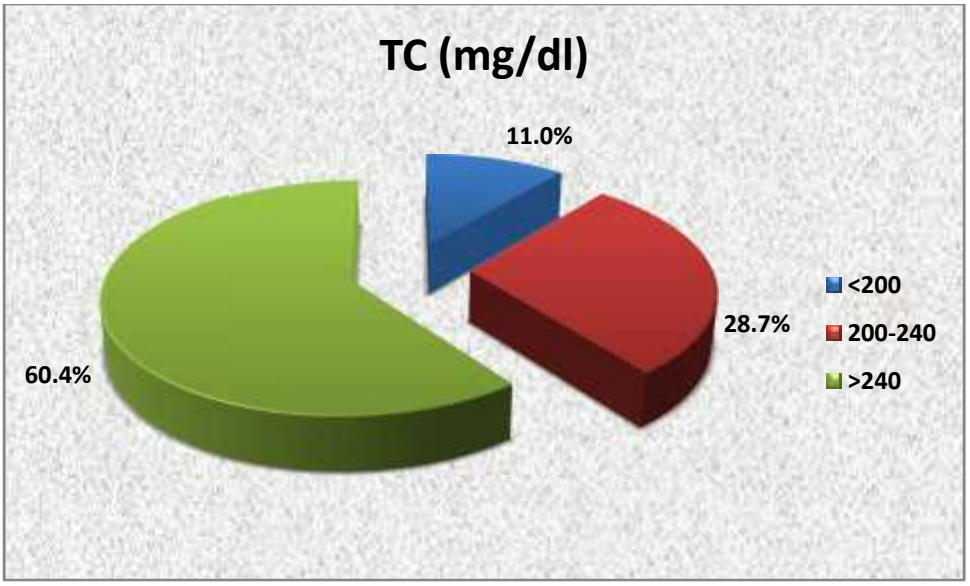


It shows the HbA1c % of patients . According to this study 27 (16.5%) patients has HbA1c value <6, 44 (26.8%) has 6-7, 40 (24.4%) had 7-8,and 53 (32.3%) patients had >8.

Table No. 23 : Distribution of patients according to TC

TC (mg/dl)	N	Percent
<200	18	11
200-240	47	28.7
>240	99	60.4
Total	164	100

Graph No. 12 : Distribution of patients according to TC

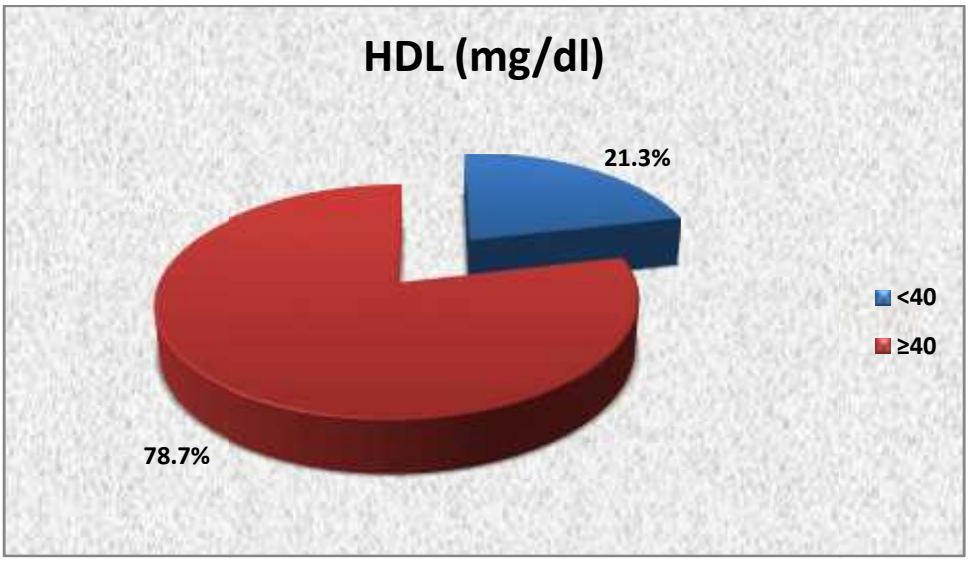


The TC value of patients were recorded which shows 18 patients (11%) had TC value <200mg/dl, 47 (28.7%) had 200-240, 99 (60.4%) had >240.

Table No. 24 :Distribution of patients according to HDL

HDL (mg/dl)	N	Percent
<40	35	21.3
≥40	129	78.7
Total	164	100

Graph No. 13 : Distribution of patients according to HDL

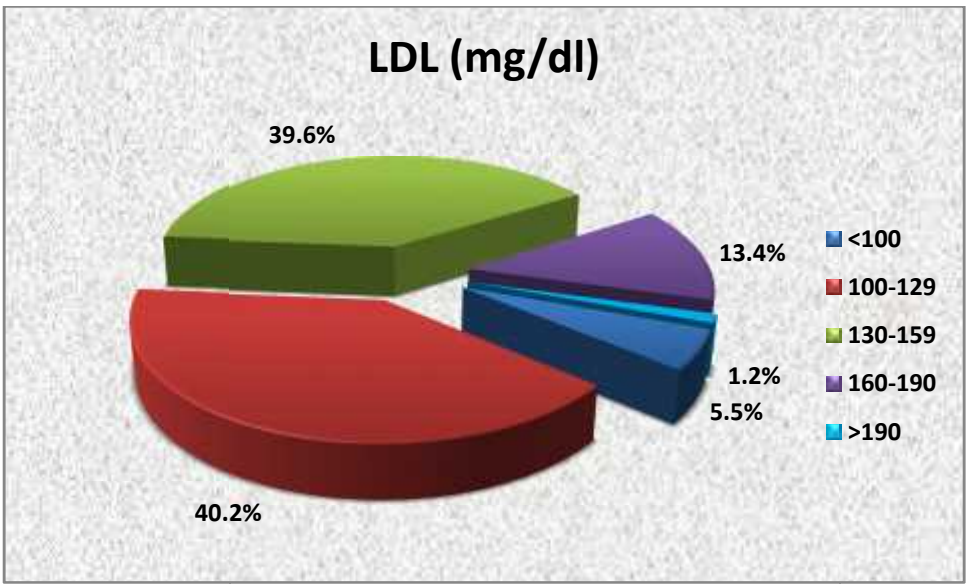


TheHDL value shows that 35 (21.3%) had <40mg/dl, 129 (78.7%) have >40.

Table No. 25 :Distribution of patients according to LDL

LDL (mg/dl)	N	Percent
<100	9	5.5
100-129	66	40.2
130-159	65	39.6
160-190	22	13.4
>190	2	1.2
Total	164	100

Graph No. 14 : Distribution of patients according to LDL

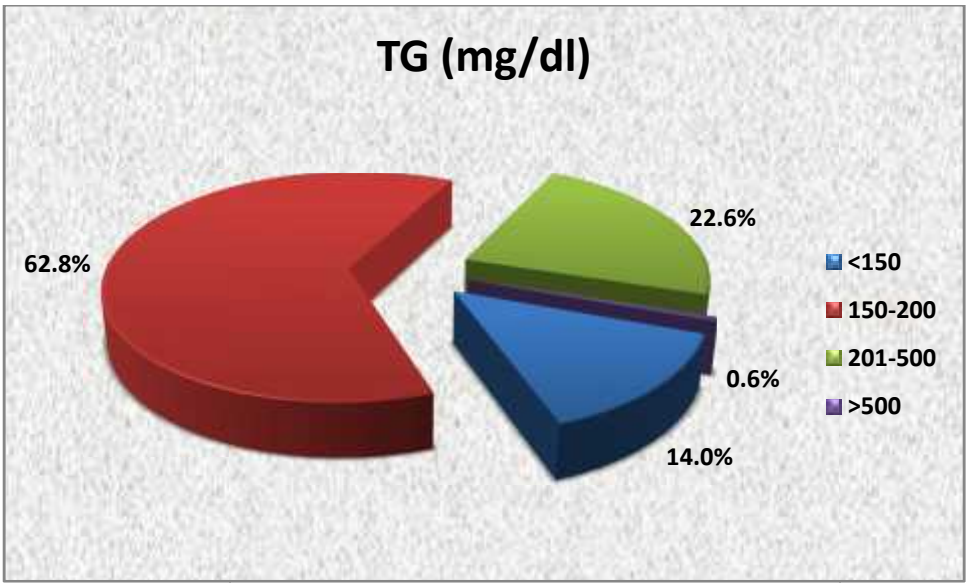


LDL value shows that 9 patients (5.5%) had<100, 66 (40.2%) had 100-129, 65 (39.6%) had 130-159, 22 (13.4%) had 160-190 and 2 (1.2%) had >190.

Table No. 26 : Distribution of patients according to TG

TG (mg/dl)	N	Percent
<150	23	14
150-200	103	62.8
201-500	37	22.6
>500	1	0.6
Total	164	100

Graph No. 15 : Distribution of patients according to TG



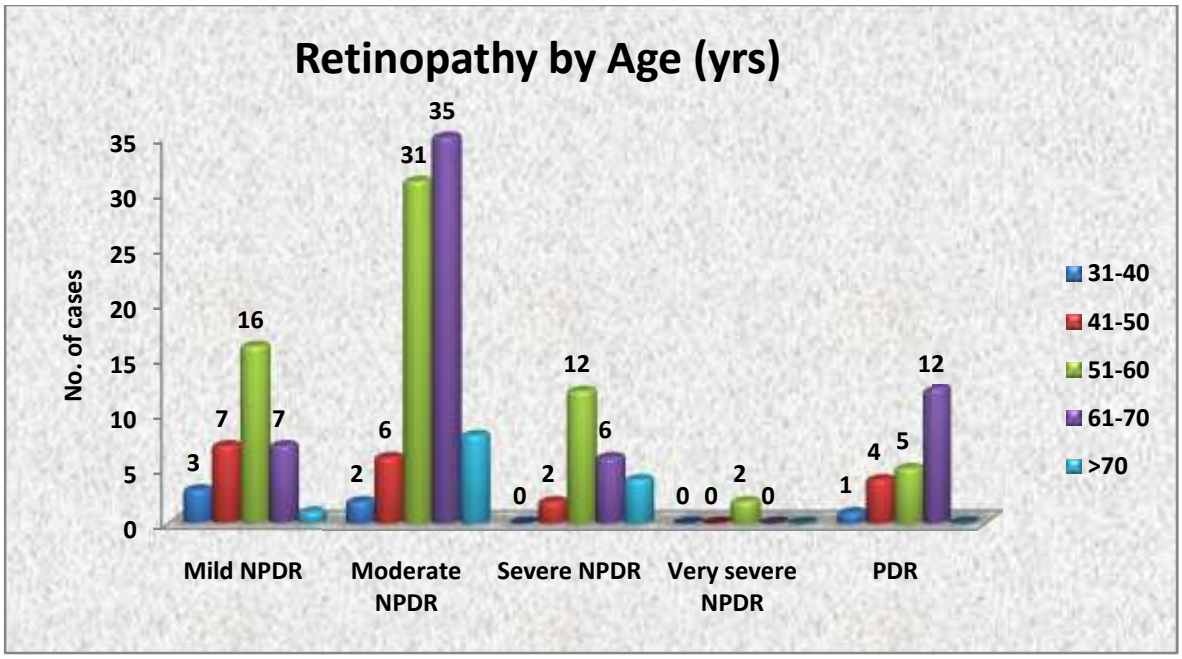
TG record shows that 23 (14%) patients had <150, 103 (62.8%) had 150-200, 37 (22.6%) had 201-500, 1 (0.6%) had >500.

Table No. 27 : Association of age with severity of diabetic retinopathy

Age (yrs)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
31-40	3	8.8	2	2.4	0	0.0	0	0.0	1	4.5	0.049*
41-50	7	20.6	6	7.3	2	8.3	0	0.0	4	18.2	
51-60	16	47.1	31	37.8	12	50.0	2	100.0	5	22.7	
61-70	7	20.6	35	42.7	6	25.0	0	0.0	12	54.5	
>70	1	2.9	8	9.8	4	16.7	0	0.0	0	0.0	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No. 16 : Association of age with severity of diabetic retinopathy



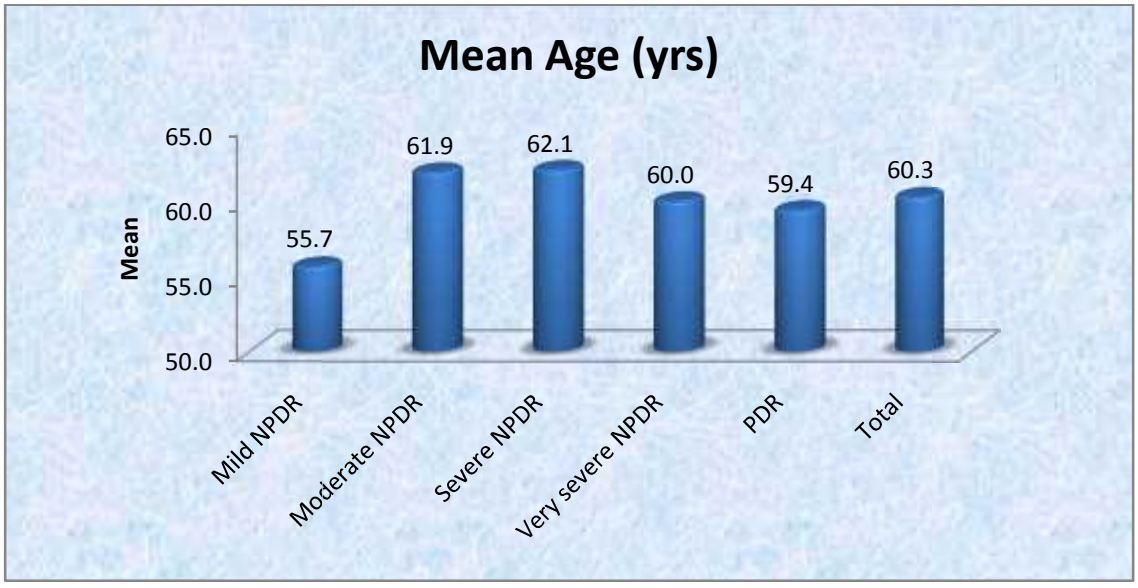
In this study, most of the patients were found to be in the age group of 51-60 years. A significant association was found between age group and diabetic retinopathy (p=0.049), with moderate NPDR most common (82) followed by Mild NPDR.

Table No. 28 :Mean age according to severity of diabetic retinopathy

Mean±SD	Mild NPDR	Moderate NPDR	Severe NPDR	Very severe NPDR	PDR	Total	p value
Age (yrs)	55.7±9.4	61.9±7.9	62.1±7.9	60±0	59.4±8.8	60.3±8.6	0.006*

Note:*significant at 5% level of significance (p<0.05)

Graph No. 17 :Mean age according to severity of diabetic retinopathy

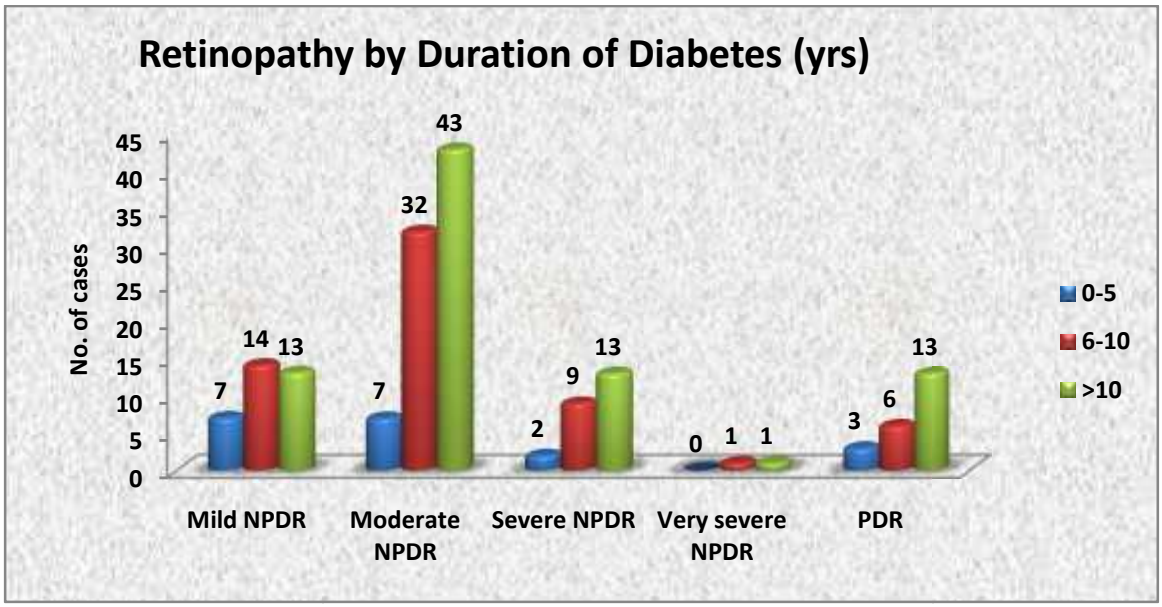


The study that the average age of severity of retinopathy with 55.7±9.4 of Mild NPDR, 61.9±7.9 of Moderate NPDR, 62.1±7.9 of Severe NPDR, 60 of Very severe NPDR and 59.4±8.8 of PDR. The age was significantly different with severity of retinopathy.

Table No. 29 : Association of Duration of Diabetes with severity of diabetic retinopathy

Duration of Diabetes (yrs)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
0-5	7	20.6	7	8.5	2	8.3	0	0.0	3	13.6	0.66
6-10	14	41.2	32	39.0	9	37.5	1	50.0	6	27.3	
>10	13	38.2	43	52.4	13	54.2	1	50.0	13	59.1	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Graph No. 18: Association of Duration of Diabetes with severity of diabetic retinopathy

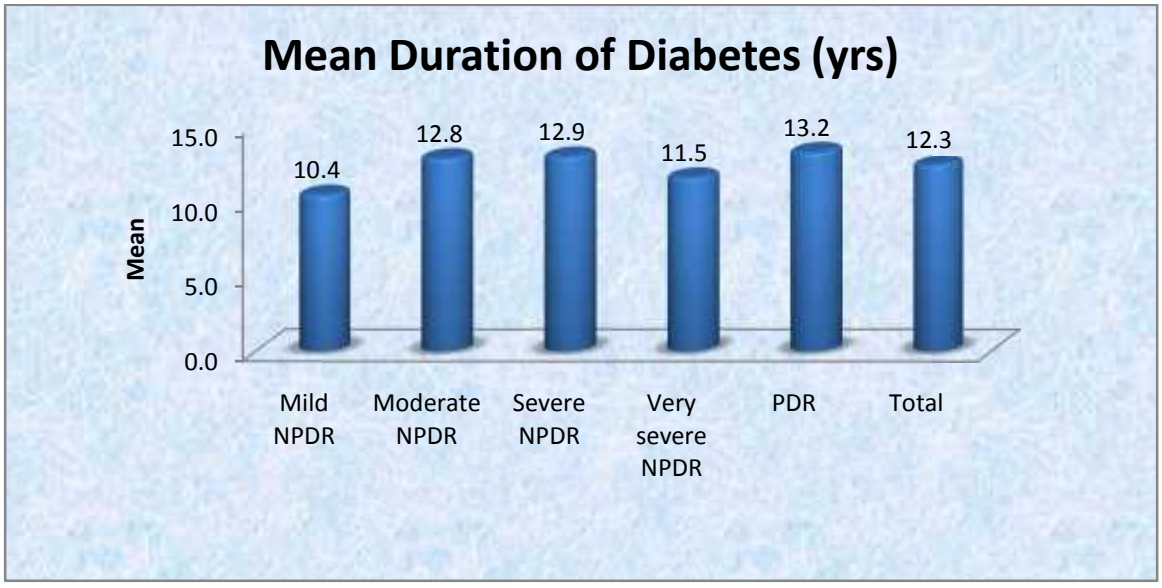


It shows association of duration of diabetes with severity of diabetic retinopathy. According to which the severity of diabetic retinopathy was not significant to duration (p=0.66) and the maximum patients of different retinopathies were above 10 years.The age groups were not significantly distributed with severity of retinopathy.

Table No. 30 : Mean Duration of Diabetes according to severity of diabetic retinopathy

Mean±SD	Mild NPDR	Moderate NPDR	Severe NPDR	Very severe NPDR	PDR	Total	p value
Duration of Diabetes (yrs)	10.4±5.4	12.8±5.6	12.9±4.9	11.5±4.9	13.2±7.2	12.3±5.7	0.262

Graph No.19 :Mean Duration of Diabetes according to severity of diabetic retinopathy



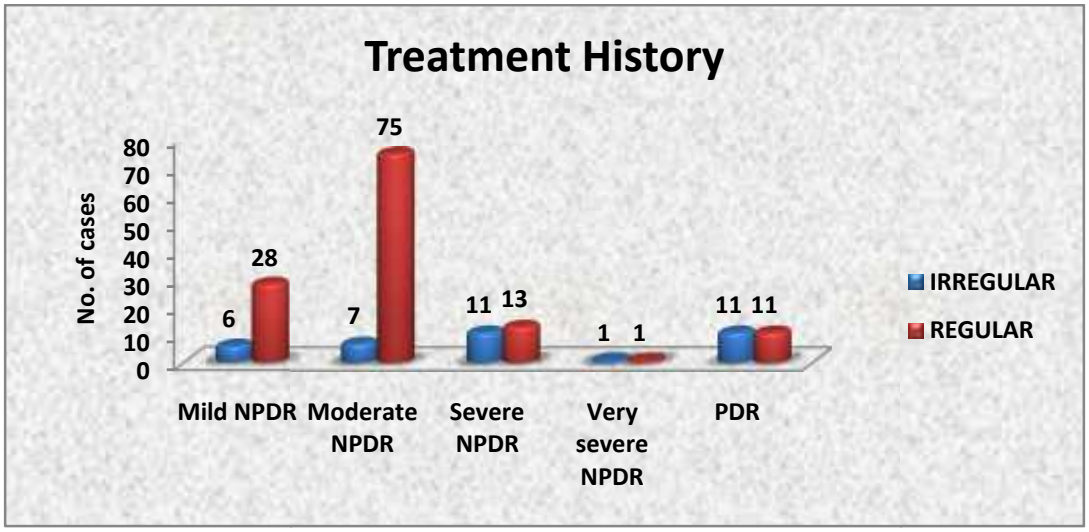
This shows mean duration of diabetes according to severity of diabetic retinopathies with value p=0.262.The duration was not significantly different with severity of retinopathy.

Table No. 31 : Association of Treatment History with severity of diabetic retinopathy

Treatment History	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
IRREGULAR	6	17.6	7	8.5	11	45.8	1	50.0	11	50.0	
REGULAR	28	82.4	75	91.5	13	54.2	1	50.0	11	50.0	<0.001*
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No. 20 : Association of Treatment History with severity of diabetic retinopathy



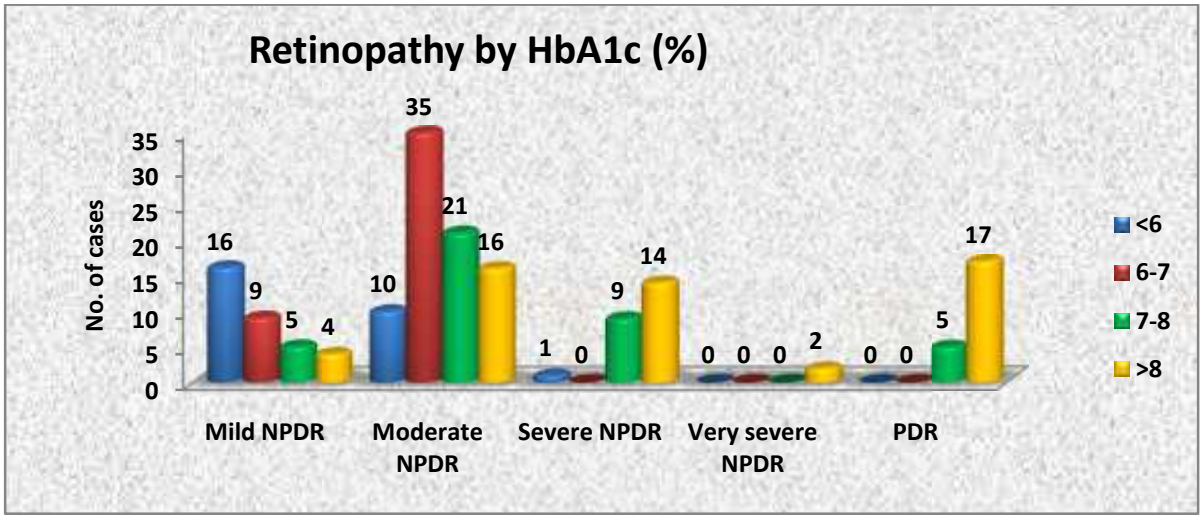
Association of treatment history with severity of diabetic retinopathy is significant (p=0.001) with most patients of different severity of diabetic retinopathies had regular treatment.

Table No. 32 : Association of HbA1c with severity of diabetic retinopathy

HbA1c (%)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
<6	16	47.1	10	12.2	1	4.2	0	0.0	0	0.0	<0.001*
6-7	9	26.5	35	42.7	0	0.0	0	0.0	0	0.0	
7-8	5	14.7	21	25.6	9	37.5	0	0.0	5	22.7	
>8	4	11.8	16	19.5	14	58.3	2	100.0	17	77.3	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No . 21 : Association of HbA1c with severity of diabetic retinopathy



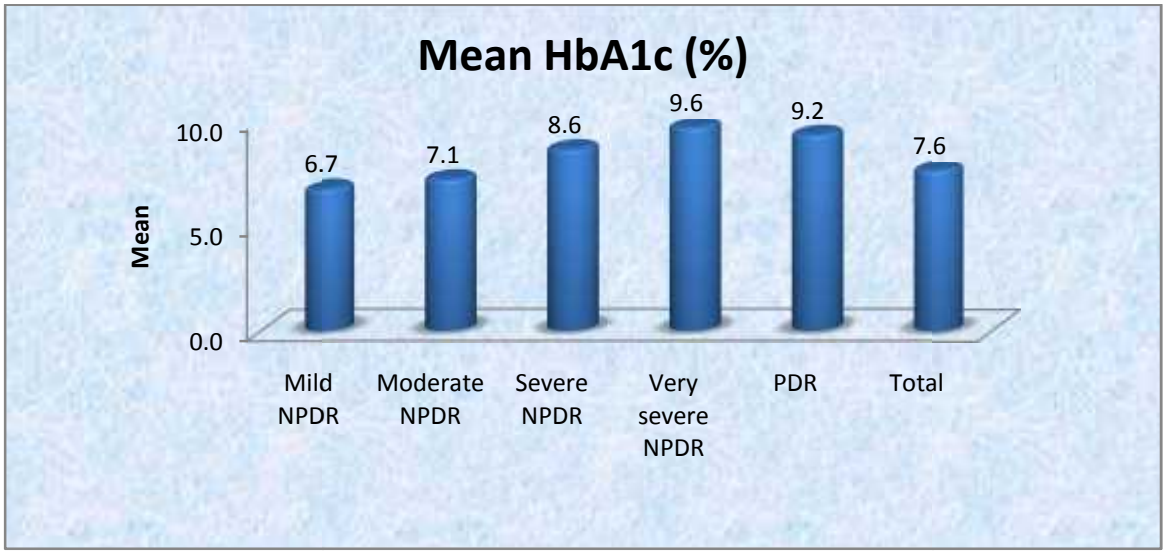
HbA1c has been known as a marker of glycemic control. We used this serum marker to identify the relation between the level of uncontrolled hyperglycemia with prevalence of different retinopathies. The study yielded a highly significant result (P=<0.001). Our study found that with increasing HbA1c levels, prevalence of retinopathies increases. From above table it is clear that Mild NPDR (47.1%) is seen at low levels of HbA1c (<6%), Moderate NPDR (42.7%) is most prevalent between 6-7% of HbA1c and Severe NPDR(58.3%) is most common at >8% levels while PDR at >8% levels.

Table No. 33 : Mean HbA1c according to severity of diabetic retinopathy

Mean±SD	Mild NPDR	Moderate NPDR	Severe NPDR	Very severe NPDR	PDR	Total	p value
HbA1c (%)	6.7±1.7	7.1±1.1	8.6±1.6	9.6±0.8	9.2±1.6	7.6±1.6	<0.001*

Note:*significant at 5% level of significance (p<0.05)

Graph No. 22 :Mean HbA1c according to severity of diabetic retinopathy



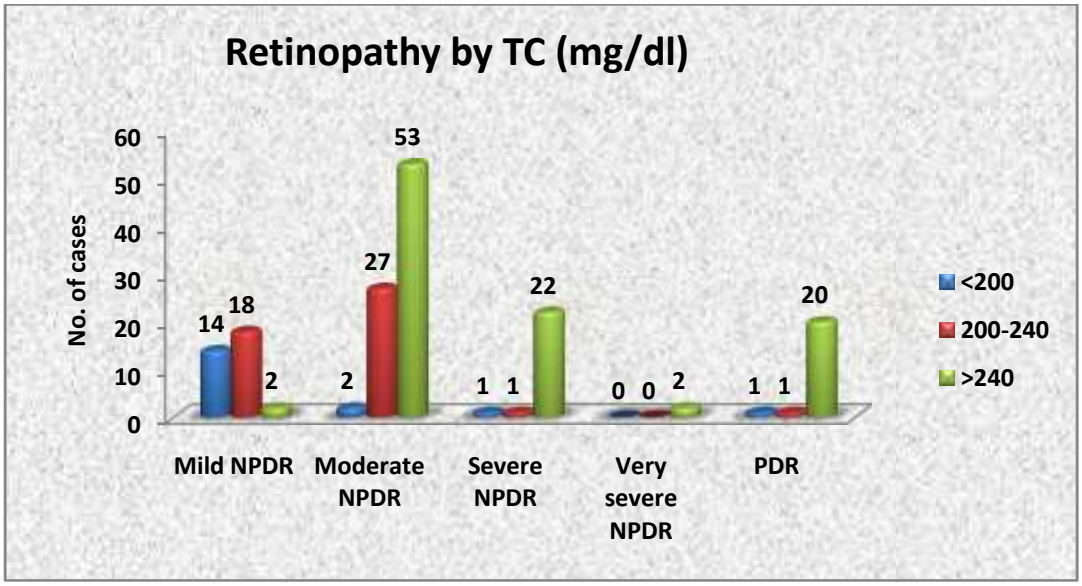
The mean value of HbA1c according to severity of diabetic retinopathy is significant with a value of p= <0.001.

Table No. 34 :Association of TC with severity of diabetic retinopathy

TC (mg/dl)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
<200	14	41.2	2	2.4	1	4.2	0	0.0	1	4.5	<0.001*
200-240	18	52.9	27	32.9	1	4.2	0	0.0	1	4.5	
>240	2	5.9	53	64.6	22	91.7	2	100.0	20	90.9	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No. 23 : Association of TC with severity of diabetic retinopathy

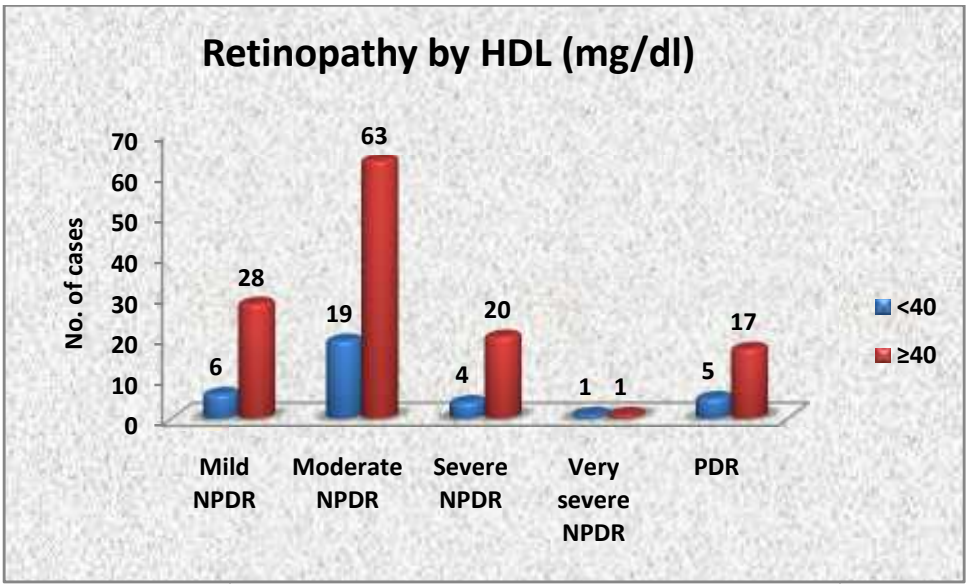


The present study showed statistically significant correlation between diabetic retinopathy and raised total cholesterol level (p=<0.001). The table shows that Mild NPDR patients were maximum (52.9%) for Tc 200-240, Moderate NPDR (64.6%) at TC >240, Severe NPDR (91.7%) at TC >240,Very Severe NPDR (100%) at TC >240 and PDR (90.9%) at TC >240.

Table No. 35 : Association of HDL with severity of diabetic retinopathy

HDL (mg/dl)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
<40	6	17.6	19	23.2	4	16.7	1	50.0	5	22.7	0.781
≥40	28	82.4	63	76.8	20	83.3	1	50.0	17	77.3	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Graph No24 :Association of HDL with severity of diabetic retinopathy



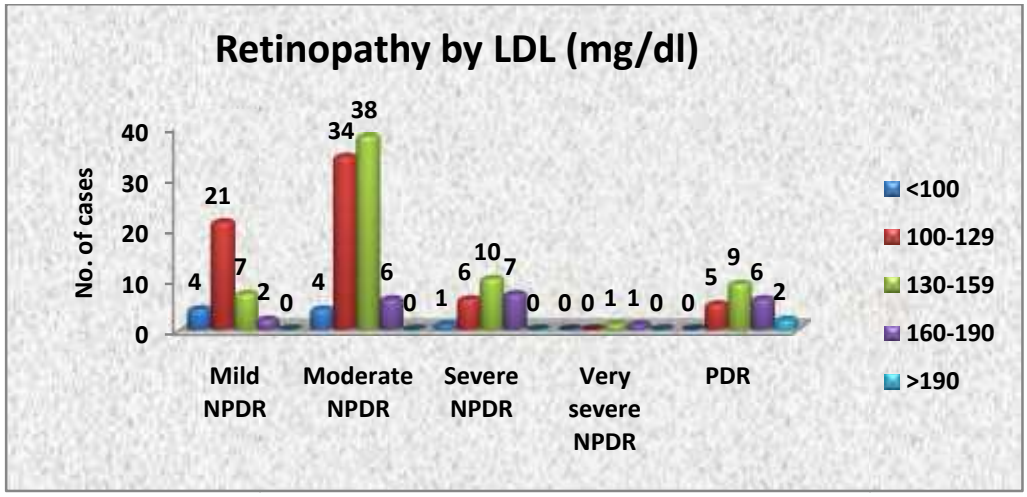
While comparing the association of different retinopathies with HDL (mg/dl) it was found that all thee retinopathies were maximum for HDL value of > 40 mg/dl with Mild NPDR 82.4%, Moderate NPDR 76.8%, Severe NPDR 83.3%, Very severe NPDR 50% and PDR 77.3%.HDL was not significantly different with severity of retinopathy.

Table No. 36 : Association of LDL with severity of diabetic retinopathy

LDL (mg/dl)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
<100	4	11.8	4	4.9	1	4.2	0	0.0	0	0.0	<0.001*
100-129	21	61.8	34	41.5	6	25.0	0	0.0	5	22.7	
130-159	7	20.6	38	46.3	10	41.7	1	50.0	9	40.9	
160-190	2	5.9	6	7.3	7	29.2	1	50.0	6	27.3	
>190	0	0.0	0	0.0	0	0.0	0	0.0	2	9.1	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No. 25 : Association of LDL with severity of diabetic retinopathy



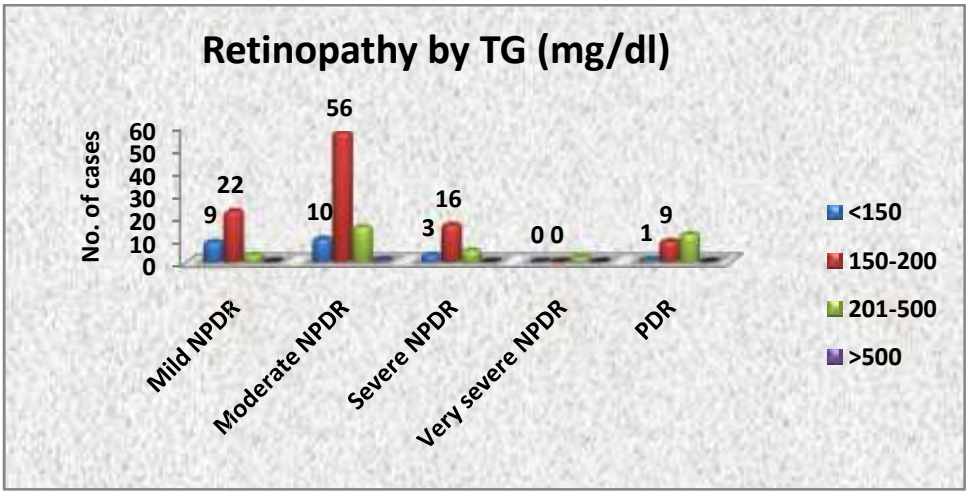
The association of LDL level and different retinopathies was found significant (<0.001) with maximum number of Mild NPDR 21 (61.8%) at 100-129 value of LDL and others retinopathies, Moderate , Severe , Very severe and PDR at 130-159.

Table No. 37 : Association of TG with severity of diabetic retinopathy

TG (mg/dl)	Mild NPDR		Moderate NPDR		Severe NPDR		Very severe NPDR		PDR		p value
	N	%	N	%	N	%	N	%	N	%	
<150	9	26.5	10	12.2	3	12.5	0	0.0	1	4.5	0.004*
150-200	22	64.7	56	68.3	16	66.7	0	0.0	9	40.9	
201-500	3	8.8	15	18.3	5	20.8	2	100.0	12	54.5	
>500	0	0.0	1	1.2	0	0.0	0	0.0	0	0.0	
Total	34	100.0	82	100.0	24	100.0	2	100.0	22	100.0	

Note:*significant at 5% level of significance (p<0.05)

Graph No. 26 : Association of TG with severity of diabetic retinopathy



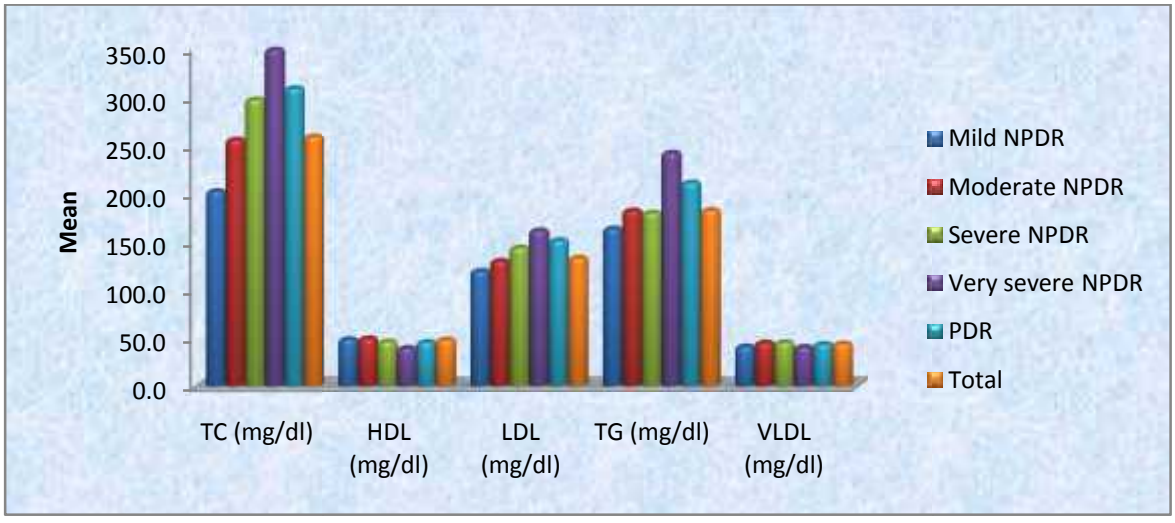
The triglyceride level where determined in different type of retinopathies which showed a significant correlation (p=0.004). All forms of retinopathies where found at high triglyceride level, with Mild NPDR max (64.7%) at 150-200, Moderate NPDR (68.3%) at 150-200, Severe NPDR (66.7%) at 150-200, and PDR (54.5%) at >500 value of TG. While comparing the association of levels of different lipid profiles of patients of different type of retinopathies a clear significant correlation was found between TC, LDL and TG (0.005).

Table No. 38 : Association of Mean Lipid profiles with severity of diabetic retinopathy

Mean±SD	Mild NPDR	Moderate NPDR	Severe NPDR	Very severe NPDR	PDR	Total	p value
TC (mg/dl)	201.2±30.8	254.4±30	295.9±46	347.5±10.6	307.7±41.1	257.7±49.8	<0.001*
HDL (mg/dl)	47.9±11.1	48.8±20	45.5±10.2	39±1.4	45±10.2	47.5±16	0.73
LDL (mg/dl)	118.3±19.6	128.6±19.1	142.4±25.4	160±28.3	149.9±28.9	131.7±24	<0.001*
TG (mg/dl)	161.9±25.6	180.5±58.2	178.2±36.4	240±28.3	209.4±49.4	180.9±50.5	0.005*
VLDL (mg/dl)	40.5±11.1	44.3±14.9	44.6±24.7	40±28.3	43±15.8	43.4±16.1	0.816

Note:*significant at 5% level of significance (p<0.05)

Graph No. 27 : Association of Mean Lipid profiles with severity of diabetic retinopathy



While comparing the association of levels of different lipid profiles of patients of different type of diabetic retinopathies, a clear significant correlation was found between TC (p=0.001), LDL (p=<0.001) and TG (p=0.005).

DISCUSSION

It is believed that the Indian population generally has an unusually efficient glucose metabolism. But with westernisation and the associated weight increase and sedentary lifestyle, the former advantage is lost and incidence of diabetes has increased. Paralleling this high prevalence of diabetes is a concern that the complications of diabetes, mainly diabetic retinopathy is increasing.[63]

Hyperglycemia and dyslipidemia are two major metabolic disorders seen in patients with diabetes mellitus. The role of diabetic dyslipidemia in the development of microvascular complications has received much less attention.[64]This study aimed to determine the relationship between plasma lipid profile and the severity of diabetic retinopathy in type 2 diabetes patients.

The present study had 164 patients with male predominance of 100 males and 64 females. The male to female ratio [M: F] was 1.5:1. In a clinical cohort in Chennai Diabetic retinopathy appeared to be prevalent more in the males compared to females (sex ratio 2:1).[65]Similar male preponderance was also seen in the CURES Eye study,[66]UKPDS study [67]Gupta et al[68]and the Andhra Pradesh Eye Disease study (APEDS).[69]

Most patients were in the age group of 51-60 years.The relationship of retinopathy with age was in concordance to that found in many other studies. Like several other epidemiologic studies, this study also showed an increased prevalence of DR with increasing age. Dondanaet al[5]CURES Eye Study [66]and APED Study [69]also have found significant correlation between the patient age and diabetic retinopathy.

In the present study, the duration since diagnosis of diabetes (diabetic age) ranged from 5 - 25 years.The study population had maximum patients with diabetic of duration more than 10 years. There may be some bias in estimating the real duration of diabetes in these patients, as the discovery of diabetes could have been delayed due to lack of symptoms and the insidious onset of type 2 diabetes. While recording the different type of retinopathies, 34 (20.7%) had Mild NPDR,82(50%) Moderate NPDR, 24(14.6%)

Severe NPDR,2(1.2%) Very severe NPDR and 22(13.4%)PDR. The mean age of different retinopathies being 60.3+8.6.

The present study showed statistically significant correlation between diabetic retinopathy and raised total cholesterol level(p=<0.001) and low density lipoprotein(p=<0.001). the correlation of diabetic retinopathy and TG was also significant(p=0.004).Rema et al (CURES eye study)[66]and Haddad et al[70] alsofound that both serum triglyceride (p= 0.001) levels and total cholesterol (P= 0.014) were higher in patients with diabetic retinopathy as compared to those without diabetic retinopathy. While Gupta et al[71]demonstrated that diabetics with raised LDL levels showed higher prevalence of Diabetic retinopathy (38%) compared to others (28.3%) (p=0.05). In contast to this study, Al-Bdouret al[8]and Larsson et al[71] also found significant correlation between higher levels of serum total cholesterol and retinopathy.

The drawbacks of the study are that the fundus photograph, which is the standard pattern of recording of fundus changes were not taken for all patients. In such conditions it is more common to underestimate than to overestimate fundus changes related to diabetic retinopathy. In the present study CSME was diagnosed by Slit lamp biomicroscopy with 78D/ 90D lens and indirect ophthalmoscope. Because of the non-availability, the newer, the more sensitive method of assessing retinal thickening such as with optical coherence tomography were. The study did not evaluate other risk factors for the development of retinopathy like anemia. Also, the referral of uncontrolled diabetics to the tertiary centre would have allowed the possibility of selection bias to creep into the study.

CONCLUSION

The present study demonstrated statistically significant correlation between diabetic retinopathy and hypercholesterolemia.

Increased cholesterol level was significantly associated with the occurrence of all grades of retinopathy especially severe NPDR, very severe NPDR and PDR.

- Increased significant total cholesterol levels in patients with different type of retinopathies.
- Increased significant low density lipoprotein in different severities of diabetic retinopathies.
- Increased significant triglyceride levels in patients with different retinopathies.

It also showed that hypercholesterolemia is significantly associated with CSME. The current treatment for diabetic retinopathy is laser photocoagulation. With the advent of systemic lipid lowering therapy over the last decade, there may be potential for medical therapy also. There is some anecdotal evidence of the effect of lipid lowering agents in reducing hard exudates.[10]Further studies are required to establish the causal relationship between dyslipidemia and diabetic retinopathy. If established, these data can lend additional support to current treatment guidelines recommending aggressive lowering of elevated lipids among diabetic patients. Rigorous lipid control, in addition to its known health benefits in preventing cardiovascular disease, may also lessen ocular morbidity and associated health care costs, thereby potentially improving quality of life and vision among people with type 2 diabetes.

SUMMARY

Diabetic retinopathy is a very common poteintially preventable, long term, microvascular complication of diabetes mellitus and a leading cause of visual disability and blindness. In India the prevalence of diabetic retinopathy in general population is 3.5% and the prevalence of diabetic retinopathy in the population with NIDDM was 18%. While there are multiple risk factors which have been associated with development and progression of retinopathy. Elevated serum lipid levels are associated with an increased risk of retinal hard exudates in retinopathy and play an important role in pathogenesis of retinopathy.

This was a prospective observational study on 164 type 2 diabetes patients. Collected data of each patient included detailed diabetes history, ophthalmological examination, serum lipid profile. The data gathered was tabulated into master chart and statistical analysis done for relevant tables.

Significant results included:

1. Different types of diabetic retinopathies are correlated with the raised level of total cholesterol(p=0.001).
2. Types of retinopathy and its severity is related to the increase in triglyceride value(p=0.004).
3. Diabetic retinopathy patients had raise LDL(p=<0.001).

   Appropriate treatment of diabetes and optimal control of dyslipidemia is the prevention of different type of diabetic retinopathies. At the same time, following the various guidelines to screen and detect these complication will make a difference in the final outcome of patients.

BIBLIOGRAPHY

1. American Diabetes Association Standards of medical care in Diabetes Mellitus. Diabetes Care 2015 Jan; volume 38 (1) :S8-S86.
2. Pradeepa R, Deepa R, Mohan V Epidemiology of Diabetes in India-current perspective and future projection . J Indian Med Assoc. 2002 Mar; 100(3):144-8.
3. Kalantzis.G, Angelou.M, Rebelakou E.F, Diabetic Retinopathy: An historical assessment. Hormones 2006, 5(1):71-74.
4. Miljanovic B, Glynn RJ, David M, Nathan, Joann E, Manson, Debra A et al. Prospective Study of Serum Lipids and Risk of Diabetic Macular Edema in Type1 Diabetes. Diabetes 2003 Nov; 53: 2883 - 2902.
5. Dandona R, Dandona L, Naduvilath TJ, McCarty A, Rao GN. Population based assessment of diabetic retinopathy in an urban population in southern India. Br. J. Ophthalmol. 1999; 83: 937- 940.
6. Sharma RA. Diabetic eye disease in southern India. J Comm Eye Health 1996; 09(20): 56-58.
7. Stephen Ryan. RETINA. 4th ed. Vol. II. Chapter 66. In: Etiologic mechanisms in Diabetic retinopathy, Robert N. Frank, eds. Philadelphia PA: Elsevier; 2006: 1241 – 1265.
8. Muawyah D Al-Bdour, Maha I Al-Till, Khawla M Abu Samra. Risk factors for diabetic retinopathy among Jordanian diabetics. Middle East African Journal of Ophthalmology 2008; 15: 77-80.
9. Hamilton AMP, Ulbig MW, Polkinghorne P. Management of diabetic retinopathy. BMJ Publishing group, London, 1996.
10. Chowdhury TA, Hopkins D, Dodson PM, Vafidis DF. The role of serum lipids in exudative diabetic maculopathy: is there a place for lipid lowering therapy? Eye 2002 (16): 689-693.

11. Reanita, Bardosono S, Victor AA. Relationship between plasma lipid profile and the severity of diabetic retinopathy in type 2 diabetes patients. Med J Indones 2008; 17: 221-225.

12. D H W Su, K T Yeo. Diabetic Retinopathy and Serum Lipids. Singapore Med J 2000; 41(6): 295-297.

13. Cetin EN,BulguY,OzdermirS,TopsakalS,AkinF,AybekH,et al. Association of serum lipid level with diabetic retinopathy. Int J Ophthalmol.2013;6(3):346-9.

14. Chew EY, Klein ML, Ferris FL 3rd, Remaley NA, Murphy RP, Chantry K et al. Association of elevated serum lipid levels with retinal hard exudate in diabetic retinopathy. Early Treatment Diabetic Retinopathy Study (ETDRS) Report 22. Arch Ophthalmol1996;114:1079-84.

15. Gupta S, Ambade A. Prevalence of Diabetic Retinopathy and Influencing Factors amongst type 2 Diabetics from Central India.International Journal of Diabetes in Developing Countries 2004;24:75-78.

16. MuawyahDA ,Maha IA , Khawla M A S .Risk factors for diabetic retinopathy among Jordanian diabetics. Middle East African Journal of Ophthalmology 2008 ;15:77-80.

17. Uçgun NI, Yildirim Z, Kiliç N, Gürsel E. The importance of serum lipids in exudative diabetic macular edema in type 2 diabetic patients. Annals of the New York Academy of Sciences 2007;1100:213–217.

18. Sharma PK and ChandakP.Brief Communication of Serum lipid profile in diabetics with and without retinopathy. Indian Journal of Clinical Biochemistry 1992;7:203-204.

19. Mathew DD, Barbara A.B. Proliferative Diabetic Retinopathy in Stephan.J.Ryan(ed).In, Retina, 4th edition. Philadelphia, Elsevier Mosby 2006;1301.

20. Iiechie AA, AdinorteyMB,Latey S, Amponsah I, Boadi-kusi SB, Kyei S, et al.Is there any association between serum lipids and diabetic retinopathy in type 2 diabetic patients in Ghana .Int J Tropical disease and health.02/2014;4(4).

21. Longo, Fauci, Kasper, Hauser, Jameson, Loscalzo. HARRISONSPRINCIPLES OF INTERNAL MEDICINE, 18TH EDITION, 2969.

22. Melmed S, Polonnsky KS, Larsen PR, .Kronenberg HM. Williams Textbookof ENDOCRINOLOGY, 12TH EDITION: 1371.

23. Rema M, Deepa R, Mohan V. Prevalence of retinopathy at diagnosis amongType 2 diabetic patients attending a diabetic centre in S India . Br J Ophthal 2000;84:1058-60.

24. Duke elder S, Dobree JH. Systems of classification of diabetic retinopathy.System of ophthalmology 1967; 10:414.

25. Ballantyne AJ, Michaelson. Classification of diabetic retinopathy. TransOphthalmolSoc UK 1947; 67:54.

26. Scott DJ, Dollery, Hill A. Classification of diabetic retinopathy. Br J Ophthalmol 1963; 47:588.

27. Alaerts, Slosse. Classification of diabetic retinopathy. Bull SocBelgeOphthalmol 1957; 115:160.

28. Lee, McMeel, Schepens. Classification of diabetic retinopathy. Am J ofOphthalmol 1966; 62:207.

29. Peymann GA, Saunders DR, Goldberg MF. Diabetic retinopathy. Principles and Practice of Ophthalmology. 1987; 2:1205.

30. Kanski JJ. Clinical Ophthalmology- A Systemic Approach. Butterwor-Heinmann 1989;302.

31. Early Treatment Diabetic Retinopathy Study Group. ETDRS Report 1991.Fundusphotographic risk factors for progression of diabetic retinopathy.Ophthalmology 1991; 98:823-33.

32. Early treatment Diabetic Retinopathy Study Group. ETDRS Report. Gradingdiabetic retinopathy from stereoscopic color fundus photographs-an extension ofmodified Arlie House classification. Ophthalmology 1991; 98:786-806.

33. Kohner EM, Dollery CT, Bullpitt CJ. Cotton –wool spots in diabetic retinopathy. Diabetes1969; 18:691-704.

34. Ballantyne AJ, Michaelson. Classification of diabetic retinopathy. TransOphthalmolSoc UK 1947; 67:54.

35. Early Treatment Diabetic Retinopathy Study Group. ETDRS Report 1991.Fundus photographic risk factors for progression of diabetic retinopathy.Ophthalmology 1991;98:823-33.

36. Das T, Jalali S, Vedantam V, Majji AB. Retinal vascular disorders. In :Saxena S Ed: Clinical practice in ophthalmology. New Delhi: jaypee brothers 2003: 350.

37. Leske MC, Wu SY, Hennis A, Nemesure B, Hyman L, Schachat A. Incidence of diabetic retinopathy in the Barbados Eye Studies. Ophthalmology. 2003;110:941–47.

38. Van Leiden HA, Dekker JM, Moll AC, Nijpels G, Heine R. Riskfactors forincident retinopathy in a diabetic and nondiabetic population. The Hoorn Study.ArchOphthalmol. 2003;121:245–51.

39. Shah SM, Sung JU, Haller JA, Nguyen QD. Persistent diabetic macular edema is associated with elevated hemoglobin A1c. Am J Ophthalmol. 2005;139: 620–23.

40. Novotny HR, Alvis DR. A method of photographing fluorescence incirculatingblood in the human retina. Circulation 1961; 24:82 .

41. Kim NR, Kim YJ, Chin HS, Moon YS. OCT patterns in diabetic macular oedema: prediction of visual outcomes after focal laser photocoagulation. Br J Ophthalmol 2009; 93:901-05.

42. Duncan LJP, Cullen LF, Ireland JT, Nolan J, Clarke BF, Oliver MF. A 3 yeartrial of atromid in exudative diabetic retinopathy. Diabetic 1986; 17:458.

43. Kohner EM, Joplin GF, Cheng H, Black R, Fraser TR. Florid diabeticretinopathyand its response to treatment by photocoagulation or pitituaryablation.Diabetes 1976; 25:104.

44. Diabetic retinopathy vitrectomy study research goup. Vitrectomyindiabeticretinopathy. Arch ophthalmol 1985; 103:1644.

45. Diabetic Retinopathy Clinical Research Network. A randomized trial comparing intravitreal triamcinolone acetonide and focal/grid photocoagulation for diabetic macular edema. Ophthalmology 2008;115: 1447-9.

46. Scott IU, Edwards AR, Beck RW. Diabetic Retinopathy Clinical ResearchNetwork A phase II randomized clinical trial of intravitrealbevacizumab fordiabetic Macular edema. Ophthalmology 2007;114: 1860-67.

47. Cunningham ET Jr, Adamis AP, AltaweelM .Macugen Diabetic RetinopathyStudy Group. Ophthalmology 2005;112:1747-57.

48. Botham KM and Mayes PA. HARPER'S ILLUSTRATED BIOCHEMISTRY.28th EDITION. Page no 121.

49. Howard BV and Howard WJ. Pathophysiology and Treatment of LipidDisorders in Diabetes. Joslin's DIABETES MELLITUS. FOURTEENTHEDITION, Page no: 563-582.

50. Panarotto D, Remillard P, Bouffard L, Maheux P. Insulin resistance affects theregulation of lipoprotein lipase in the postprandial period and in an adiposetissuespecific manner. EurClin Invest 2002;32:84-92.

51. Krentz AJ. Lipoprotein abnormalities and their consequences for patients withtype 2 diabetes. Diabetes ObesMetab 2003;5:S19-S27.

52. Garber AJ, Vinik AI, Crespin SR. Detection and management of lipiddisorders in diabetic patients: a commentary for clinicians. Diabetes Care1992; 15:1068-1074.

53. Howard BV, Knowler WC, Vasquez B, Kennedy AL, Pettit DJ, and BennettPH. Plasma and lipoprotein cholesterol and triglyceride in the Pima. Indianpopulation. Comparison of diabetics and nondiabetics. 1984. *Arteriosclerosis.*4: 462-471.

54. Bowie A, Owens D, Collins P. Glycosylated low density lipoprotein is moresensitive to oxidation: implications for the diabetic patient? Atherosclerosis1993; 10:63-67.

55. Matsui J, Onuma T, Tamasawa N, Suda T. Effects of advanced glycationendproducts on the generation of macrophage-mediated oxidized low-densitylipoprotein. J Diabetes Complications 1997; 11:338-342.

56. Taskinen, MR, Beltz WF, Harper I, Fields RM, Schonfeld G, Grundy SM, andHoward BV. 1986. The effects of noninsulin-dependent diabetes mellitus onVLDL triglyceride and VLDL apoB metabolism: studies before and aftersulfonylurea therapy. *Diabetes.* 35:1268-77.

57. Haffner, SM, Foster DM, Kushwaha RS, and Hazzard WR. 1984. Retardedchylomicron apolipoprotein-B catabolism in type 2 (non-insulin-dependent)diabetic subjects with lipaemia. *Diabetologia.* 26: 349-354.

58. Howard BV, Abbott WF, Beltz WF, Harper I, Fields RM, Grundy SM andTaskinen RM. 1986. The effect of non-insulin-dependent diabetes on very lowdensity lipoprotein and low density lipoprotein metabolism in man.*Metabolism.* In press.

59. Schonfeld, G, Birge C, Miller JP, Kessler G, and Santiago J. 1974.Apolipoprotein B levels and altered lipoprotein composition in diabetes.*Diabetes.* 23**:** 827-834.

60. Ramavat PR, Ramavat MR., Ghugare BW, Vaishnav RG, Joshi MU. Prevalence of diabetic retinopathy in western Indian type 2 diabetic population:A Hospital based Cross Sectional Study. J ClinDiagnRes.Jul 2013;7(7):1387-1390.

61. Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das A K, et al. Diabetes Epidemiology study Group in India(DESI). High prevalence of diabetes and impaired glucose tolerance in India:National Urban Diabetes survey. Diabetologia 2001; 44 :1094-101.

62. Daniel W W.Biostatistics:A Foundation for Analysis in the Health Science.,7th ed.1999,John Wiley & Sons, New york.

63. Epidemiologic Study of Diabetic Retinopathy (WESDR), XIII: relationship between serum cholesterol to retinopathy and hard exudate. Ophthalmology 1991; 98: 1261-5.

64. Weiqin Chen, Donald B. Jump, Maria B. Grant, Walter J. Esselman, and Julia V. Busik. Dyslipidemia, but Not Hyperglycemia, Induces Inflammatory Adhesion Molecules in Human Retinal Vascular Endothelial Cells. IOVS, 2003 Nov; 44(11): 5016-5022.

65. Mohan V, Sandeep S, Deepa R, Shah B. Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res March 2007; 125: 217-30.

66. Rema M, Srivastava BK, Anitha B, Deepa R, Mohan V. Association of serum lipids with diabetic retinopathy in urban South Indians-the Chennai Urban Rural Epidemiology Study (CURES) Eye Study-2. Diabetic Medicine 2006; 23: 1029-1036.

67. Rema M, Ponnaiya M, Mohan V. Prevalence of retinopathy in non insulin dependent diabetes mellitus at a diabetes centre in southern India. Diabetes Res ClinPract 1996; 34: 29-36.

68. Sunil Gupta, Ajay Ambade. Prevalence of Diabetic Retinopathy and Influencing Factors amongst type 2 Diabetics from Central India. International Journal of Diabetes in Developing Countries, 2004; 24: 75-78.

69. Paromita K, Peacock I, Donnelly R. The UK Prospective Diabetes Study (UKPDS): clinical and therapeutic implications for type 2 diabetes. Br J ClinPharmacol. 1999 November; 48(5): 643-648.

70. Haddad E, Ossama A W, Mohammed Kamal Saad. Prevalence and risk factors for diabetic retinopathy among Omani diabetics. Br J Ophthalmol 1998; 82: 901-90.

71. Lill-Inger Larsson, Albert Alm, FolkeLithner, GostaDahlen, Reinhold Bergstrom The association of hyperlipidemia with retinopathy in diabetic patients aged 15-50 years in the county of Umea. ActaOphthalmol. Scand. 1999; 77: 585-591.

ANNEXURE –I

ANNEXURE - II

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned,_____ , S/O D/O W/O _____, aged ____years, ordinarily resident of _____ do hereby state/declare that _____

has examined me thoroughly on _____ at _____ (place) and it has been explained to me in my own language that I am suffering from _____ disease (condition) and this disease/condition mimic following diseases. Further Dr. informed me that he/she is conducting dissertation/research titled "Lipid Profile In Diabetic Retinopathy In Type 2 Diabetis Mellitus" under the guidance of Dr Sunil G. Biradar requesting my participation in the study. Apart from routine treatment procedure, the pre-operative, operative, post-operative and follow-up observations will be utilized for the study as reference data.

Doctor has also informed me that during conduct of this procedure like adverse results may be encountered. Among the above complications most of them are treatable but are not anticipated hence there is chance of aggravation of my condition and in rare circumstances it may prove fatal in spite of anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study help in evaluation of the results of the study which is useful reference to treatment of other similar cases in near future, and also I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made/ photographs/ video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on information given by me, I can ask any clarification during the course of treatment / study related to diagnosis, procedure of treatment, result of treatment or prognosis. At the same time I have been informed that I can withdraw from my participation in this study at any

time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged.

After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Shri/Smt _____ under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of patient:

Signature of doctor:

Witness: 1.

2.

Date:

Place

Consent form for Fundus Fluorescein Angiography:

Fluorescein angiography is a diagnostic test which uses special camera to photograph the structures of the back of the eye. This test is used to detect any leakage or damage to the blood vessels that nourish the retina.

Flourescein dye glows in visible light and is injected into a vein in the arm of the patient , the dye reaches the retinal vessels through the vascular system. This test does not involve the use of X-ray or any other harmful form of radiation.

Fundus fluorescein angiography is considered to be a very safe test and side effects are uncommon. However the possibility of side effects cannot be completely ruled out. Fluorescein does not contain iodine and hence can be used in patients allergic to iodine. Some patients can experience slight nausea and vomiting. some patients who are allergic to the dye can develop rashes, which require medications such as antihistaminics and steroids. Very rarely a life threatening allergic reaction known as anaphylaxis can occur which requires prompt medical treatment.There may be infiltration of the dye into the skin at the site of injection, which could cause discolouration of the skin at that site. Flourescein dye will turn a patients urine orange and may slightly discolour the skin as well for a brief period of time.

I understand that fluorescein angiography is only a test and not a treatment modality. I understand the risks involved with this test and hereby give consent to undergo this test.

I hereby also give my consent to be included as a participant in the study

“Lipid profile in diabetic retinopathy in type 2 diabetis mellitus”

Name of the patient _____ Date _____

Signature of the patient_____ Time _____

Name of the impartial Witness_____

Signature of the impartial witness _____

Name of Investigator_____

Signature of the Investigator _____

ANNEXURE - III

PROFORMA

NAME :

AGE/SEX : I.P/OP. NO:

OCCUPATION :

ADDRESS:

CONTACT NO:

SOCIO ECONOMIC STATUS:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

TYPE OF DIABETES:

DURATION OF DIABETES:

PAST HISTORY: HTN / TB / DRUG ALLERGY / NEPHROPATHY

HISTORY OF SIMILIAR COMPLAINTS:

HISTORY OF OCULAR DISEASES:

TREATMENT HISTORY: REGULAR / IRREGULAR

PREVIOUS OCULAR CHECK UP: YES/NO

FAMILY HISTORY:

GENERAL PHYSICAL EXAMINATION :

BP :PULSE : PALLOR : ICTERUS:

CLUBBING : OEDEMA : CYNOSIS:

OCULAR EXAMINATION:

LOCAL OCULAR EXAMINATION:

Head posture:

Facial Symmetry:

Ocular position:

Ocular movements: (RE) (LE)

Visual Acuity:

Lids:

Conjunctiva:

Cornea:

Anterior Chamber :

Iris:

Pupil:

Lens:

Sac Syrnging:

Fundus examination:

Media:

Disc:

Bloodvessels:

Background:

Macula:

Grades of Diabeic retinopathy:

INVESTIGATIONS

FBS:

PPBS:

$HbA_{1C:}$

LIPID PROFILE : (Total cholesterol, HDL, LDL, Triglycerides, VLDL)

URINE ALBUMIN:

IOP :

FUNDUS FLUORESCEIN ANGIOGRAPHY(whenever indicated):

TREATMENT ADVISED:

FOLLOW-UP:

ANNEXURE IV

KEYS TO MASTER CHART

M - male

F - female

DUR OFDIAB -Duration of Diabetes

FBS - Fasting Blood Sugar

RE - right eye

LE - left eye

PSEUDO P - PseudoPhakia

PSC - posterior subcapsular cataract

PRP - Pan Retinal Photocoagulation

NS - Nuclear sclerosis

CF - Counting fingers

PPBS - Post Prandial Blood Sugar

MT - Meter

CSME - Clinicalllysignificant macular edema

MOD - Moderate

SEV - Severe

V SEV - Very severe

NPDR - Non proliferative diabetic retinopathy

PDR - Proliferative diabetic retinopathy

HbA1C - Glycosylated haemoglobin

TC - Total cholesterol

HDL - High density lipoprotein

LDL - Low density lipoprotein

TG - Triglycerides

VLDL - Very low density lipoprotein

SUR - Surgery

MED MAG - Medical management

MASTER CHART

SlNo	NAME	IP /OP NO.	AGE(Yrs)	SEX	DUR OF DIAB(Yrs)	TREATMENT HISTORY	VISION		PIN HOLE		EYELID		CONJUNCTIVA		CORNEA		PUPIL		LENS		IOP(mmHg)		FUNDUS/DIAGNOSIS		FBS (mg/dl)	PPBS (mg/dl)	HbA1c (%)	Urine Albumin	LIPID PROFILE (mg/dl)					TREATMENT
							RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT EYE	LEFT EYE					TC	HDL	LDL	TG	VLDL	
1	SAHIBAGAODA	35	55	M	5	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	12.2	12.2	SEV NPDR	SEV NPDR	130	170	4	Nill	223	31	169	114	22.8	MED MAG
2	BHINU	358	65	F	20	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	14.6	13.4	MILD NPDR	MILD NPDR	100	180	8	Nill	210	40	100	190	38	MED MAG
3	HASANSAB	367	50	M	4	REGULAR	6/18p	6/18p	6/18	6/18	NORMAL	NORMAL	PTERYGIUM	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	13.4	10.2	PDR WITH CSME	PDR WITH CSME	180	210	7.4	+	270	53	193	149	29	PRP +MED MAG
4	BHIMMA	471	58	M	7	REGULAR	6/18	6/18	6/6	6/6	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MILD NPDR	MILD NPDR	90	130	6	Nill	190	31	100	210	30	MED MAG
5	NINGAPPA	1341	55	M	15	IRREGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	13.4	MOD NPDR	MOD NPDR	310	318	10.8	++	240.4	28	160	180	32.4	MED MAG
6	ASHOK	2458	64	M	13	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MOD NPDR	120	180	7	Nill	220	38	140	220	35	MED MAG
7	MALAKAPPA	2984	80	M	20	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	MOD NPDR	MOD NPDR	120	190	8.2	Nill	240	38	130	240	38	MED MAG
8	CHANDABAI	3050	60	F	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MILD NPDR	MILD NPDR	130	160	6.2	Nill	230	65	140	170	50	MED MAG
9	CHANDABAI	3050	60	F	15	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MILD NPDR	MILD NPDR	110	160	6	Nill	180	60	120	130	40	MED MAG
10	MENAKSHI	3154	60	F	15	REGULAR	6/24	6/24	6/9	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS2	10.2	10.2	MOD NPDR	MOD NPDR	86	112	5	Nill	212	47.2	135	128	28.6	MED MAG
11	KRISHNA	3182	62	M	12	IRREGULAR	6/24	6/24	6/12p	6/12p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	10.2	PDR	PDR	210	325	10	++	380	30	160	180	60	PRP +MED MAG
12	LAXMI	3639	50	F	10	IRREGULAR	6/36	6/36	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	13.4	SEV NPDR	SEV NPDR	240	400	11.1	++	280	40	179	180	28	PRP +MED MAG
13	NOORSAHAB	4214	47	M	2	REGULAR	6/6	6/6	6/5	6/5	NORMAL	NORMAL	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	98	130	5.4	Nill	180	40	90	130	30	MED MAG
14	NARAYANA	5838	41	M	4	REGULAR	6/6p	6/6p	6/6	6/6	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	80	110	5	Nill	190	48	96	120	30	MED MAG
15	IRAYYA	6256	55	M	8	REGULAR	6/24	6/24	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MOD NPDR	MOD NPDR	120	170	8.2	Nill	260	32	130	260	40.1	MED MAG
16	NILABAI	8526	65	F	28	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	PDR	PDR	180	320	8.2	+	300	40	150	220	60	PRP +MED MAG
17	SURENDRA	8857	69	M	20	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	12.2	13.4	MOD NPDR	MOD NPDR	160	347	8.9	+	280	42	138	237	50.2	MED MAG
18	SHIVAPPA	10956	65	M	8	REGULAR	6/24	6/24	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MILD NPDR	MILD NPDR	80	130	6	Nill	180	48	100	200	32	MED MAG
19	SIDRAYYA	11142	60	M	15	REGULAR	CF-2MT	6/60	NI	6/24	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS3	NS2	14.6	14.6	MOD NPDR	MOD NPDR	140	190	8	+	240	40	140	26.2	51	PRP +MED MAG+(RE)SUR
20	ASHOK	12344	53	M	18	IRREGULAR	6/60	6/24	6/18	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	14.6	13.4	PDR WITH CSME	PDR WITH CSME	105	180	10.2	Nill	280	31	105	218	43.6	PRP +MED MAG
21	PRAKASH	14412	49	M	2	IRREGULAR	CF-3MT	CF-3MT	6/18p	6/24	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	13.2	PDR	PDR	289	326	13	++	310	48	140	210	46	PRP +MED MAG
22	PARVITAMMA	16294	60	F	20	REGULAR	6/18p	6/18p	6/12p	6/12p	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	14.6	SEV NPDR	SEV NPDR	140	178	9	+	350	25	130	210	33.4	PRP +MED MAG
23	BASANNA	16495	67	M	20	IRREGULAR	6/24	6/24	6/12	6/12	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSC WITH NS2	PSC WITH NS2	14.6	14.6	SEV NPDR	SEV NPDR	190	320	7.8	+	340	50	140	210	60	MED MAG
24	UDAY KUM,AR	18215	57	M	10	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS2	10.2	13.4	MOD NPDR	MOD NPDR	146	182	7.4	+	102	50	140	240	50	MED MAG
25	VARUN	18220	46	M	5	REGULAR	6/9	6/9	6/6	6/6	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	126	160	4.2	Nill	180	62	135	155	46	MED MAG
26	LAKAWWA	20164	48	F	10	REGULAR	6/24	6/24	6/18	6/18	XANTHELASMA	XANTHELASMA	NORMAL	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	OD NPDR WITHCSM	OD NPDR WITHCSM	116	220	7.4	Nill	323	40	176	536	107	MED MAG
27	BASLINGAMMA	22116	66	F	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS2	10.2	13.4	MOD NPDR	MOD NPDR	140	240	6.8	+	290	62	140	180	45	MED MAG
28	BASLINGAMMA	22116	66	F	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MOD NPDR	MOD NPDR	130	288	6.8	Nill	260	60	120	140	30	MED MAG
29	JAGANATA	22118	55	M	12	IRREGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	12.2	10.2	PDR	SEV NPDR	80	380	11	Nill	280	58	105	83	16.6	PRP +MED MAG
30	VITTAL TALWAR	22932	55	M	10	REGULAR	6/18p	6/18p	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.6	13.4	EV NPDR WITH CSM	EV NPDR WITH CSM	110	169	8	Nill	167	26	119	112	22.4	PRP +MED MAG
31	BASAPPA	23865	38	M	15	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	OD NPDR WITHCSM	OD NPDR WITHCSM	348	386	7.4	++	216	52	128	100	120	MED MAG
32	SHIVAPPA	26462	70	M	20	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	MOD NPDR	MOD NPDR	142	185	7.2	+	240	60	140	180	30	MED MAG
33	SHRIAPPA	26462	70	M	25	REGULAR	6/18	6/18	6/9	6/9	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	12.2	12.2	MOD NPDR	MOD NPDR	110	180	7.1	Nill	280	50	120	180	40.2	MED MAG
34	ISMAILSAB	28248	60	M	10	IRREGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	12.2	MOD NPDR	MOD NPDR	135	222	7.2	Nill	225	30	125	182	32	MED MEG
35	SIDAMMA	28751	65	M	8	REGULAR	6/12	6/12	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	14.6	13.4	MOD NPDR	MOD NPDR	130	280	8	Nill	260	52	140	180	60	MED MAG
36	FATIMA	30927	80	F	30	IRREGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	10.2	MOD NPDR	MOD NPDR	130	150	6.4	Nill	280	30	130	160	40	MED MAG
37	PASHURAM	31733	68	M	10	REGULAR	6/18	6/60	6/18	6/24	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	NS2	10.2	10.2	OD NPDR WITHCSM	OD NPDR WITHCSM	250	340	8.9	++	196	42	135	96	42	MED MAG
38	NEELAMMA	31844	65	F	12	IRREGULAR	6/36	6/36	6/24	6/24	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MILD NPDR	MILD NPDR	120	180	7.8	Nill	220	40	101	125	35	MED MAG
39	VISHWANATH	31875	55	F	15	IRREGULAR	6/60	6/36	6/24	6/24	BLEPHARITIS	BLEPHARITIS	PTERYGIUM	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	13.4	SEV NPDR	SEV NPDR	343	267	8.7	++	280	40	140	260	40	PRP +MED MAG
40	VISHWANATH	31875	58	M	12	REGULAR	6/18	6/18	6/6p	6/6p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	13.4	13.4	MOD NPDR	MOD NPDR	120	189	6.2	Nill	220	48	110	210	38	MED MAG
41	YASHWANT	31940	67	M	25	REGULAR	6/24p	6/24p	6/18p	6/18p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	13.4	MILD NPDR	MILD NPDR	112	186	6.6	Nill	139	30	173	178	35.8	MED MAG
42	GANGABAI	33209	50	F	5	REGULAR	6/36	6/12	6/18p	6/9	BLEPHARITIS	BLEPHARITIS	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS1	13.4	13.4	MOD NPDR	MOD NPDR	257	302	8.2	++	240	46	135	188	37	MED MAG +(RE)SUR
43	RENUKA	33737	68	F	10	REGULAR	6/24	6/24	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	12.2	12.2	MOD NPDR	MOD NPDR	120	160	6.8	Nill	330	40	120	170	30	MED MAG
44	MANGALAPPA	34675	60	M	10	REGULAR	6/18	6/18p	6/9P	6/9P	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	10.2	PDR WITH CSME	PDR WITH CSME	215	315	9.2	++	325	30	170	180	25.2	PRP +MED MAG
45	HONU	35083	36	M	14	REGULAR	6/6	6/6	6/5	6/5	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	170	305	8.1	+	281	42	120	160	40	MED MAG
46	SAROJNI	35577	55	F	10	IRREGULAR	6/18	6/18	6/9P	6/9P	NORMAL	CHALAZION	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	12.2	12.2	MILD NPDR	MILD NPDR	219	271	11	++	220	48	170	150	28	MED MAG
47	CHADAMAND	35639	78	M	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	OD NPDR WITHCSM	OD NPDR WITHCSM	130	181	8.1	Nill	280	35	120	240	38.2	MED MAG
48	LAGAMANNA	35762	52	M	8	REGULAR	6/12	6/12	6/9	6/9	BLEPHARITIS	BLEPHARITIS	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	120	160	7.2	Nill	180	60	130	150	40.2	MED MAG
49	MOHIT	35926	43	M	2	REGULAR	6/9	6/9	6/6	6/6	STYE	STYE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	80	160	7	Nill	220	38	100	205	38	MED MAG
50	YALLAWA	36189	66	F	20	IRREGULAR	6/24	6/24	6/18p	6/18p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	PDR	PDR	189	320	8.1	+	320	40	149	220	50	PRP +MED MAG
51	IRAPPA	37209	55	M	10	IRREGULAR	6/18	6/18	6/9	6/9	BLEPHARITIS	BLEPHARITIS	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.6	14.6	MOD NPDR	MOD NPDR	110	190	8.1	Nill	240	40	120	210	28	MED MAG
52	SIDRAM	37398	65	M	22	REGULAR	6/18	6/24	6/6	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	NS2	13.4	13.4	MOD NPDR	MOD NPDR	160	240	8.6	+	270	45	100	305	40	MED MAG
53	BASAVARAJ	38043	63	M	15	REGULAR	6/36	6/36	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MOD NPDR	81	170	7.8	Nill	280	36	110	210	38	MED MAG
54	SURYA	38259	62	F	13	REGULAR	6/60	6/60	6/24	6/24	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS3	NS3	13.4	13.4	MOD NPDR	MOD NPDR	104	211	6.4	Nill	248	26	98.4	157	23.5	MED MAG+(BE)SUR
55	SIDDAMMA	38788	40	M	4	REGULAR	6/6p	6/6p	6/6	6/6	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	80	130	5.8	Nill	21	48	100	180	32	MED MAG
56	HARIBA	38795	55	M	12	REGULAR	6/24	6/24	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MOD NPDR	MOD NPDR	87	174	9	Nill	240	40	140	180	36	MED MAG
57	SANGAPPA	38797	75	M	20	IRREGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MILD NPDR	MILD NPDR	100	160	6.2	Nill	200	40	120	180	40	MED MAG
58	SAROJINI	39022	59	F	10	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	SEV NPDR	SEV NPDR	220	290	7.6	++	310	50	140	210	60	PRP +MED MAG
59	CHANDRASHEKHA	39084	65	M	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	12.2	12.2	MOD NPDR	MOD NPDR	110	180	6.8	Nill	280	61	120	60	40	MED MAG
60	CHANDRASHEKHR	39084	65	M	15	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MOD NPDR	130	175	6.5	Nill	230	60	130	165	50.2	MED MAG
61	MUDAMALAPPA	39092	70	M	30	IRREGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSC WITH NS2	PSC WITH NS2	14.6	13.4	PDR	PDR	255	374	10.2	++	330.2	45	185	240	20.2	PRP +MED MAG
62	AFEED HUSSAIN	39124	43	M	15	REGULAR	6/9	6/9	6/6	6/6	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	12.2	14.6	MILD NPDR	MILD NPDR	85	90	8	Nill	208	42	129	160	42.1	MED MAG
63	JANGIR	39838	40	M	10	IRREGULAR	6/6	6/6	6/5	6/5	STYE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	13.4	13.4	MOD NPDR	MOD NPDR	319	438	10	++	270	39	173	286	57.2	MED MAG
64	SABU	40401	60	M	10	REGULAR	6/24	6/24	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	14.6	MOD NPDR	MOD NPDR	160	287	6.2	+	270	34	78	177	35	MED MAG
65	ROHIT	40707	48	M	5	REGULAR	6/24	6/24	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MOD NPDR	MOD NPDR	91	148	8.1	Nill	262	35	89	187	37	MED MAG
66	VIMAPPA	40725	60	M	15	REGULAR	6/24	6/24	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	13.4	MILD NPDR	MILD NPDR	130	160	5.2	Nill	230	30	110	210	38	MED MAG
67	SHIVALINGAPPA	40768	60	M	12	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	13.4	13.4	MOD NPDR	MOD NPDR	130	210	8.1	Nill	250	60	140	170	50	MED MAG
68	BHIMANNA	41022	50	M	7	REGULAR	6/24	6/24	6/9	6/9	CHALAZION	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.6	14.6	MOD NPDR	MOD NPDR	110	180	8	Nill	280	38	140	210	30	MED MAG
69	NABILAL	41072	50	M	8	IRREGULAR	6/6	6/6	6/5	6/5	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.6	13.4	MILD NPDR	MILD NPDR	281	270	9.9	++	150	28.4	88	170	24	MED MAG
70	SHIVANAD	41823	40	M	2	REGULAR	6/6p	6/6p	6/6	6/6	STYE	NORMAL	PTERYGIUM	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MILD NPDR	MILD NPDR	96	140	7.8	Nill	179	46	106	131	30	MED MAG
71	SIDRAYA	42027	60	M	8	REGULAR	6/24	6/24	6/28	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSC WITH NS2	PSC WITH NS2	12.2	12.2	PDR WITH CSME	PDR WITH CSME	160	280	7.8	+	294	50	106	375	75.2	PRP +MED MAG
72	SAROJNI	42421	50	F	5	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	12.2	MOD NPDR	MOD NPDR	120	190	6.6	Nill	260	14.2	140	200	46.2	MED MAG
73	PARSAPPA	45989	60	M	12	REGULAR	6/18	6/18	6/12	6/12	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	12.2	12.2	MOD NPDR	MOD NPDR	139	160	8	Nill	300	48	70	195	60	MED MAG
74	PARSAPPA	45989	65	M	10	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	SEV NPDR	SEV NPDR	100	190	8	Nill	260	60	120	180	30	MED MAG
75	PARSAPPA	45989	55	M	10	REGULAR	6/18	6/18	6/9	6/9	CHALAZION	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MILD NPDR	MILD NPDR	108	160	6.1	Nill	210	50	110	160	40	MED MAG
76	SHIVAPPA	45997	62	M	10	REGULAR	6/12	6/12	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	13.4	MOD NPDR	MOD NPDR	130	182	7.8	Nill	210	52	140	175	60	MED MAG
77	SHIRIAPPA	45997	62	M	10	REGULAR	6/12	6/12	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MED MAG	90	120	6	Nill	280	60	180	140	30	MED MAG
78	SIDDAMMA	55520	65	F	15	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	SEV NPDR	SEV NPDR	180	280	7.4	+	340	50	150	180	60	PRP +MED MAG
79	SHAKUNTALA	55542	62	F	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MOD NPDR	MOD NPDR	130	190	6.8	Nill	260	55	140	170	60	MED MAG
80	SHAKUNTALA	55560	70	F	20	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	12.2	MOD NPDR	MOD NPDR	120	180	6.2	Nill	260	60	180	175	40	MED MAG
81	JAYAMMA	55626	60	F	10	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	12.2	12.2	MILD NPDR	MILD NPDR	90	130	6	Nill	240	60	120	180	40	MED MAG

SlNo	NAME	IP /OP NO.	AGE (Yrs)	SEX	DUR OF DIAB (Yrs)	TREATMENT HISTORY	VISION		PIN HOLE		EYELID		CONJUNTIVA		CORNEA		PUPIL		LENS		IOP(mmHg)		FUNDUS/DIAGNOSIS		FBS (mg/dl)	PPBS (mg/dl)	HbA1c (%)	Urine Albumin	LIPID PROFILE (mg/dl)					TREATMENT
							RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT EYE	LEFT EYE					TC	HDL	LDL	TG	VLDL	
82	CHANDAPPA	63199	65	M	8	REGULAR	6/36	6/60	6/12	6/36	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS3	NS3	14.6	13.4	MOD NPDR	MOD NPDR	130	240	7.8	Nill	280	48	120	160	28	MED MAG+(LE)SUR
83	KASTURI	64138	60	F	12	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS12	10.2	13.4	MOD NPDR	MOD NPDR	120	189	6.6	Nill	280	40	130	160	49	MED MAG
84	KASTURI	64138	60	F	15	REGULAR	6/18	6/18	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	13.4	14.6	MOD NPDR	MOD NPDR	110	180	6.6	Nill	240	60	121	140	35	MED MAG
85	SY JAREALAGI	73232	66	M	10	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MOD NPDR	92	190	6.2	Nill	240	40	129	140	40	MED MAG
86	DEVAKI	82755	65	F	18	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	PDR	PDR	150	220	8.8	+	300	48	140	255	50	PRP +MED MAG
87	ARNARAY	82937	62	M	10	REGULAR	6/24	6/24	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MOD NPDR	MOD NPDR	120	165	6.4	Nill	230	60	120	160	40	MED MAG

88	ANNAPPA	83251	56	F	6	IRREGULAR	6/18	6/18	6/12	6/12	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	PDR	PDR	220	310	9.8	++	310	60	150	210	50	PRP +MED MAG
89	CHANDBASA	87291	60	M	10	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	13.4	MILD NPDR	MILD NPDR	100	125	5	Nill	180	60	130	160	45	MED MAG
90	MAHADEV	91061	58	M	10	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	13.4	MOD NPDR	MOD NPDR	130	180	6.6	Nill	240	60	135	160	42	MED MAG
91	MAHADEV	91061	78	M	25	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	MOD NPDR	MOD NPDR	120	160	6	Nill	210	60	120	160	40	MED MAG
92	NIRMALA	93008	60	F	15	REGULAR	6/18	6/18	6/12	6/12	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.6	14.6	V SEV NPDR	V SEV NPDR	160	320	9	+	340	40	140	220	60	PRP +MED MAG
93	SK GIRI	99975	64	M	13	IRREGULAR	6/12	6/24	6/9	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSC WITH NS2	10.2	13.4	PDR	PDR	160	320	10.2	+	320	60	130	190	60	PRP +MED MAG
94	MAHADEV	108624	60	M	8	IRREGULAR	6/24	6/24	6/18p	6/18p	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	14.6	V SEV NPDR	V SEV NPDR	180	380	10.2	+	355	38	180	260	20	PRP +MED MAG
95	RATANABAI	109335	63	F	10	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MOD NPDR	MOD NPDR	108	165	6.4	Nill	250	40	120	150	40	MED MAG
96	GANGABAI	109377	71	F	25	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MOD NPDR	MOD NPDR	130	160	6.2	Nill	260	60	120	180	40	MED MAG
97	GANGABAI	109377	71	F	20	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	14.6	MOD NPDR	MOD NPDR	90	160	6.1	Nill	220	60	110	150	40	MED MAG
98	TANMAY	109411	60	M	10	REGULAR	6/18p	6/24p	6/9	6/9p	BLEPHARITIS	BLEPHARITIS	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MOD NPDR	MOD NPDR	126	160	6.2	Nill	210	50	120	180	45	MED MAG
99	GAURAMMA	112439	65	F	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MILD NPDR	MILD NPDR	90	120	6	Nill	260	50	120	180	30	MED MAG
100	GANGABAI	116846	78	F	20	IRREGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	SEV NPDR	SEV NPDR	160	310	8.1	+	320	50	140	180	40	MED MAG
101	BASAPPA	116872	68	M	10	REGULAR	6/24	6/24	6/18	6/18	CHALAZION	CHALAZION	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	10.2	MILD NPDR	MILD NPDR	120	180	6.2	Nill	200	50	110	170	62	MED MAG
102	PRAMILABAI	123601	76	F	20	REGULAR	6/24	6/24	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	14.6	13.4	MOD NPDR	MOD NPDR	100	160	6.2	Nill	248	40	120	180	40	MED MAG
103	YAMNAWWA	127366	75	F	25	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MOD NPDR	MOD NPDR	120	165	6	Nill	240	50	120	150	40	MED MAG
104	JAYACHAND	133208	60	M	12	IRREGULAR	6/60	6/60	6/18p	6/18p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	12.3	EV NPDR WITH CSM	EV NPDR WITH CSM	232	399	11.7	++	256	44	181	153	30.6	PRP +MED MAG
105	NINGAPPA	135438	70	M	15	REGULAR	6/24	6/24	6/12	6/12	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	17.3	SEV NPDR	SEV NPDR	210	350	8.2	++	340	50	120	180	140	PRP +MED MAG
106	PRATIBHA	135455	66	F	15	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS2	10.2	13.4	MOD NPDR	MOD NPDR	110	140	6	Nill	260	30	110	180	30	MED MAG
107	LALITABAI	153336	72	F	20	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	SEV NPDR	SEV NPDR	210	300	7.8	++	350	50	160	180	40	PRP +MED MAG
108	KAMLAKASI	164265	55	F	8	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS2	10.2	13.4	MILD NPDR	MILD NPDR	100	150	6	Nill	210	50	120	130	35	MED MAG
109	SHIVAPPA	177561	75	M	20	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	SEV NPDR	SEV NPDR	220	330	8.6	++	290	60	100	180	40.1	PRP +MED MAG
110	SHIVLINGAPPA	177687	53	M	8	REGULAR	6/12	6/12	6/9	6/9	NORMAL	STYE	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MILD NPDR	MILD NPDR	90	120	6.2	Nill	240	40	130	170	60	MED MAG
111	DANNAMMA	181427	45	F	4	REGULAR	6/6p	6/6p	6/6	6/6	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	12.2	12.2	MOD NPDR	MOD NPDR	130	180	8.2	Nill	260	30	110	180	30	MED MAG
112	SATTAWWA	186760	65	M	12	REGULAR	6/24	6/24	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	13.4	13.4	MOD NPDR	MOD NPDR	90	220	6.3	Nill	260	60	120	150	66	MED MAG
113	SATTAWWA	186760	65	F	12	REGULAR	6/12	6/12	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MOD NPDR	MOD NPDR	108	185	6.2	Nill	280	160	110	170	30	MED MAG
114	SURESH	238312	57	M	10	REGULAR	6/24	6/24	6/18	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	SEV NPDR	SEV NPDR	160	180	7.2	+	280	55	120	170	35	PRP +MED MAG
115	CHANDSAS	238600	65	M	15	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	10.2	PDR	PDR	180	210	7.1	+	360	58	120	180	30	PRP +MED MAG
116	MAHADEV	244082	60	M	15	REGULAR	6/24	6/24	6/18	'6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	14.6	SEV NPDR	SEV NPDR	130	320	9	Nill	280	30	150	180	45	PRP +MED MAG
117	RAMESH	257712	64	M	15	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	13.4	PDR	PDR	210	321	9	++	320	40	180	210	40	PRP +MED MAG
118	MAHADEVI	261601	60	F	12	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	13.4	13.4	MILD NPDR	MILD NPDR	80	120	6	Nill	210	60	120	140	60	MED MAG
119	BASAVARAJ	267309	60	M	12	REGULAR	6/12	6/12p	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.6	14.6	MILD NPDR	MOD NPDR	110	200	6.4	Nill	240	50	120	150	42	MED MAG
120	MALLANA	274950	65	M	6	REGULAR	6/36	6/36	6/18p	6/18p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSC WITH NS1	PSC WITH NS1	14.6	13.4	EV NPDR WITH CSM	EV NPDR WITH CSM	175	240	9	+	280	40	179	180	25.4	PRP +MED MAG
121	BASAPPA	282480	65	M	15	REGULAR	6/24	6/24	6/18	,6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	13.4	PDR	PDR	180	220	7.4	+	340	50	140	280	50	PRP +MED MAG
122	SHIVAPPA	283689	65	F	18	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MOD NPDR	130	155	6.2	Nill	270	50.2	120	180	40	MED MAG
123	NEELAMMA	287693	65	F	18	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MILD NPDR	MOD NPDR	120	140	6.2	Nill	250	40	110	170	30	MED MAG
124	GANGABAI	287703	62	F	10	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	14.6	MOD NPDR	MOD NPDR	100	160	6	Nill	270	50	120	180	42	MED MAG
125	KAMALANA	303385	65	F	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MOD NPDR	98	130	6	Nill	240	30	160	210	60	MED MAG
126	LAGAMAWWA	316707	50	M	5	REGULAR	6/12	6/12	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MILD NPDR	MILD NPDR	82	120	6	Nill	190	55	140	160	60	MED MAG
127	CHANDAPPA	326446	60	M	12	REGULAR	6/24	6/24	6/18	6/18p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	PDR	PDR	190	315	8.1	+	320	30	180	220	60	PRP +MED MAG
128	RULABAI	335717	65	F	11	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	13.4	MOD NPDR	MOD NPDR	90	160	6.1	Nill	260	48	120	180	46	MED MAG
129	BASAPPA	336207	56	M	6	REGULAR	6/18	6/18	6/6p	6/6p	BLEPHARITIS	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.3	10.2	MOD NPDR	MOD NPDR	80	130	7	Nill	250	50	140	180	60	MED MAG
130	NEELAPPA	336215	56	M	8	REGULAR	6/12	6/12	6/6	6/6	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	13.4	13.4	MILD NPDR	MILD NPDR	80	120	6	Nill	200	60	120	170	60	MED MAG
131	HANUMANTH	348090	60	M	10	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	17.3	17.3	SEV NPDR	SEV NPDR	180	310	8.1	+	330	40	150	190	70	PRP +MED MAG
132	GOPAL	355020	59	M	10	IRREGULAR	6/12	6/24	6/6p	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	NS2	13.4	10.2	SEV NPDR	V SEV NPDR	180	315	8.6	+	310	50	150	210	60	PRP +MED MAG
133	GOPAL	355020	59	M	15	IRREGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	10.2	SEV NPDR	SEV NPDR	190	230	8	+	280	52	180	200	50	MED MAG
134	AMRUTA	358717	63	F	8	REGULAR	6/18	6/18	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	NS2	10.2	10.2	MOD NPDR	MOD NPDR	120	189	6.5	Nill	280	40	130	180	48	MED MAG
135	VV PUJARI	362033	52	F	5	REGULAR	6/12	6/12	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MOD NPDR	MOD NPDR	120	186	6.8	Nill	270	40	130	60	46.2	MED MAG
136	YAMNAPPA	362841	60	M	8	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	13.4	MOD NPDR	MOD NPDR	120	180	8	Nill	240	40	100	166	60	MED MAG
137	SHIVABASAPPA	362923	60	M	8	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	14.6	MOD NPDR	MOD NPDR	130	160	8	Nill	260	482	130	161	40.2	MED MAG
138	BHIMAPPA	365624	60	M	5	REGULAR	6/24	6/24	6/18	6/1'8	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSC WITH NS2	PSC WITH NS2	10.2	13.4	MOD NPDR	MOD NPDR	120	180	8.9	Nill	260	42	130	170	42	MED MAG
139	BHINU	371760	58	M	10	REGULAR	6/18	6/18	6/6p	6/6p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	14.6	14.6	MOD NPDR	MOD NPDR	98	140	6	Nill	270	55	110	180	40	MED MAG
140	NEELAMMA	372770	66	F	10	IRREGULAR	6/12	6/12	6/9p	6/9p	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	SEV NPDR	SEV NPDR	140	210	8	+	380	42	160	190	42.2	MED MAG
141	SHANTA	374777	60	F	6	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	13.4	13.4	MOD NPDR	MOD NPDR	110	200	8	Nill	260	40	150	180	46	MED MAG
142	SUGALABAI	386690	60	F	8	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	14.6	MOD NPDR	MOD NPDR	100	160	6	Nill	280	150	120	160	40	MED MAG
143	KANTAPPA	408440	60	F	10	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	PTERYGIUM	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	14.6	13.4	MOD NPDR	MOD NPDR	130	170	6.2	Nill	280	60	140	200	40	MED MAG
144	KANTAPPA	414253	60	M	20	REGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	ONJUNCTIVIT	ONJUNCTIVIT	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	13.4	MOD NPDR	MOD NPDR	155	220	7	+	260	52	136	170	60	MED MAG
145	UMLU	425829	65	M	6	REGULAR	6/60	6/60	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	14.6	MOD NPDR	MOD NPDR	160	210	8	+	230	40	135	240	38	MED MAG
146	MALLAMMA	425953	58	F	8	IRREGULAR	6/36	6/60	6/24	6/24	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSC WITH NS1	PSC WIH NS1	14.6	13.4	MILD NPDR	MILD NPDR	241	280	12.7	++	148	29	96.6	112	22.4	MED MAG
147	SANTA	426640	48	F	3	IRREGULAR	6/60	6/24	6/24	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	TRANSPARENT	10.2	10.2	SEV NPDR	SEV NPDR	262	342	10	++	265	42	95	155	31	PRP +MED MAG
148	SRIDEVI	428516	65	F	10	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	12.2	12.2	MILD NPDR	MILD NPDR	130	170	6.2	Nill	210	60	120	140	45	MED MAG
149	IB PADASHALI	429942	63	M	12	IRREGULAR	6/24	6/36	6/9	6/24	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	13.4	13.4	PDR	PDR	188	210	11.5	+	191	32	118	204	11	PRP +MED MAG
150	GANGABAI	433373	75	F	16	IRREGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	17.3	14.6	SEV NPDR	SEV NPDR	160	230	10.6	+	310	57	140	190	48	MED MAG
151	UMA	435031	62	F	10	REGULAR	6/24	6/24	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	NS2	10.2	13.4	MOD NPDR	MOD NPDR	176	360	7.4	+	260	63	147	249	49.8	MED MAG
152	KASHIBAI	448867	70	F	10	IRREGULAR	CF-2MT	CF-2MT	NI	NI	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSC WITH NS3	PSC WITH NS3	12.2	12.2	PDR	PDR	210	310	12	++	310	40	140	200	48	PRP +MED MAG
153	SHANTABAI	454570	45	F	4	IRREGULAR	6/12	6/12	6/9	6/9	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	PDR	PDR	140	190	9.8	+	280	50	190	160	30	PRP +MED MAG
154	BASAVVARAJESH	455034	46	F	10	REGULAR	6/12	6/12	6/6	6/6	XANTHELASMA	XANTHELASMA	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	PDR	PDR	128	180	9	Nill	340	43	120	195	30	PRP +MED MAG
155	KALLAPPA	458833	70	M	25	IRREGULAR	6/18	6/18	6/9p	6/9p	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	14.6	MOD NPDR	MOD NPDR	140	240	10	+	280	38	135	171	40	MED MAG
156	KALAPPA	458833	70	M	11	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	MOD NPDR	MOD NPDR	130	180	7.4	Nill	290	60.2	140	185	50.2	MED MAG
157	RAJU	458887	39	M	8	REGULAR	6/18	6/18	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	PDR	PDR	180	280	9	+	230	51	201	160	28	PRP +MED MAG
158	SUSALABAI	459687	60	F	10	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	13.4	13.4	MOD NPDR	MOD NPDR	180	260	7.8	+	280	60	140	170	70	MED MAG
159	SUSALABAI	459687	60	F	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	14.6	14.6	MILD NPDR	MILD NPDR	120	140	6.2	Nill	155	67	128	160	40	MED MAG
160	SUSALABAI	459687	60	F	10	REGULAR	6/24	6/24	6/12	6/12	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS2	NS2	10.2	10.2	MOD NPDR	MOD NPDR	80	120	6	Nill	248	38.1	140	160	50.1	MED MAG
161	RAVINDRA	468403	55	M	5	IRREGULAR	6/18	6/18	6/6p	6/6p	NORMAL	NORMAL	PTERYGIUM	PTERYGIUM	NORMAL	NORMAL	NORMAL	NORMAL	TRANSPARENT	TRANSPARENT	10.2	10.2	MOD NPDR	MOD NPDR	120	130	6.1	Nill	260	45	130	170	60	MED MAG
162	MAYAPPA	476466	70	M	20	IRREGULAR	6/24	6/24	6/18	6/18	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	10.2	13.4	PDR	PDR	189	310	7.6	+	340	60	130	150	50	PRP +MED MAG
163	RAMAPPA	476761	60	M	15	REGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	PSEUDO P	PSEUDO P	14.6	14.6	MOD NPDR	MOD NPDR	120	180	7.1	Nill	240	58	140	180	45	MED MAG
164	RAMAPPA	476761	60	M	12	IRREGULAR	6/18	6/18	6/9	6/9	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NS1	NS1	10.2	10.2	MILD NPDR	MILD NPDR	98	120	6	Nill	220	50	130	170	60	MED MAG