"DIABETIC RETINOPATHY CHANGES AND ITS AWARENESS AMONG PATIENTS ATTENDING DIABETIC EYE CAMPS IN KOLAR DISTRICT"

By

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SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

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In partial fulfillment
Of the requirements for the degree of

MASTER OF SURGERY IN OPHTHALMOLOGY

Under the Guidance of
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DEPARTMENT OF OPHTHALMOLOGY

SRI DEVARAJ URS MEDICAL COLLEGE

TAMAKA, KOLAR (MAY - 2018)

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

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I hereby declare that this dissertation entitled **"DIABETIC RETINOPATHY CHANGES AND ITS AWARENESS AMONG PATIENTS ATTENDING DIABETIC EYE CAMPS IN KOLAR DISTRICT"** is a bona fide and genuine research work carried out by me under the guidance of **DR. K.KANTHAMANI, M.S.** Professor and H.O.D. Department of Ophthalmology Sri Devaraj Urs Medical College Kolar, Karnataka.

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LIST OF ABBREVATIONS USED:

ABCD	Appropriate Blood pressure Control in Diabetes
ACE	Angiotensin Converting Enzyme
AGE	Advanced glycation end products
Anti VEGF	Anti vascular endothelial growth factor
ARMD:	Age Related Macular Degeneration
BCVA:	Best Corrected Visual Acuity
BMI:	Body Mass Index
BP:	Blood pressure
CF:	Counting fingers
CME:	Cystoid macular edema
CSME:	Clinically Significant Macular Edema
CURES:	Chennai Urban Rural Epidemiology Study
D:	Diet
DAG:	Diacylglycerol
DCCT:	Diabetes Control and Complications Trial
DM:	Diabetes Mellitus
DME:	Diabetic Macular Edema

DMI:	Diabetic Macular Ischemia
DR:	Diabetic Retinopathy
DRS:	Diabetic Retinopathy Study
E:	Exercise
ETDRS:	Early Treatment Diabetic Retinopathy Study
F:	Female
FA:	Fluorescein Angiography
FAZ:	Foveolar Avascular Zone
FBS:	Fasting blood sugar
FDA:	U.S Food and Drug Administration
FFA:	Fundus Fluorescein Angiography
FPG:	Fasting Plasma Glucose
G:	Grams
GAPDH:	Glyceraldehyde 3-Phosphate Dehydrogenase
HbA1C:	Glycosylated Hemoglobin
Hb:	Hemoglobin
HM:	Hand movements
HTN:	Hypertension

I: Insulin

ICMR: Indian Council of Medical Research

IDO: Indirect Ophthalmoscope

IRMA: Intra Retinal Microvascular Abnormalities

IVTA: Intravitreal triamcinolone acetonide

KAP: Knowledge, attitude and practice

LE: Left Eye

LP: Laser Photocoagulation

M: Male

ME: Macular Edema

mRNA: Messenger Ribonucleic Acid

mTOR: Mammalian Target of Rapamycin

NAD^+/NADH: Nicotinamide adenine dinucleotide

NADPH: Nicotinamide Adenine Dinucleotide Phosphate

NPDR: Non-proliferative diabetic retinopathy

NUDS: National Urban Diabetes Survey

NVD: Neovascularization of Disc

NVE: Neovascularization Elsewhere

NVG:	Neovascular Glaucoma
OCT:	Optical coherence tomography
OHA:	Oral Hypoglycaemic Agents
OPD:	Out Patient Department
PDR:	Proliferative diabetic retinopathy
PEDF:	Pigment epithelium-derived factor
PKC:	Protein Kinase C
PPBS:	Postprandial blood sugar
PRP:	Pan Retinal Photocoagulation
PVD:	Posterior Vitreous Detachment
RE:	Right Eye
SD:	Standard Deviation
sRNA :	Small Ribonucleic Acid
TGF:	Tumor Growth Factor
TNF:	Tumor Necrosis Factor
UDPN:	UDP-Nacetylglucosamine
UKPDS:	United Kingdom Prospective Diabetes Study
USG:	Ultra Sonography

VH:	Vitreous Haemorrhage
VEGF:	Vascular Endothelial Growth Factor
WESDR:	Wisconsin Epidemiological Study of Diabetic Retinopathy
WHO:	World Health Organisation

ABSTRACT

TITLE OF THE STUDY

"DIABETIC RETINOPATHY CHANGES AND ITS AWARENESS AMONG PATIENTS ATTENDING DIABETIC EYE CAMPS IN KOLAR DISTRICT."

NEED FOR STUDY:

Diabetic Retinopathy (DR) is a major cause of blindness among the working age group. According to WHO, India will become one of the major hubs of diabetic population over the next 2 decades, with the number of diabetics increasing from 18 million in 1995 to 80 million in 2030[1].It is estimated that diabetic retinopathy develops in greater than 75% of diabetic patients within 20 years from the onset of diabetes[2]. The prevalence of diabetic retinopathy in Karnataka is about 21.2%, in rural South India it is estimated to be around 10.5%[1] and globally 34.6%[2]. Since diabetes and its complications are a public health problem, data on the prevalence of DR will help in formulating primary and secondary prevention programs in India[1].

The knowledge, attitude and practice (KAP) studies conducted in rural South Indian population demonstrated that, only 37.1% had the knowledge about diabetic retinopathy[3]. In a study conducted in Karnataka in 2010, it was concluded that poor awareness and practices are some of the important variables influencing the development and progression of diabetes and its complications, which are largely preventable. Thus the knowledge of Diabetic retinopathy plays an important role in encouraging people to seek timely eye care .[4]

Hence this study was undertaken to detect the diabetic retinopathy changes and assess its awareness among the patients attending the diabetic eye camps in Kolar district, as

no study about this has been conducted in this area. Thus, we would also like to create awareness about DR and its effect on visual outcome, thereby encouraging regular screening so that early detection and timely intervention can be given to patients.

OBJECTIVES OF THE STUDY:

1. To examine and document the diabetic retinopathy changes among patients attending diabetic eye camps in Kolar district.
2. To assess the awareness of diabetic retinopathy and the factors influencing it, in patients attending the diabetic eye camps.

MATERIALS AND METHODS

250 patients attending diabetic retinopathy camps conducted in Kolar district were examined.

Informed written consent of the participating patients were taken. A pre structured proforma and questionnaire was used to collect baseline data. Every patients fundus was examined with an IDO or DO and the changes were documented. Those who required further evaluation were referred to our hospital.

The responses obtained were analyzed using Statistical Package for Social Studies (SPSS) and were grouped into excellent (≥75%), good (50 to 74%), poor (25 to 49%) and very poor (0-24%) based on their awareness score. They were also compared among epidemiologic variants.

RESULTS

Our study was conducted on 250 diabetic patients comprising of 149 male (59.6%) and 101 female (40.4%) patients attending diabetic eye camps conducted in Kolar district. Mean age of the patients was found to be 58.01± 14.28 years. Mean age

among males was 59.2 ± 14.9 years and among females was 56.3 ± 13.2 years.

Prevalence of diabetes among the study population was found to be 20.80 %.

Among Type 1 DM subjects, 9% had DR and among the Type 2 DM, 23.3% had DR, and this was found to be statistically significant. ($p = 0.03$). In our study, 17.35 % of those with diabetes of duration less than 10years had DR and 45.16 % of those with duration of more than 10years had DR. This was also found to be statistically significant ($p = 0.003$). Diabetic retinopathy patients comprised of 34 males and 18 females i.e. 65.4% of the patients were males, showing male preponderance. Prevalence of NPDR was 96.40% and PDR was 3.49%.

Among NPDR Patients, NPDR consisted of 45 eyes (52.33%) with mild NPDR, 32 eyes (37.21%) with moderate NPDR, 3 eyes (3.49%) with severe NPDR and 3 eyes (3.49%) with very severe NPDR. PDR was present in 3eyes (3.49%). CSME was seen in 17 eyes (20.48%).

Among type 1 DM, 26.6% had a positive family history and among type 2 DM 73.4% had family history. This difference in family history with respect to type of DM was statistically significant. ($p <0.001$).

In this study 11.2% had excellent, 24.8% had good, 9.6% had poor and 54.4% had very poor awareness regarding DR. We also found that 2.8% had good, 10.4% had poor and 86.8% had very poor awareness regarding treatment of diabetic retinopathy. When asked about attitude, 6.4% had excellent, 14.4% had good, 14% had poor and 65.2% had very poor attitude towards the disease. Our study also showed that , 30.4% had excellent, 21.6% had good, 6.4% had poor and 41.6% had very poor practices regarding diabetic retinopathy.

CONCLUSION

Diabetic retinopathy is a major health problem in patients with diabetes.

Risk factors such as age, duration of diabetes, hypertension, hyperglycaemia, hyperlipidaemia etc., should be taken into consideration for regular check-ups and early detection of diabetic retinopathy.

Awareness and knowledge about diabetic retinopathy was poor among the patients in our study. Lack of knowledge regarding the need for screening for diabetic retinopathy was found to be a major barrier to compliance with regular screening. Good knowledge about diabetic retinopathy was associated with a positive attitude towards diabetes and good practice patterns with respect to retinopathy. Therefore, there is an urgent need to develop strategies to educate diabetic patients about this potentially blinding complication of diabetes.

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INTRODUCTION

INTRODUCTION

Diabetes Mellitus currently affects more than 171 million people in the world and will affect approximately 366 million by 2030, with most rapid growth observed in the developing countries among populations of working age. It is estimated that, by 2030, the number of people affected with diabetes, in those more than 64 years of age, will be more than 82 million in developing countries and more than 48 million in the developed countries. The prevalence of diabetes among all age groups throughout the world was estimated to be 2.8% in 2000 and will rise to 4.4% in 2030. It is anticipated that the number of people with diabetes will double as a consequence of population aging and urbanization [1]. The International Diabetes Federation has estimated that about 314 million people will have impaired glucose tolerance and the number will increase to 472 million by 2030 [2]. India will be the country with maximum number of diabetics in the world by the year 2030. India now has 40.9 million people with diabetes mellitus and the projected estimate for the year 2030 is 80 million [3].

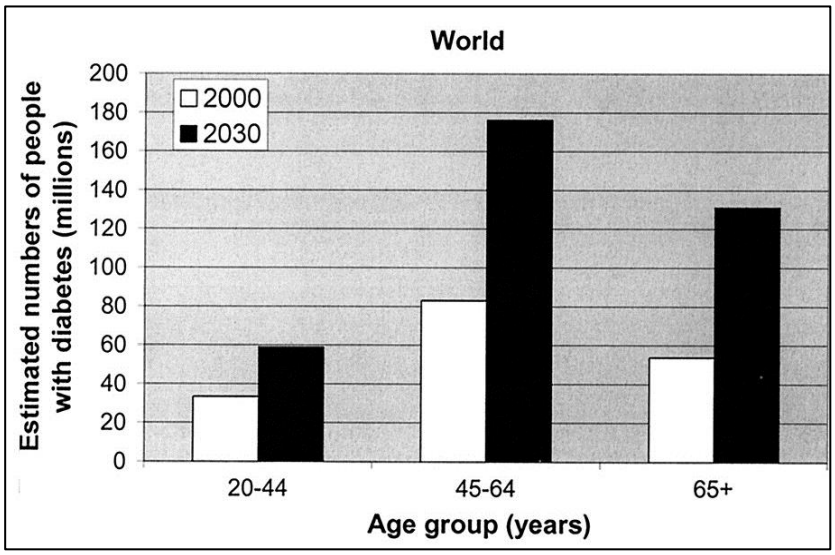


Figure 1:[1] Number of diabetics in 2000 and projected increase in 2030

Indians are reported to have a racial and genetic susceptibility to diabetes. Type2 diabetes occurs at a much younger age in Asian Indians when compared to Caucasians.[4, 5] An improvement in economic standards resulting in adoption of a more sedentary lifestyle has contributed to this epidemic, especially of Type 2 Diabetes.

The most common ophthalmic complication of diabetes mellitus is diabetic retinopathy. WHO has estimated that diabetic retinopathy is responsible for around 4.8% of 37 million cases of blindness throughout the world [6]. The incidence of blindness is 25 times greater in people with diabetes than in the general population [7]. In India, the estimate of the prevalence of diabetic retinopathy suggests that there are more than 5.6 million people with diabetic retinopathy with more than 0.3 million suffering from proliferative diabetic retinopathy.[8]

In India, there is marked difference in the prevalence of diabetes between urban and rural areas. The prevalence of diabetes in urban India was 12.1% in 2002 when compared to rural India, which had a prevalence of 6.4%. [9, 10]

Diabetic retinopathy develops in almost all people with Type I diabetes and in more than 77% of those with Type2 diabetes who survive more than 20 years of the disease [11]. Studies suggest that retinopathy progression almost does not exist in younger onset diabetics (less than 13 years-old). Theories suggest that puberty hormones might be a protective factor. Evidence shows that retinopathy begins to develop about 7-12 years before the clinical diagnosis of Type 2 diabetes [12].

There is direct correlation between the duration of diabetes and the prevalence and

severity of diabetic retinopathy. It is well established that longer the patients have diabetes, higher the prevalence of diabetic retinopathy [13, 14]. Studies of various complications in Indian diabetics is thus of great interest and not many studies are available on the prevalence of diabetic retinopathy from India [15].

The Knowledge possessed by a community refers to their understanding of any given topic, which in this case is, diabetes and diabetic retinopathy. Attitude refers to their feelings towards the subject, as well as any pre-conceived ideas that they may have towards it. Practice refers to ways in which they demonstrate their knowledge and attitude through their actions[16].

There is a paucity of data in the literature on awareness of diabetic retinopathy amongst the diabetic patients. In a study of an urban population in southern India, 2,522 subjects were asked whether diabetes could reduce vision. Only 28% were aware of such a possibility[16].

In case of diabetes, definite cure cannot be provided by medicines alone. It is well understood that diabetes management requires patient involvement for better disease control. Improving the awareness of the patients regarding the disease and drugs can improve the medication adherence behavior, which in turn improves the therapeutic outcomes[17].

There is always a need to investigate knowledge, attitude and behavior among diabetic patients to help in future development of national health programs and techniques for effective health education[18].

AIMS & OBJECTIVES

OBJECTIVES OF THE STUDY:

1. To examine and document the diabetic retinopathy changes among patients attending diabetic eye camps in Kolar district.
2. To assess the awareness of diabetic retinopathy and the factors influencing it, in patients attending the diabetic eye camps.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The Indian Council of Medical Research (ICMR, New Delhi) conducted the first national study on the prevalence of Type 2 diabetes in India between 1972 and 1975. The prevalence was 2.1% among the urban population and 1.5% in the rural population while in those above 40 years of age, the prevalence was 5% in the urban and 2.8% in the rural areas.[19]

Epidemiological studies conducted in different parts of the country showed a prevalence of diabetes varying from 5.4% in North Eastern states to as high as 15.5% in the South Indian state of Tamilnadu.[20]

A study conducted in S.P. Medical College, Bikaner, on the prevalence of diabetic retinopathy in type 2 diabetes highlighted the high prevalence of diabetic retinopathy in type 2 diabetes in India. This study was carried out on 4069 subjects and it showed evidence of diabetic retinopathy in 28.9% of patients. This comprised of about 79.8% cases of non-proliferative diabetic retinopathy, 5.8% cases of maculopathy and 14.6% cases with proliferative diabetic retinopathy. Multiple logistic regression analysis showed that age of the patient, duration of diabetes, systolic blood pressure and glycosylated Hb had positive contributions.[21]

The prevalence of DR was higher in men than in women. This study showed that the prevalence of diabetic retinopathy was the least in urban South Indians than in other ethnic groups. The risk for development of DR increases with every 5-year increase in the duration of diabetes, 1.89-fold.[22] In a study by Gupta and Ambade in Central India revealed that 34 % of Type II diabetics had DR. About 81% of subjects with diabetic nephropathy (albuminuric diabetics) had DR. In subjects with duration of diabetes <5

years had 22.9% of DR, while those > 5 years had 53.5% DR. The prevalence as per the severity of DR, 27.7% had mild NPDR, 6% had moderate to severe NPDR, 3% had PDR and CSME was seen in 17.9% of Diabetic retinopathy[23]

The Aravind Comprehensive Eye Study reported the prevalence of DR (in self-reported subjects with diabetes) in rural South India to be 10.5%.[24] The National Urban Diabetes Study (2000) showed the prevalence of diabetes in a population older than 40 years to be 23.8% in 6 cities in India[25].

The Sankara Nethralaya Diabetic Retinopathy Epidemiology and Molecular Genetic Study (SN-DREAMS 1), a population-based cross-sectional study, used multistage random sampling that was stratified, based on economic criteria[26].

Studies conducted in Karnataka showed the prevalence of DR to be 21.2%, whereas that of the South Indian population as a whole was 18%[26].Other global studies show that the overall prevalence was 34.6% for diabetic retinopathy. It was concluded that diabetic retinopathy prevalence increases with duration of diabetes, hemoglobin A1c, and blood pressure levels[27].

P K Rani, R Raman et al., in a study to assess the knowledge of diabetes and diabetic retinopathy and the influence of knowledge of diabetic retinopathy on attitude and practice in 2008 among rural South Indian population, observed that, only 37.1% had the knowledge about diabetic retinopathy, of which, the majority were women, literates or those belonging to upper strata of the society[28].It was concluded that spreading knowledge of diabetes and related complications will motivate individuals to visit ophthalmologists which is an important step in preventing diabetes- related blindness[28]

A study on KAP of ocular complications due to diabetes among type II diabetics visiting a tertiary teaching hospital conducted by Jacob Koshy, et al., in Ludhiana in 2012 concluded that, with an increase in the duration of illness, the awareness about diabetic retinopathy is more. 51.4% of the diabetics did not know how diabetes can affect the eye, 49.7% of diabetics did not know if diabetic retinopathy can be treated and 67.4% had not heard of any treatment modality for diabetic retinopathy. This shows that lack of knowledge about the disease was significant[29].

A study conducted by Dandona R, Dandona L et al., to assess the level of awareness of eye diseases in the urban population of Hyderabad brought out that patients belonging to the upper and middle socio economic strata were more likely to be aware regarding diabetic retinopathy[16] and another study showed that the level of knowledge and practices varied greatly with reference to educational level and there was also lack of understanding about importance of diet and lifestyle.[30]

Studies have shown that a majority of people belonging to the lower socioeconomic have minimal awareness about the etiology, features, prognosis and diagnosis of diabetes. In this group of people even those who had been diagnosed with diabetes had minimal knowledge about the complications of the condition[31].

HISTORY:[32]

Laurence and Hays noted the characteristic retinal disorders in a standard textbook that was written prior to the invention of the ophthalmoscope, but diabetes was not recognized as the causal factor.

In 1855, Jaeger described the first case of diabetic retinopathy, which was a few years after the invention of an ophthalmoscope.

Noyes in 1868,described small retinal hemorrhages and yellow exudates in the fundus of a 60 year old woman having diabetes of three-year duration.

In 1873, Haltenhoff described a similar case with hemorrhages but no exudates. Manz reported three patients with extensive proliferative changes in 1876.

In 1879, McKenzie published a report on diabetic retinopathy.

Between 1882-1888, Nettleship presented a case series of diabetes. He described the venous changes, background retinopathy, neovascular glaucoma and proliferative retinopathy, though these proliferative changes were not recognized as a separate entity.

In 1890-1891, Hirschberg presented the first comprehensive classification of diabetic retinopathy.

Dodd reviewed the literature and reported 47 cases of diabetic retinopathy, all of which had diabetes and albuminuria, in 1895.

In 1920, Weigener and Wilder studied a large number of cases with diabetes and concluded that diabetic retinopathy was almost always associated with other vascular and renal disorders.

In 1928, Salus describe the condition of rubeosis iridis.

In 1934, Wagener, Dry and Wilder studied diabetic cases and noted that Proliferative retinopathy was more frequent than generally realized.

In 1939, Bedell showed that venous irregularities were a common feature of retinopathy. In 1935 Waite and Beetham, Duke Elder in 1941 and Wagener in 1945 showed the increasing incidence of diabetic retinopathy with increase in duration of diabetes. Ballentyne and Lowenstein rediscovered showed that fatty infiltration of the endothelial lining of the vessels leads to the formation of microaneurysms in 1943.

In 1945, Lowenstein reported venous dilatation without any other changes in juvenile diabetics of 5 to 15 years of age.

Ballantyne and Michaelson in 1948 reported that the characteristic retinal changes are seen primarily in the veins and capillaries.

In 1960, Cogan, Toussiant and Kuwabara introduced the types of digestion techniques for the study of the histopathological changes of the retina. Thus the 'mural cell' or 'pericyte' was discovered by this technique, in 1961.

In 1961, Novotny and Alvis introduced fluorescein angiography by which the details of diabetic retinopathy were better studied.

CLASSIFICATION OF DR

Hirschberg Classification[33]

Hirschberg made the first attempt on classification in 1890, which recognized three clinical types: inflammatory - characterized by spots and hemorrhages, (central punctate diabetic retinitis), hemorrhagic and pigmentary

Ballantyne and Lowenstein Classification

The five stages in his classification are

1. Micro-lesions comprising of either microaneurysms alone or accompanied by minute hemorrhages and punctate exudates.
2. Macro-lesions like dot and blot hemorrhages
3. Vascular changes and gross changes in retinal veins
4. Destructive changes, intra ocular hemorrhages, retinitis proliferans, detachment of retina and glaucoma
5. Mixed forms Diabetic changes associated with arteriosclerosis and/or hypertension.

Scott's classification of Diabetic Retinopathy [34]

Stage 1a	Capillary microaneurysms
Stage 1b	Changes in the larger veins such as phlebosclerosis, loop, coils, or knots in the distended veins.
Stage 2a	Punctuate hemorrhages with or without discrete flecks of exudates
Stage 2b	Larger round or "blot" hemorrhages with confluent exudates.
Stage 3a	More numerous hemorrhage and exudates
Stage 3b	Hemorrhages into the vitreous.
Stage 4	Reinitis Proliferans. Detachment of the retina. Gross degenerative

VAHEX Classification[35]

This classification mainly depends on the ophthalmoscopic appearance and dynamic factors affecting the ultimate prognosis of any case of diabetic retinopathy. It also helps in the treatment of the disease.

Here it is divided into –

- Background retinopathy.
- Proliferative retinopathy.

The stages of background retinopathy may be remembered by the anonym –

V - Venous dilation

A – Microaneurysms

H - Hemorrhages

E - Edema

X - Exudates

AIR LIE Classification of Diabetic Retinopathy[36]

According to this classification diabetic retinopathy is divided into the following four lesions:

Non proliferative:

This includes haemorrhages, hard exudates, soft exudates, venous abnormalities, and intraretinal microvascular abnormalities and retinal oedema at the macula.

Fluorescein:

Fluorescein angiography helps in grading non-proliferative stage by dye leak in arterio-venous phase and late phase.

Proliferative lesions:

Comprising neovascularisation within one disc diameter of the disc and anywhere other than the disc.

Vitreous haemorrhages:

It is grouped into pre-retinal haemorrhages, vitreous haemorrhage and history of vitreous haemorrhages. Each lesion is further graded into

Grade 0- none

Grade I- mild to moderate lesion

Grade II- moderate to severe

Thus picture of diabetic retinopathy is grouped into particular lesions and graded by comparing it with standard records.

DUKE-ELDER Classification:[37]

Pre-retinopathy stage: Characterized by venous fullness A: V ratio changes form 2:3 to 2:4

1. Simple diabetic retinopathy

Characterized by

a) Microaneurysms

b) Retinal haemorrhages

- Superficial
- Deep

c) Exudative lesion

- Hard
- Soft

d) Late changes in retinal veins

2. Proliferative Phase:

Stage I - Stage of naked vessels

Stage II - Stage of condensation of connective tissue around the naked vessels.

Stage III - Stage of cicatrisation.

ETDRS CLASSIFICATION

I) NON-PROLIFERATIVE DIABETIC RETINOPATHY (NPDR):[38, 39]

A) Mild NPDR:

- At least one micro aneurysm
- Definition not met for B,C,D,E,F

B) Moderate NPDR :

- Haemorrhages/ micro aneurysm ≥ standard photograph No.2A
- Soft exudates; venous beading and IRMA are definitely present to a mild degree.
- Definition not met for C, D, E, F

C) Severe NPDR [4-2-1 rule] :

- Characterized by Any 1 of the following:
- Haemorrhages / micro aneurysms ≥ standard photograph No.2A in all 4 quadrants.
- Venous beading in 2 (or) more quadrants.
- IRMA > standard photograph 2A is at least 1 quadrant.

D) Very severe NPDR

- Any 2 (or) more of C
- Definition not met for E, F.

II) PROLIFERATIVE DIABETIC RETINOPATHY (PDR):[39]

- Composed of:
- NVD (or) NVE
- Pre retinal (or) vitreous haemorrhage
- Fibrous tissue proliferation.

A) Early PDR

- New vessels
- Definition not met for F

B**) High risk PDR**

- NVD >1/3 disc area
- NVD and vitreous (or) pre-retinal haemorrhage.
- NVE ≥ 1⁄2 disc area and vitreous (or) pre retinal haemorrhage.

C) Advanced Diabetic Eye Disease

- Tractional retinal detachment
- Tractional Retinoschisis
- Rubeosis iridis
- NVG

III) DIABETIC MACULOPATHY: [40, 41]

- Focal
- Diffuse
- Ischemic
- Mixed
- Clinically significant macular edema.

Diabetic macular edema can be associated with any of the stages of retinopathy [NPDR/PDR].

1) Focal Maculopathy: [40]

- It is characterized by well circumscribed retinal thickening associated with complete (or) incomplete rings of hard exudates.
- FA shows late, focal hyperfluorescence due to leakage from micro-aneurysm often with extra vascular lipoprotein in circulate pattern around them.
- Focal maculopathy will have good macular perfusion.

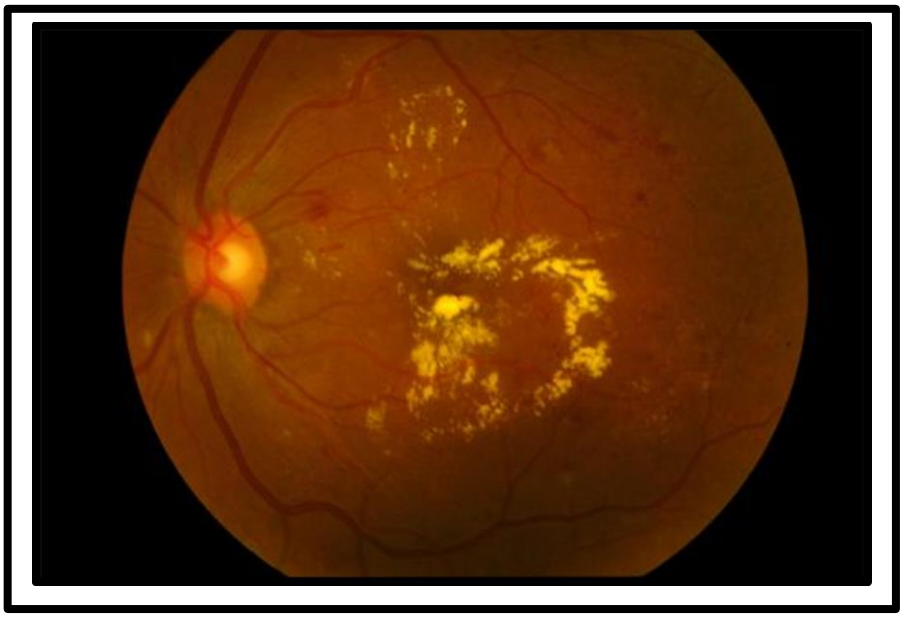


Figure 2 A: CIRCINATE RETINOPATHY

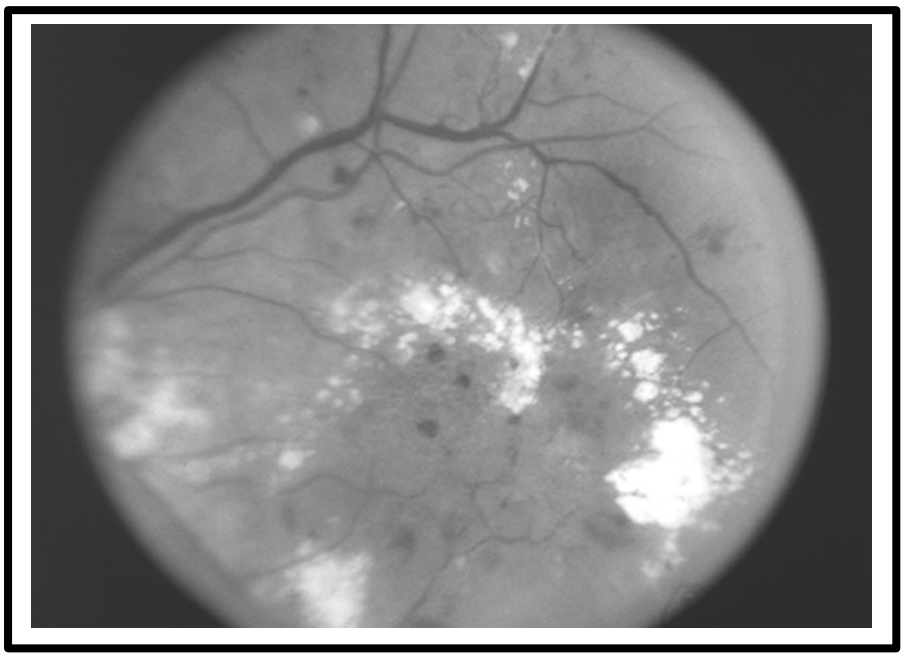


Figure 2 B: FFA- CIRCINATE RETINOPATHY

[40]

2) Diffuse Maculopathy:

- It is characterized by diffuse retinal thickening, which may be associated with cystoid changes.
- Because of severe edema, landmarks are obliterated which may render localization of the fovea impossible.
- It results from generalized breakdown of blood retinal barrier resulting into profuse leakage from entire capillary bed into the posterior pole.
- FA shows late diffuse hyperfluorescence and which may assure a central flower petal if CME is present.[40]

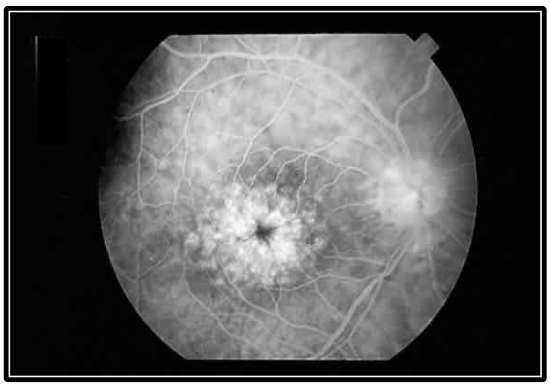


Figure 3 A. FFA - CME

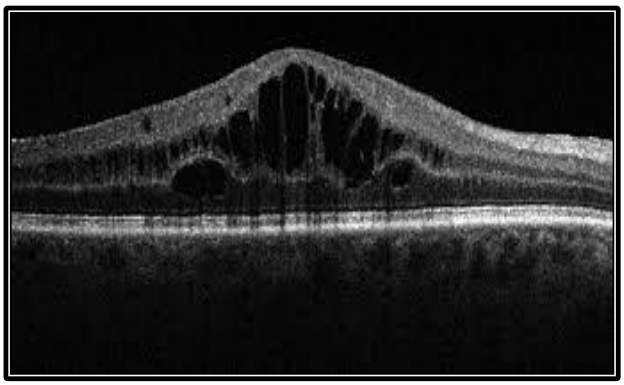


Figure 3 B. OCT- CME

CYSTOID MACUALR EDEMA

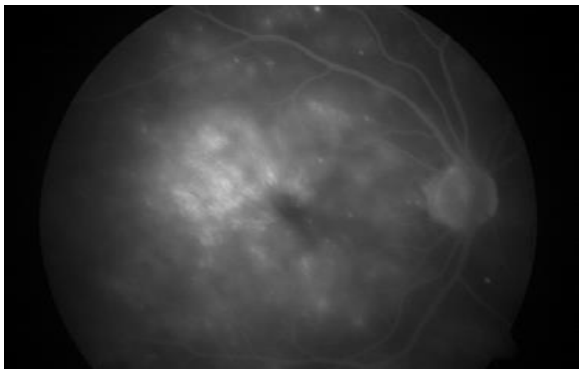


Figure4. DIABETIC MACULOPATHY

[40]

3) Ischemic Maculopathy:

- There is enlargement of FAZ due to capillary closure

- It is characterized by the presence of reduced levels of visual acuity disproportionate to the clinical appearance of macula. (signs are variable and macula may look relatively normal, despite reduced VA).

- FA shows it is very much important to detect as treatment differs from other types. Photocoagulation is contraindicated – capillary non-perfusion at fovea and frequently other areas of capillary non-perfusion at the posterior pole of periphery.[40]

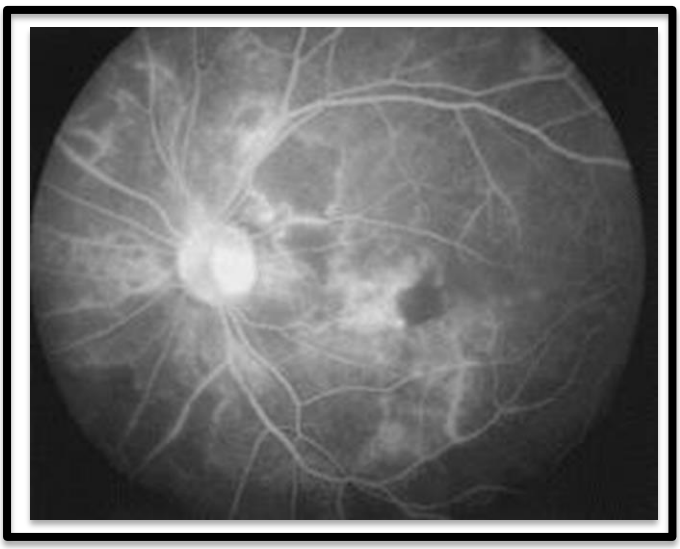


Figure 5.Capillary closure and area of focal leakage in ischemic maculopathy

Ophthalmoscopic signs of significant macular ischaemia include

- Large dark retinal hemorrhages.
- "Cloudy swelling" of the retina.
- Multiple cotton wool spots
- Small, white threadlike arterioles in the macular region

4) CLINICALLY SIGNIFICANT MACULAR EDEMA/(CSMO)/(CSME): [42,43]

- The International Clinical Diabetic Macular Edema Disease Severity Scale summarizes

- Clinically Significant Macular Edema as

- Retinal thickening at or within in 500μm of the center of macula.

- Hard exudates at or within 500μm of the center of macula and associated with retinal thickening.

- Retinal edema one disc area (1500μm) or larger, any part of which is within one disc diameter from the center of the macula.

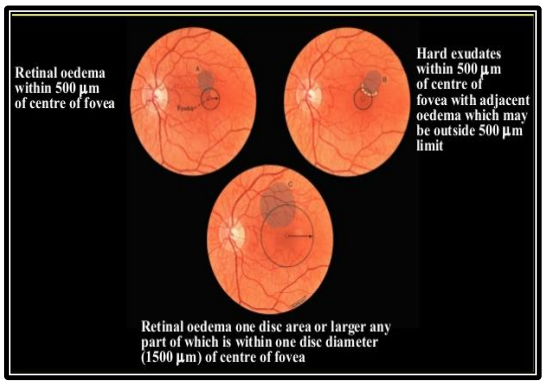


Figure 6A : CSME

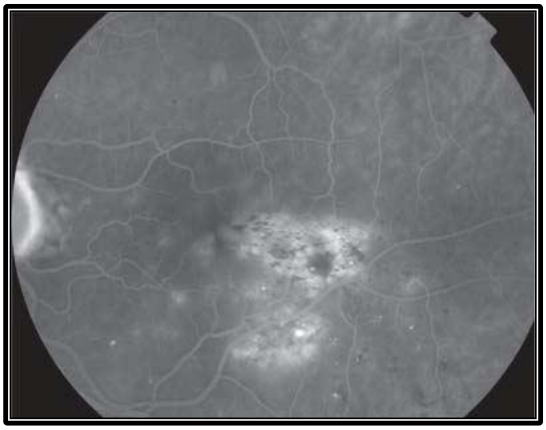


Figure 6 B :CSME- FFA

5) MIXED MACULOPATHY:

- Ischemic with cystoid macular edema

I. PATHOGENESIS: BIOCHEMICAL BASIS FOR DIABETIC RETINOPATHY [44]

In the early stages of diabetes mellitus, chronic hyperglycemia leads to blood flow alterations and increase in vascular permeability.

This is characterized by decrease in the activity of vasodilators such as nitric oxide along with increase in activity of vasoconstrictors such as angiotensin II and endothelin-1 with the release of vasopermeability augmenting cytokines such as VEGF.

Hyperglycemia decreases the production of neuronal cell trophic factors and endothelial factors resulting in ischemia, edema, and hypoxia-driven neovascularisation[45].

Four hypotheses have previously been proposed to explain the mechanism of hyperglycemia-induced microvascular changes:

1. Increased polyol pathway flux
2. Advanced glycation end products (AGEs)
3. Activation of protein kinase C (PKC)
4. Increased hexosamine pathway flux.

Inhibitors of aldose reductase, AGE formation, PKC activation, and the hexosamine

pathway each prevent various diabetes-induced abnormalities, but no apparent common element was noted until the recent discovery that each of these causes overproduction of superoxide by the mitochondrial electron-transport chain.[46]

It has been proved that both diabetes and hyperglycemia increase oxidative stress

1. Increased polyol pathway flux:[44]

This pathway converts hexose sugars (like glucose and galactose) into sugar alcohols (polyols) e.g. glucose is converted into sorbitol, galactose to dulcitol via the action of enzyme aldose reductase. Sorbitol can be oxidized to fructose although this conversion occurs very slowly. The rate limiting enzyme for this pathway is Aldose reductase. Normally aldose reductase has low affinity for glucose and in hyperglycemia glucose is principally metabolized by hexokinase (the first enzyme in glycolytic pathway). But in hyperglycemia high intracellular glucose levels saturate hexokinase, and the proportion of glucose converted by the polyol pathway increases. It is suggested that this increase flux through polyol pathway plays a significant role in the pathogenesis of diabetic retinopathy.[47]

Enhanced activity of aldose reductase and sorbitol accumulation has been reported in the retina of diabetic animals. Since sorbitol does not diffuse easily across cell membranes, it can significantly increase the intracellular tonicity and cause cellular damage by an osmotic process. Further, the increased polyol pathway activity alters the redox state of pyrimidine nucleotides, NADPH and NAD+. As these are co-factors in many enzyme catalyzed reactions, other metabolic pathways may also be affected. In retinal capillaries, the highest level of aldose reductase is found in pericytes which leads to loss of autoregulation of

retinal capillaries[48].

Electrolyte imbalance caused by high intracellular aldose reductase levels leads to cell death, especially in retinal pericytes, which cause microaneurysm formation. Increased retinal leucostasis has been reported to causes capillary occlusions and dropout, nonperfusion, endothelial cell damage and vascular leakage as a result of its less deformable nature. Retinopathy thus exhibits features of both microvascular occlusion and leakage [49].

This leads to increase in the formation of methylglyoxal – a precursor of AGEs – and diacylglycerol, thus activating PKC.

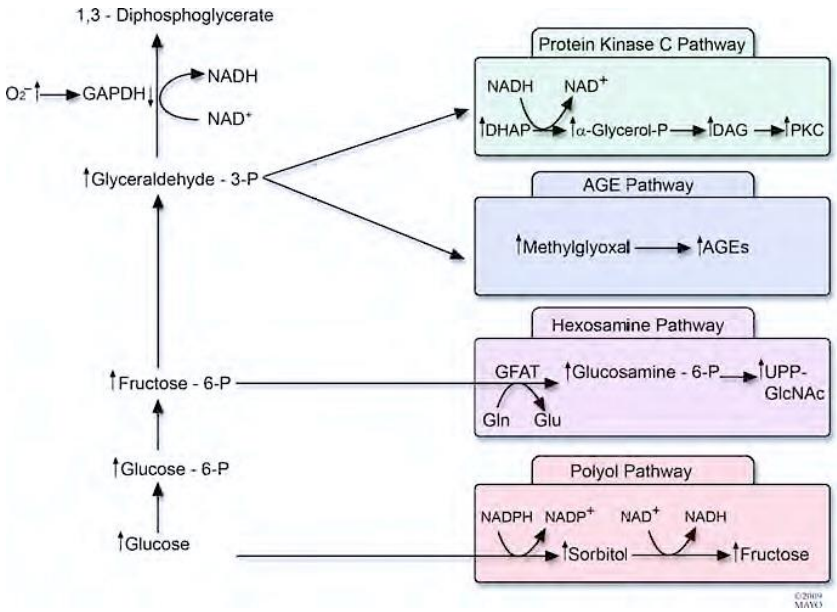


Figure7[44]

This schematic shows the mechanism by which superoxide production in the mitochondria activates the four biochemical pathways that lead to diabetic retinopathy. Hyperglycemia induced superoxide (O2) production inhibits GAPDH,

causing an accumulation of upstream metabolites. These are diverted into the four alternative metabolic pathways, each of which leads to vascular and interstitial tissue damage.

2. Advanced glycation end products (AGEs)[44]

A consequence of hyperglycemia there is formation of modified proteins known as glycation end products.[50] They are formed non-enzymatically via a series of intermediate steps. This process is initiated by the attachment of glucose to the amino groups of proteins, to form a Schiff base, which quickly undergoes a chemical modification known as Amadori rearrangement, to produce early glycation products. One of the best-known examples of such a modified protein is glycosylated hemoglobin (HbA1c) which is monitored clinically as an index of glycemic control. Early glycation products thus formed can combine irreversibly with each other to form cross-linked proteins known as "Advanced Glycation End

Products" (AGE). These molecules are stable and are long lived, and therefore do not return to normal once hyperglycemia has been controlled. AGE modification of proteins can occur both intracellularly and extracellularly, leading to an alteration in their functional properties. Many studies have linked

AGE formation to the pathological changes that are seen in diabetic angiopathy via several possible mechanisms[50]:

- AGE crosslinking induces an expansion of the molecular packing of collagen type1, thereby altering the function of vessels[51]. AGE-modified collagen and laminin (which are important components of basement membrane) are less susceptible to proteolytic degradation. Accumulation of these abnormal

proteins are thus responsible for the basement membrane thickening that is seen in early diabetic retinopathy. The glycation of basement membrane matrix of cultured cells has been shown to affect their proliferation as well.[52]

- AGE formation within the endothelial basement membrane inactivates endothelial- derived nitric oxide, which normally acts on perivascular smooth muscles causing vasodilatation. This results in impaired blood flow[53].

- Many cells including vascular endothelial cells, possess receptors for AGE. Binding of AGE to the endothelial cell receptor causes a change in the vascular permeability and favors thrombosis at the endothelial cell surface[53].

- Pharmacological inhibition of AGE formation in experiments using the drug aminoguanidine offers strong support for involvement of glycation end products in the pathogenesis of diabetic retinopathy[53].

- Several cell-associated-binding proteins for AGEs have been identified: OST-48, 80K-H, galectin-3, macrophage scavenger receptor type II, and RAGE. They mediate the long-term effects of AGEs on macrophages, glomerular mesangial cells, and vascular endothelial cells.

- Thus macrophages and mesangial cells cause the expression of cytokines and growth factors (interleukin-1, insulin-like growth factor I, tumor necrosis factor-a, TGF b)[54]

3. Activation of protein kinase C (PKC) [44, 58]

- Protein kinase C is a family of 11 isoforms, 9 of which are activated by diacylglycerol (DAG).

- Intracellular hyperglycemia causes an increase in DAG in the retina which results in the activation of PKC in vascular cells.[55]

- Hyperglycemia indirectly activates PKC isoforms through the ligation of AGE receptors and via increase in the activity of polyol pathway[56, 57].

- Activation of PKC-b isoform mediates the retinal and renal blood flow abnormalities by decreasing the nitric oxide production, increasing endothelin-1 activity and induces expression of VEGF in smooth muscle cells.

4. Increased hexosamine pathway flux[44]

- An excess in the activation of the hexosamine pathway causes changes in gene activation, which is known to lead to vascular endothelial dysfunction.

- Increase in the intracellular glucose in the form of fructose-6-phosphate is diverted from glycolysis to provide substrates for reactions requiring UDP N-acetyl glucosamine.

- This leads to increase in the synthesis of glycolipids, glycoproteins, proteoglycans, and TGF-b.

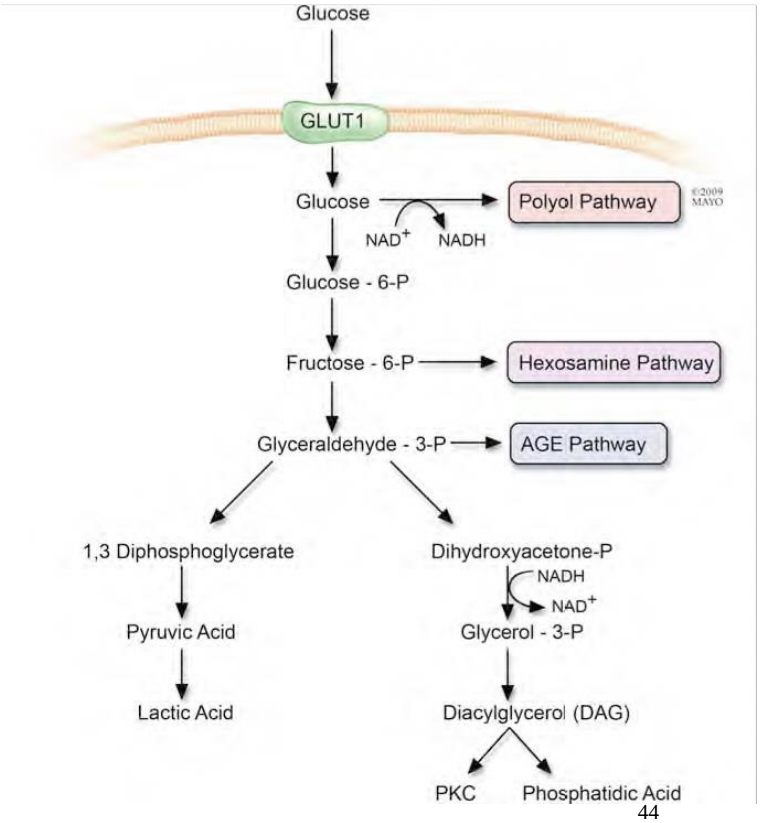


Figure 8[44]

Hyperglycemia induced superoxide production prevents the normal conversion of glyceraldehyde-3-P into 1,3 diphosphoglycerate. This diverts upstream metabolites into the polyol, hexosamine, and AGE pathways. Excess glyceraldehyde-3-P is converted into diacylglycerol (DAG), which subsequently activates protein kinase C (PKC)[44]

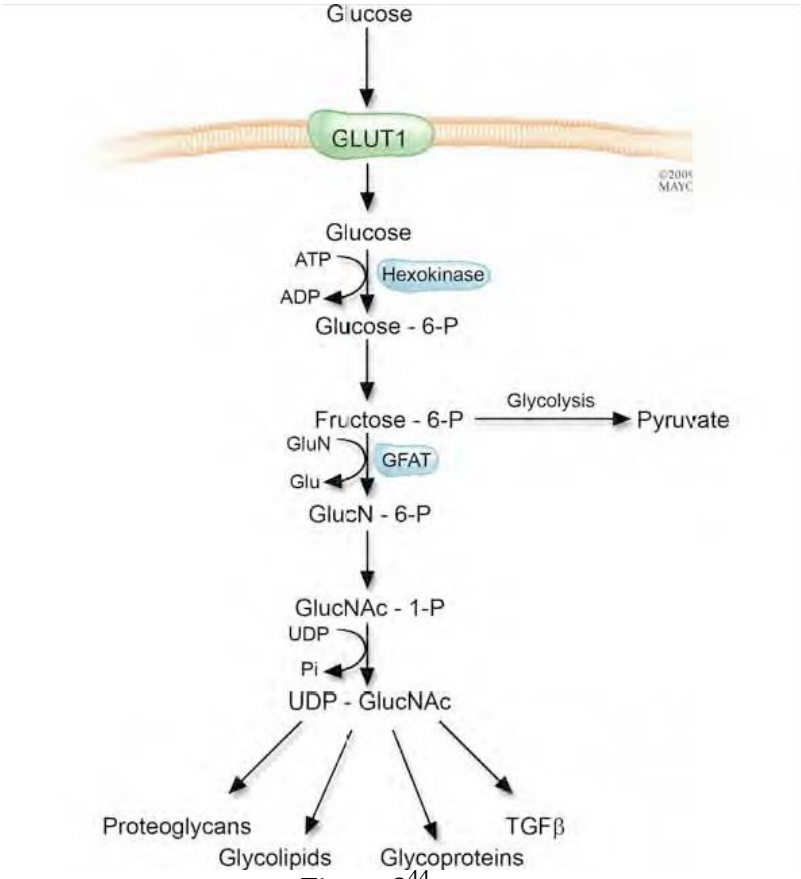


Figure 9[44]

The hexosamine pathway produces glucosamine-6-P by diverting excess fructose-6-P from glycolysis. This leads to the synthesis of glycolipids, glycoproteins, proteoglycans, and TGF-b[44]

PATHOLOGY OF DIABETIC RETIONPATHY[56]

It is most likely that a number of processes are operating simultaneously. Early changes include the obstruction of capillaries and retinal arterioles, which forms the fundamental aspect of the change. This obstruction results in ischemia of the retina, which in turn leads to neovascularization. These changes also stimulate the venous changes and formation of micro aneurysms.

MECHANISM OF VESSEL CLOSURE[59]

In diabetic retinopathy the possible pathogenic mechanism for capillary and arteriolar closure can be divided into three categories.

- Intraluminal
- Intramural
- Extramural

Intraluminal:

It is mainly due to abnormalities in the blood elements, which promotes thrombosis within the small retinal vessels, leading to decreased oxygen transport[59].

Intramural:

These include changes in the endothelium and basement membrane.

The possible intramural factors include, loss of pericytes, proliferation of endothelium, progressive thickening of basement membrane and thickening of endothelium. Changes that occur in states of hyperglycemia due to accumulation of sorbitol, can be contributory to the vessel closure.[60]

Other probable intramural factors include plasma insudation through an abnormal vessel wall, causing thickening of the vessel wall. This is increased in presence of systemic hypertension[59].

Extramural: [59, 61]

The extramural factor proposed is compression of the vessel wall by accumulated retinal edema. As a result of retinal capillary non-perfusion, retinal ischemia develops, which is initially in the mid retinal periphery. The two main effects of retinal hypoxia are:

1. Arteriovenous shunts, which are associated with capillary occlusion, run from arterioles to venules. Since it is unclear whether these lesions represent new vessels or opening of preexisting vascular channels, they are often referred to as 'intraretinal microvascular abnormalities' (IRMA).

2. 'Vasoformative substances' that are produced by hypoxic retinal tissue in an attempt to revascularize the hypoxic retina can cause Neovascularization. These substances stimulate neovascularization of the retina, the optic nerve head and occasionally on the iris (rubeosis iridis). Of the many growth factors that have been identified, vascular endothelial growth factor (VGEF) appears to be the most important.

THE CAPILLARY MICROANEURYSMS

This theory was explained by McKenzie and Nettleship in 1879 and confirmed by Ballantyne in 1943. It is a characteristic feature but is not pathognomonic because, it is also seen in conditions like branch retinal vein occlusion, dysproteinaemias and hypertensive retinopathy. The mechanism of formation of microaneurysms in diabetic patients:

The degeneration of pericytes weakens the capillary wall causing a saccular out pouching at the site of degeneration. Microaneurysms usually appear in clusters and surround the area of capillary occlusion. On ophthalmoscopic examination it is

difficult to visualize the small microaneurysms. They vary from 20 microns to 120 microns in size and it is difficult to appreciate microaneurysms of less than 30 microns with a direct ophthalmoscope. They appear as bright red, well-defined globular forms mostly at the posterior pole and are bounded by superior and inferior temporal arteries, where most of the changes are usually seen. They may be seen singly or in clusters. They originate from the venous side of circulation and are easily diagnosed by fluorescein angiography. The evolution cycle of a microaneurysms begins with proliferation of capillary endothelial cells. It then becomes thick walled. In later stages it gets hyalinised and obliterated.

Microaneurysms are normally lined by endothelium and thin basement membrane. Later it undergoes hyalinization due to deposition of PAS- positive material. F luorescein angiography shows staining of the wall of the aneurysms or leakage of the dye from the wall. They appear as bright fluorescent spots. There may be leaky junctions between the endothelial cells, which show leaks in fluorescein angiography. Microaneurysms are very significant in diabetic retinopathy for several reasons. Firstly they are the earliest structural alterations of the retinal blood vessels that are visible with the ophthalmoscope. Secondly, in the earliest stages increase or decrease in number of microaneurysms can be used as an indication of progression or regression. Lastly, leakage from microaneurysms is an important cause of retinal edema that may be diffuse or localized.

RETINAL HAEMORRHAGES [59,63]:

Intraretinal hemorrhages may appear as a result of ruptured microaneurysms, capillaries and venules. As in t h e other retinal vascular diseases, hemorrhages may assume several morphologic forms, depending on its depth within the retina.

Dot hemorrhages with distinct borders may be clinically indistinguishable from microaneurysms and blot hemorrhages with fuzzier borders are located deep within the outer plexiform and inner nuclear layers. Flame shaped hemorrhages are superficial in the nerve fiber layer. The source of retinal hemorrhages is usually from fragile capillaries or from thin walled microaneurysms. Some retinal hemorrhages in diabetes may show a white center either because they contain fibrin and platelets or because the hemorrhage may have originated from a partially hyalinized microaneurysm.

Retinal hemorrhages in diabetes are significant because their severity parallels with the severity of background retinopathy in general. Of particular importance is the presence of multiple large, dark blot hemorrhages, that occur with arteriolar occlusion, and as one of the components of the pre-proliferative stages of retinopathy, they indicate an increase in the risk of neovascularization. Generally, intraretinal hemorrhages do not cause significant visual disturbance, but occasionally visual acuity may be decreased as a result of a foveolar hemorrhage; the acuity can improve with the resolution of foveolar hemorrhage.

CAPILLARY OBSTRUCTION:

The perifoveal capillary bed is an area in the fundus in which capillary closure in diabetic retinopathy can be most easily demonstrated, especially by fluorescein angiography. This is maybe due to several factors.

- The increase in melanin in the underlying pigment epithelium is densest in the fovea, thereby enhancing contrast between retinal capillaries and background fluorescence.
- The retinal capillary bed is normally reduced to a single layer in the immediate

perifoveolar region thereby increasing the visibility of capillary bed abnormalities.

- The most reliable signs of foveal avascular zone (FAZ) abnormalities in diabetes include irregularity of the FAZ margins, capillary budding into and widening of the inter capillary spaces in the perifoveolar capillary bed. Many eyes that have mild and moderate enlargement of FAZ can maintain normal vision. Hence there is probably a "reserve" of retinal circulation to the central macula. Once the longest FAZ diameter exceeds 1000 microns the visual acuity is reduced probably due to macular ischemia.

RETINAL VENOUS ABNORMALITIES[59, 64, 65]

These include:

- Dilatation
- Beading
- Reduplication
- Looping, Kinking
- Branch retinal vein occlusion
- Central retinal vein occlusion.

Generalized dilatation is one of the earliest changes. It is considered as one of the features of established diabetic retinopathy. Beading and sausaging of venous segments may lead to local ischemia. Histology demonstrates the proliferation of endothelial cells.

Duplication of a vein may occur. There is an increase in the size of the new vessel, gradually more than the original vein.

Typical branch retinal vein occlusion (BRVO) or central retinal vein occlusion (CRVO) may develop in patients with diabetes mellitus, with a greater incidence when compared to the non diabetic population. The only fundus abnormality present may be signs of venous occlusion, but usually they are superimposed upon the fundus changes of diabetic retinopathy. Thus, the diagnosis of venous occlusion can be difficult in a diabetic patient because diabetic retinopathy and venous occlusion sometimes produce similar fundus pictures. However, the distribution of the lesions and the mode of onset help in distinguishing between the two. Venous occlusion is usually more sudden than diabetic retinopathy. With BRVO, the fundus lesions are usually most severe in the distribution of the occluded branch. In CRVO, the fundus lesions may involve all the quadrants similar to diabetic retinopathy. But there is more asymmetry between the eyes when compared to diabetic retinopathy alone.

HARD EXUDATES[59,66]:

Hard exudates are also known as "edema residues", and are formed due to leakage of plasma lipoprotein and lipids through abnormally permeable retinal capillaries and micro aneurysms. These are usually located within the outer plexiform layer. They can be seen in three forms.

1. Cluster of small deposits.
2. Ring or Circinate arrangement
3. Large waxy plaques.

The stimulating factor is usually a hemorrhage or leaked plasma, which attracts the macrophages, that diffuse through the tissues and engulf the lipid and lipoproteins and are left behind once the edema subsides. As the interface between the ischemic and relatively normal areas is circular, the deposition of hard exudates is also circular. Greater the zone of ischemia, larger the size of the circle.

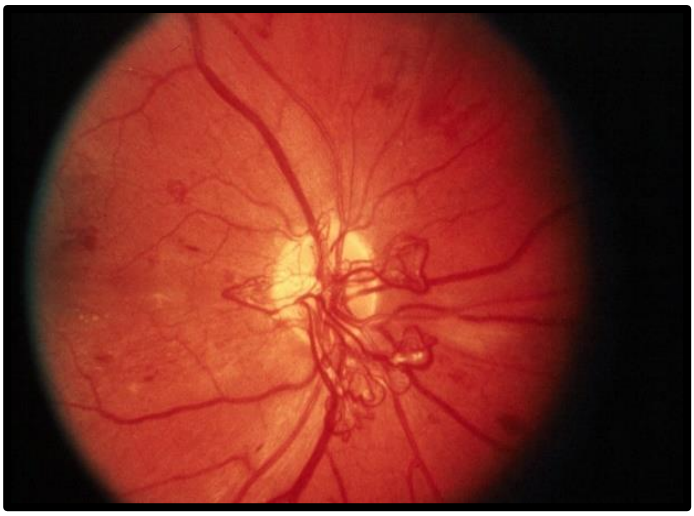
SOFT EXUDATES[59]:

They are also known as cotton wool spots and are secondary to nerve fibre layer infarct. They appear as fluffy white or yellow-spots. They show early blockage of dye on fluorescein angiography. Generally resolve in 2-3 months.

PROLIFERATIVE PHASE [59,67,68,69]

This phase is characterized by the growth of fibrous, glial and neovascular tissue in response to ischemia. It can be associated with vitreous changes like vitreous contraction, detachment of posterior hyaloid phase and thickening of posterior hyaloid membrane. In such conditions the visual symptoms are mostly due to vitreous hemorrhage, vitreous opacities due to fibrous and glial proliferation, or due to vitreous detachment.

Figure 10 A-NVD



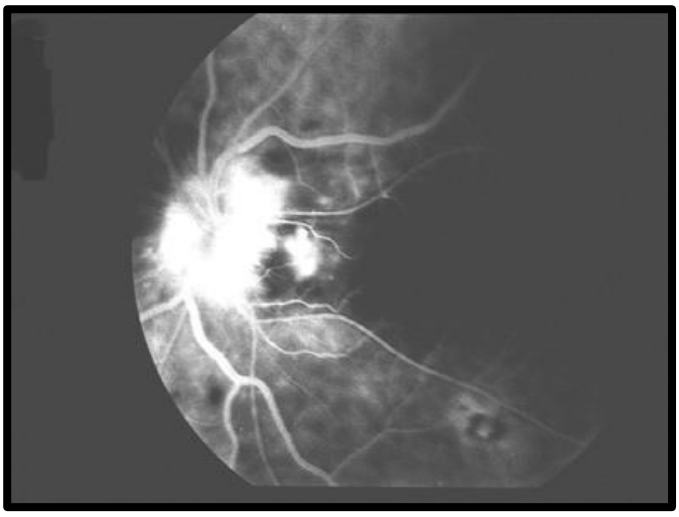


Figure 10 B-FFA - NVD

The incidence of development of proliferative phase mainly depends on the duration of diabetes mellitus. It is approximately twenty years in juvenile diabetes, (not less than eight years in juvenile onset) and about 16 years in adult onset.

Proliferative stage usually begins with neovascularisation. The endothelial cells of the new vessels are thought to retain the ability of giving rise to primitive mesenchymal element. These new vessels can differentiate into endothelial cells to form new vessels or can differentiate into fibrocytes, which proliferate to form fibrous component of fibrovascular tissues. The new vessels are formed in the

areas of ischemia and these areas of the retina are shown to have lesser viability when compared to the normal tissue.

The hypothesis is that an ischemic retina due to a decreased blood supply liberates a diffusible biochemical substances which have vasoproliferative properties on not only the adjacent vessels but also the more distant vessels. However, the infarcted and dead retina has no such effect, thus making the principle of treatment of photocoagulation.

It is found that mild degree capillary nonperfusion results in neovascularisation away from the optic disc (NVE), moderate degree gives rise to neovascularisation at the optic disc (NVD) whereas neovascularisation of iris and angle are seen with a high degree of capillary nonperfusion.

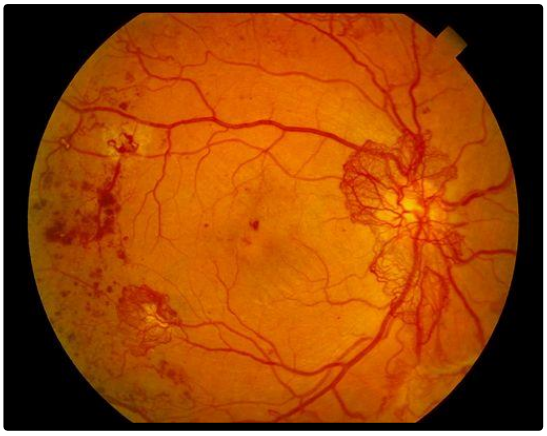


Figure 11A-NVD WITH NVE

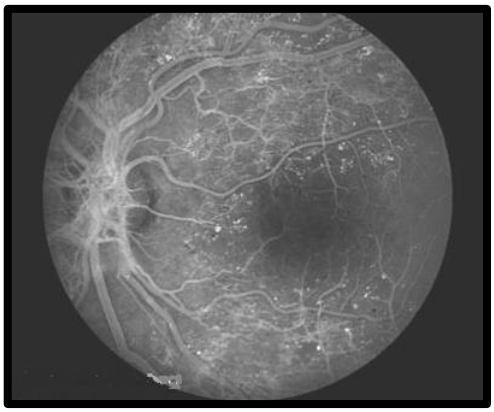


Figure 11B -FFA-NVD WITH NVE

NEOVASCULARIZATION ELSEWHERE (NVE):

The new vessels do not share the blood retinal barrier characteristic of normal blood vessels. These new vessels leak and forms the basic feature to be observed in a fluorescein angiogram. The new vessels break through the internal limiting

membrane and arborise between the posterior hyaloid membrane and internal limiting membrane. Usually the fibrovascular tissue does not penetrate into the vitreous. It forms dense adhesions with posterior hyaloid membrane, presumably due to intermingling of the collagenous elements of the posterior hyaloid and the new fibrous tissue. These adhesions cause hindrance to retinal function by forming fibrovascular vitreous bands. New vessels from the disc may penetrate the vitreous in the region of the optic disc because the posterior limiting membrane of retina is thin in this region.

These changes are preferentially seen near arteriovenous crossings with a predilection for the superotemporal quadrant and occasionally seen at the periphery.

NATURAL COURSE OF PDR[59, 70,71,72]

Initially new vessels may rarely be visible. Later their caliber is about one eighth to one fourth of that of a major retinal vein at the disc margin, and occasionally they may be as large as normal veins. These new vessel patches often lie over retinal veins and appear to drain into them. The superior temporal vein is usually more frequently involved than the others.

Nearly all these new vessel patches are adherent to the posterior vitreous surface that becomes apparent when posterior vitreous detachment occurs adjacent to the patch, pulling its edge forward.

New vessels are usually asymptomatic before the beginning of posterior vitreous detachment. Small hemorrhages in the posterior vitreous may be occasionally seen near the growing ends of new vessels, but usually remain subhyaloid or hang suspended in the posterior portion of the vitreous without

becoming evident to the patient. When symptomatic vitreous hemorrhages does occur it is usually associated with some degree of localized posterior vitreous detachment.

Traction exerted by the fibrous bands on the new vessels appears to be an important factor contributing to the recurrent vitreous hemorrhages that often coincide with extension of vitreous detachment. Hemorrhages may also occur spontaneously, sometimes in relation to bouts of vigorous coughing or vomiting and occasionally at the time of insulin reactions. More often they occur during sleep and are not related to any obvious factor. Blood present in the fluid vitreous, that lies posterior to the detached vitreous framework is usually absorbed within a period of weeks or several months, retaining its red color until it is absorbed completely. Hemorrhage in the formed vitreous tends to lose its red colour and becomes white before absorption is complete. Absorption of a large hemorrhage from the vitreous cavity is usually slow, requiring many months and often not completed before a fresh hemorrhage occurs, except when the PDR enters the burned out stage. Contraction of these fibrovascular proliferations may lead to retinal detachment. This may be limited to avulsion of a retinal vessel, most commonly a vein, and is sometimes accompanied by vitreous hemorrhage or elevation of a thin fold of retina. In some cases retinal detachment may be more

extensive, but the concave shape typical of traction detachment is generally maintained. Sometimes, small apparently full thickness retinal holes may be seen near the proliferations. These may lead to rhegmatogenous retinal detachment. When rhegmatogenous detachment occurs, it tends to have a flat or convex anterior surface and is more extensive, often reaching up to the ora serrata. The timing and degree of shrinkage of the vitreous and fibrovascular proliferations and

also by the type, extent and location of the new vessels, responsible for vitreoretinal adhesions, generally influence the occurrence of retinal detachment.

When vitreous contraction reaches completion, i.e. when the vitreous has detached from all areas of the retina except those at which vitreoretinal adhesions associated with new vessels prevent such detachment, proliferative retinopathy tends to enter into the burnt out stage or involutional stage. Vitreous hemorrhages may decrease in frequency and severity and may stop completely, although many months may elapse before there is substantial vitreous clearing. Some degree of retinal detachment may be present at this stage. If this is localized and the macula remains intact, the visual acuity may be good. However, dragging or distortion of the macula or long standing macular edema leads to substantial decrease in vision.

Later in the disease, new blood vessels may form from within the stroma of the iris and the angle of anterior chamber, with accompanying fibrosis. This development blocks the outflow of aqueous humor, causing neovascular glaucoma, with a drastic elevation of the intraocular pressure.

Another change that occurs as the diabetic retinopathy progresses is diabetic macular edema, which is due to the breakdown of the blood–retinal barrier, with leakage of plasma from small blood vessels in the macula. This results in swelling of the central retina[73].

Resorption of the fluid elements from plasma results in the deposition of lipid and lipoprotein components and the formation of hard exudates. Even though diabetic macular edema does not cause complete blindness, it frequently leads to severe loss of central vision.

In Type I diabetes, diabetic retinopathy induces vision loss mainly due to the formation of new vessels in the fundus resulting in the development of proliferative diabetic retinopathy, whereas in Type II diabetes, vision loss is mainly due to macular edema, and proliferative retinopathy is usually rare[74].

ROLE OF VASOACTIVE FACTORS:

Several vasoactive factors like: VEGF, PKC, Heparin, angiotensin II, PEDF, metalloproteases and biochemical pathways may be affected by chronic hyperglycemia in diabetes, resulting in the development of structural and functional changes in diabetic retinopathy[75].

All these factors are interrelated.

a) Vascular endothelial growth factor – A [VEGF-A]: [76,77,78]

- It is one of the most potent angiogenic factors.
- It is a major cause for increased retinal permeability.
- 6 major isomers - 121, 145, 165, 183, 189, 206.
- VEGF – 165 isoform is emerging as one of the most important factors in the pathophysiology of PDR/ DME.
- RPE cells, ganglion cells, Muller cells, pericytes, endothelial cells, glial cells, neuronal and smooth muscle cells of the diabetic retina are known to produce it.
- Hypoxia as a result of vasoconstriction and capillary loss leads to up-regulation in the expression of VEGF and increases vascular permeability.

b) Protein kinase – C [PKC]: [79]

- Activation of PKC by phorbol esters is associated with increased permeability in epithelial and endothelial culture cells.

- Certain isoforms of PKC play an important role in VEGF induced vasopermeability.

RISK FACTORS FOR DIABETIC RETINOPATHY[38,80,81,82]

1) **Duration of diabetes:** It is a significant risk factor for the development of diabetic retinopathy. After 20 years of diabetes nearly all patients with type-1 and more than 60% of patients with type – 2 diabetes develop some degree of retinopathy.
2) **Age:** One of the most important determinants of the prevalence of retinopathy is the patient's age at the time of diagnosis of diabetes mellitus. Studies have found that NPDR and macular edema appeared sooner in patients when the diagnosis of diabetes mellitus was made after the age of 40 years when compared to the onset in younger patients[81,82].

3) **Sex**: There is higher prevalence of PDR in males than females among the younger age group whreas among the older diabetic patients, there is no sex differentiation. Women are usually more liable to diabetes when compared to men (3:2) and hence, liable to develop retinopathy[83].

4) **Glycemic control:** The most effective medical treatment for DR so far, is glycemic control. Trials like DCCT (diabetic control and complications trial) and the UKPDS (United Kingdom Prospective Diabetes Study), concluded

that intensive glycemic control reduces the risk of DR and its progression in both type 1 and type 2 diabetes significantly, though not preventing retinopathy completely.[84] The aim is to achieve fasting blood sugar (FBS) of 5.0-7.0 mmol/l, post prandial blood sugar (PPBS) of <10.0 mmol/l and glycocylated haemoglobin (HbA1C) of <7%. [85]

5) **Blood pressure control:** [82,86,87] Hypertension may contribute to worsening of DR by increasing the stress on endothelium and the release of VEGF that allows stretching of the vessel walls resulting in altered retinal auto-regulation and increased perfusion pressure. The appropriate blood pressure control in diabetics (ABCD) and UKPDS trials showed a beneficial effect of tight blood pressure control on the progression of diabetic retinopathy and visual loss. In type I DM patients treated with ACE inhibitors (antihypertensive) resulted in 23% reduction in progression of diabetic retinopathy. In patients with type 2 diabetes, it resulted in 34% reduction in significant deterioration of retinopathy and visual acuity. The current recommendations for BP control for diabetic adults is <130/85 mmHg.

6) **Lipid control:** [88, 89,90] Elevated serum lipid level is positively associated with retinal hard exudates in diabetic retinopathy. Hard exudates are associated with an increase in the risk for moderate visual loss and sub retinal fibrosis from macular edema. Control of blood lipid levels is very important. Proper diet and exercises and reduced serum lipid levels may result in a decrease in retinal vessel damage.

7) **Nephropathy:** [91] Diabetic nephropathy may increase the risk of progression of diabetic retinopathy and is independent of duration of diabetes and level of glycemic control in both type 1 and type 2 diabetes. Renal failure in diabetics

results in worsening of their retinopathy particularly affecting the macula and also increases risk of development of PDR.

8) **Intraocular surgery:** [38] Cataract surgery and any other intraocular surgery can lead to an increase in macular edema by inflammatory and angiogenic mediators in eyes with or without diabetic retinopathy. However pre-existing DME in patients are at a higher risk for exacerbation. Hence DME should be treated prior to surgery.

9) **PRP** [38]**:** Treatment with PRP or scatter laser for PDR causes vision loss due to worsening of DME, as a result of vascular leakage due to the inflammatory response to the laser treatment.

   ETDRS research group suggested, the risk of vision loss from PRP is reduced by first treating the DME with focal/ grid photocoagulation later by PRP.

10) **Pregnancy** [92]**:** If a previously diabetic woman with no diabetic retinopathy gets pregnant, her chances of developing retinopathy is only 10%. If she gets pregnant when she has NPDR there may be a significant increase in the number of microaneurysms, haemorrages, and thickening of the vessel wall leading to ophthalmoscopically visible venular thickening. These changes tend to regress after childbirth. Only in 4% of patients there is progression to PDR.Women who begin pregnancy with PDR are at a higher risk of progression of the disease. In a study of risk of progression of diabetic retinopathy it was found that it was 2.3% higher during pregnancy when compared with controls during a similar period.

11) **Alcohol:** increase alcohol intake is associated with a threefold increase in risk of severe retinal diseases [93].

AGGRAVATING FACTORS [94]:

Humoral factors like the onset of puberty and pregnancy can results in the progression of diabetic retinopathy.

PROMPTNESS OF REFERRAL [95]

One of the reasons for diabetic patients losing vision from retinopathy is that they don't receive timely ophthalmic attention. There is high risk of developing diabetic retinopathy shortly after diagnosis of NIDDM, though it seems to be less (6.7%) in India. Many a time's elderly patients do not report visual complaints.

IMAGING IN DIABETIC RETINOPATHY

Though the primary method for evaluating diabetic retinopathy involves direct and indirect ophthalmoscopy, various other imaging modalities are of significant utility in the screening, evaluation, diagnosis, and treatment of the different presentations of this disease. Many imaging techniques can be useful depending on the manifestation of diabetic retinopathy namely, colour fundus photography, fluorescein angiography (FA), B-scan ultrasonography, and optical coherence tomography (OCT). [96]

FUNDUS PHOTOGRAPHY

Colour fundus photography is a useful investigative tool in diagnosis of diabetic eye disease. Traditionally, fundus photography was performed using film, but more recently, digital fundus photography has become widely adopted. A digital image allows easy and immediate review of images, image magnification, and allows one to enhance and manipulate images. Fundus photography is also useful in documentation of the diabetic retinopathy as well as counselling the patient and in demonstrating

what their disease looks like. Fundus photography is also useful in monitoring the improvement or progression of diabetic retinopathy over time. [96]

There are different types of fundus photography: standard, wide field, and stereoscopic.

1) Standard fundus photography

Standard macular fundus photography captures 30° of the posterior pole of the eye, which includes the macula and the optic nerve[97]. The main advantage of this form of colour photography includes that it is easy to use and is easily available, and can be utilized to assist in documentation.

The finer details are usually not apparent, and it can be difficult to obtain good imaging if there is any media opacity[98]

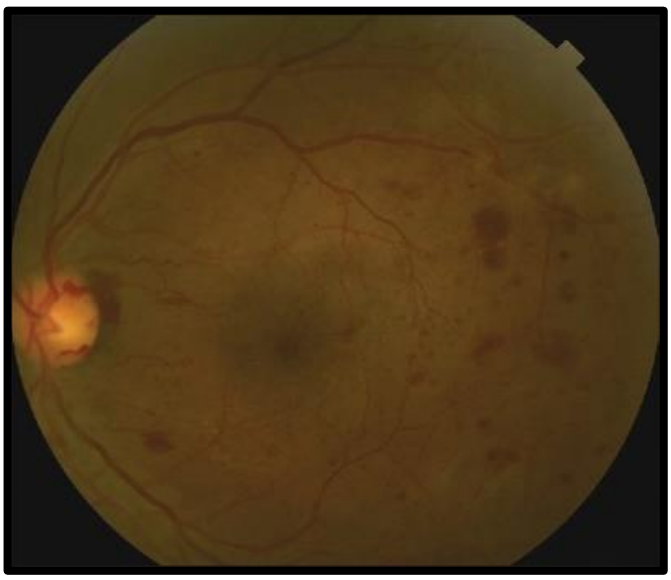


Figure 12-Standard color fundus photo, showing the posterior pole including the optic nerve and the macula of the left eye of a patient with proliferative diabetic retinopathy.[97]

2) Widefield fundus photography

Widefield fundus photography has been developed recently, which can image the peripheral retina. With a standard fundus camera, about seven fundus fields are collected and are combined to create a montage image that shows a 75° field of view. The newer fundus cameras that have now been developed, can capture up to a 200° field of view, even with an undilated pupil, allowing a view of over 80% of the total retinal surface area.[99] Theoretically, the main advantage of larger fields of view is that they allow for a more thorough documentation and detection of peripheral retinal pathology with a minimally invasive technique. However, widefield imaging has some limitations which include distortion of images due to the spherical nature of the globe, artifacts due to eyelashes, false colour representation of fundus findings, and expensive equipment costs. Thus, because of these limitations, the traditional 30° fundus photography continues to be the standard method to photograph the fundus.

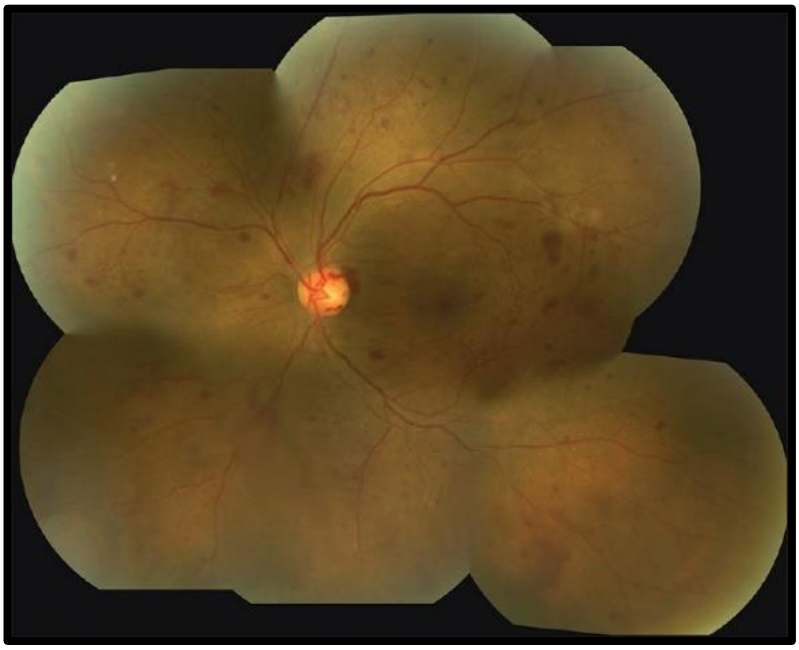


Figure 13-Montage image of the left eye of the same patient seen in Figure 12 using 7-field fundus photography. There is more visualization of the periphery as well as a better appreciation for the extent of the diabetic retinopathy

3) **Stereoscopic fundus photography**

With the stereoscopic fundus photography, two images are created photographically, one for each eye, and when viewed correctly, they become fused in the brain to form a single stereoscopic picture. This form of photography allows the examination of the patient's pathology in three-dimensions, very similar to that of direct ophthalmoscopy [100].

As digital fundus photography continues to improve and the number of patients with this disease increases, colour fundus photography will likely serve as an important screening modality in future.

FLUORESCEIN ANGIOGRAPHY

Another important imaging technique in the evaluation of diabetic retinopathy is FA. The technology was first described in 1961 and was introduced by Gass in 1967, into mainstream ophthalmology[101]. FA is very useful in evaluating diabetic eye disease and is currently the gold standard for evaluating the retinal vasculature, the part of the retina that is most affected by diabetes.[96]

Imaging photos from FA, seen in patients with diabetic retinopathy, can show microaneurysms, which manifest as punctate areas of hyperfluorescence. Small areas of hypofluorescence can signify ischemia from nonperfused retinal capillaries. An increase in the foveal avascular zone due to macular ischemia can be visualized using FA, which may help explain vision loss in diabetic patients. FA also demonstrates abnormal blood vessels in the eye such as intraretinal microvascular abnormalities (IRMA) or retinal neovascularization. As fluorescein is partially unbound in the blood stream, it can leak out of incompetent blood vessels. [102] This leakage of fluorescein

dye over time indicates the breakdown of the blood-retinal barrier. This is best demonstrated in diabetic macular edema, which is visualized as fluorescein leakage over time in the macula. Retinal neovascularization also causes fluorescein leakage, thus, FA is a useful test in confirming the diagnosis of neovascularization of the disc and elsewhere in proliferative diabetic retinopathy[96].

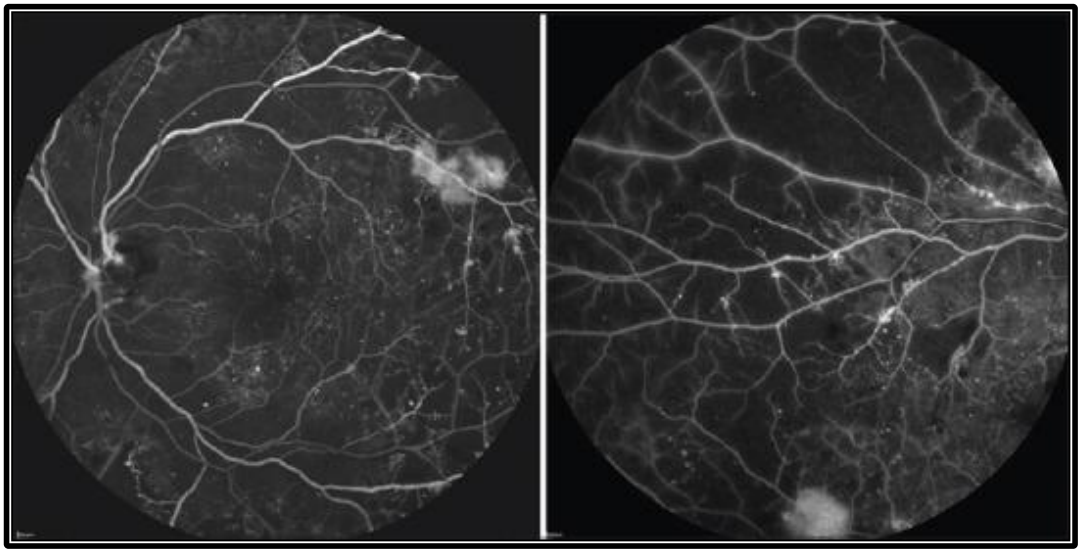


Figure 14 A & B-Fluorescein angiography of the same eye seen in Figures 12 & 13. The standard 30° photo on the left shows scattered microaneurysms throughout the macula. There is IRMA present along the superior arcade as well as leakage

WIDEFIELD FLUORESCEIN ANGIOGRAPHY

Widefield FA has been developed more recently, which allows for improved imaging of the peripheral retina . Hence, this technology is helpful in detecting the peripheral neovascularization, as well as the extent of retinal nonperfusion. Widefield FA helps reveal peripheral areas of capillary nonperfusion which are difficult to visualize with standard field FA[103,104]. These nonperfused regions may be an important source of vascular endothelial growth factor (VEGF), which may contribute to formation of

diabetic macular edema[105]. VEGF release may be halted by "targeted" panretinal photocoagulation (PRP) to these areas of nonperfusion, converting hypoxic areas to anoxic areas, thus leading to an improvement in macular edema[106]

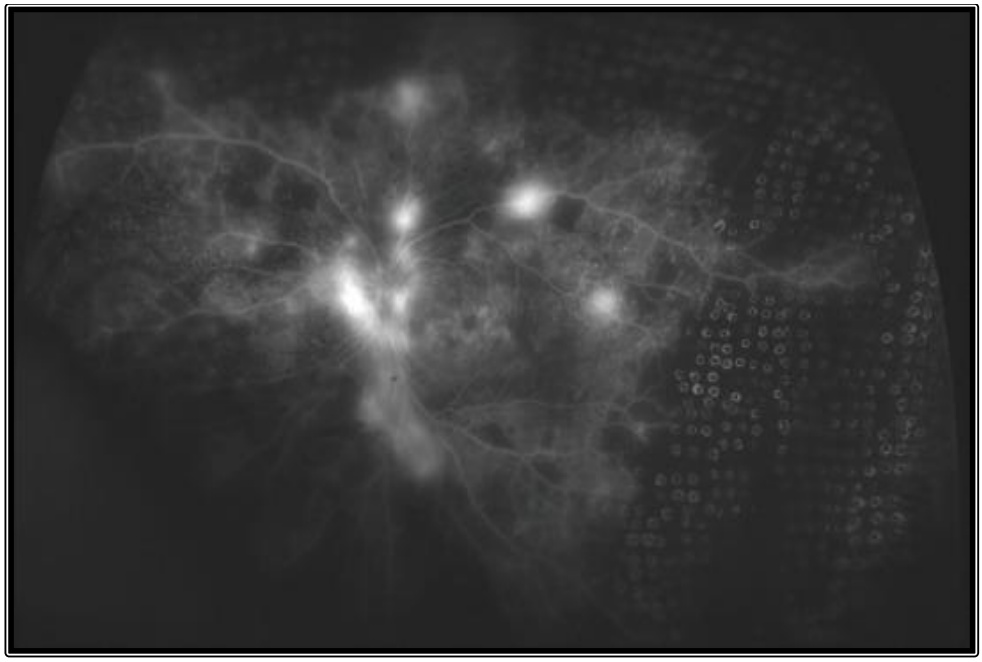


Figure 15-Widefield fluorescein angiography in a patient with proliferative diabetic retinopathy. Note the numerous areas of leakage by the disc and along the arcades corresponding to neovascularization of the disc and elsewhere, respectively. There are extensive areas of nonperfusion in the periphery, and multiple laser scars from panretinal photocoagulation have been targeted to these nonperfused areas.

ULTRASONOGRAPHY

Another imaging modality which is useful in proliferative diabetic retinopathy is B-scan ultrasonography. B-scan ultrasonography creates an image of the eye by transmitting sound waves at high frequency from a transducer to the target tissue, which then returns to the transducer at varying times and amplitudes. These signals are then interpreted to construct a two-dimensional image of the eye[107]. Higher amplitude corresponds to higher densities of tissue that reflect more signal back to the transducer and appear more white in colour on the image. Lower amplitudes indicate low-density tissues that do not reflect as much ultrasound signal. B-scan

ultrasonography is most useful in patients with vitreous haemorrhage or other media opacities in which the retina cannot be visualized directly on ophthalmoscopic examination. B-scan ultrasonography can demonstrate if a retinal detachment is present and can show other retinal pathologies like tractional bands or posterior vitreous detachment[108]. It is not very useful in imaging diabetic retinopathy if the media is clear.

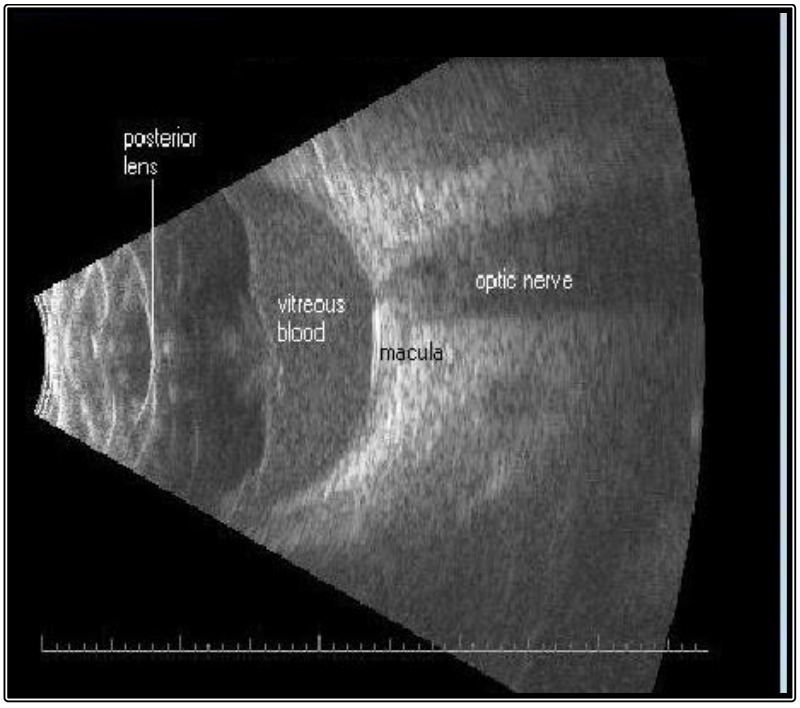


Figure 16 – USG B Scan showing Vitreous Haemmorhage

OCT[109]

OCT is one of the newer, fundamental diagnostic imaging techniques in ophthalmology.

It compliments ophthalmoscopy and FA in patients with diabetic maculopathy. OCT can perform micrometre-resolution, cross-sectional imaging of the retina that closely approximates its histological layers. One of the most important advantages of OCT is

that patients find this procedure very comfortable because it is noncontact, non invasive and the measurement time is very short. In patients with diabetic retinopathy, OCT can be utilized as an objective monitoring technique of the macular thickening before and after therapy. Thereby it quantifies the retinal oedema. OCT is also very useful for the assessment of vitreous, showing whether or not, it is attached to the macula. It is also helpful in detecting the vitreoretinal traction that may not have been identified clinically.

OCT may thus, assist in patient selection with diabetic maculopathy who can benefit from treatment, identify what treatment is indicated and allow the precise monitoring of treatment response. OCT also helps in understanding the anatomy of DME and the intraretinal damage that is caused. It seems to be the investigation of choice for the early detection of macular oedema and for the follow up of diabetic maculopathy.

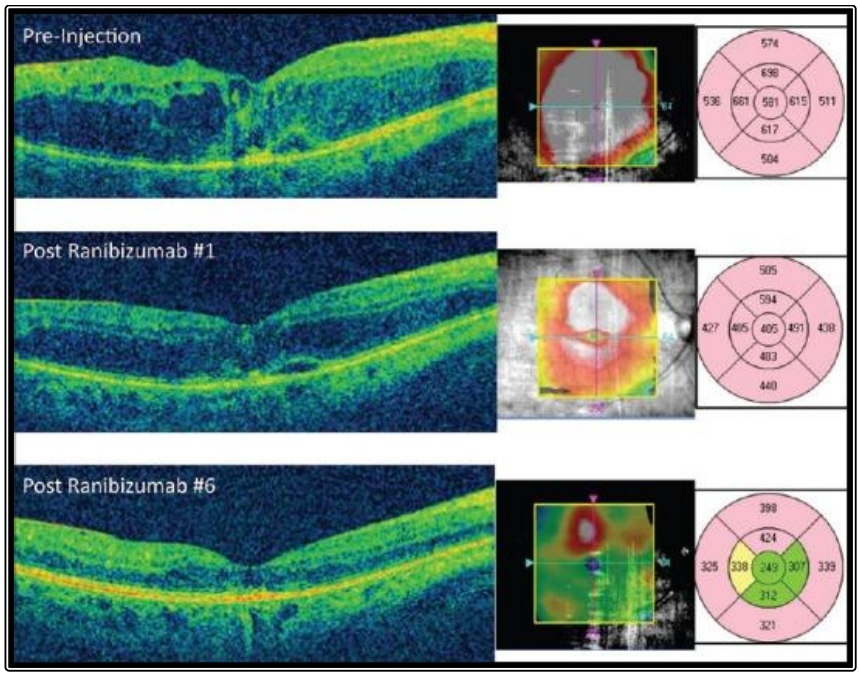


Figure 17A-Optical coherence tomography (OCT) of the macula in a patient with diabetic macular edema. On the presentation (top), the patient has severe macular edema with retinal thickening and intraretinal fluid, shown in red on the OCT map to the right. The picture in between was after the first injection of ranibizumab, showing that the retinal edema improves on OCT. After 6 injections, the OCT (Bottom) shows resolution of fovea macular edema.

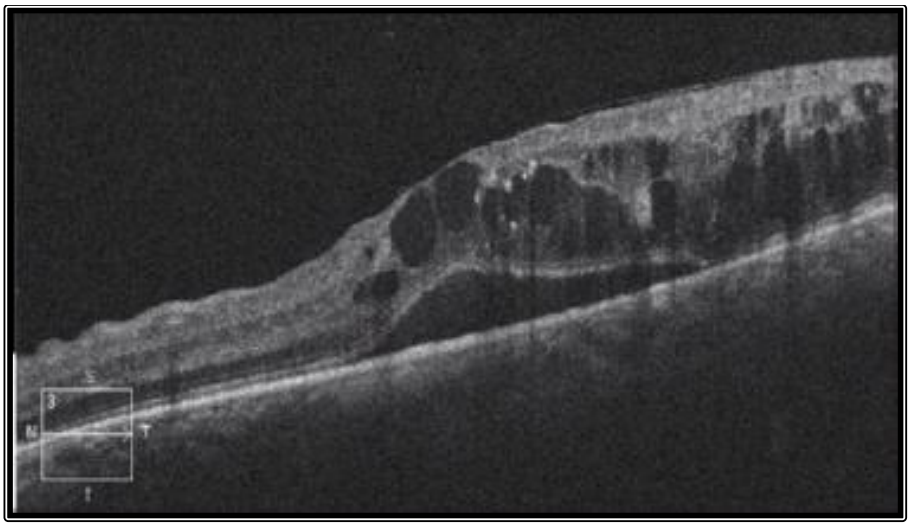


Figure 17B- Optical coherence tomography of the macula of the left eye of a patient with diabetic macular edema. There is subretinal fluid as well as cystoid intraretinal fluid present, causing loss of the normal foveal contour. There are areas of increased hyperreflectivity in the outer plexiform layer corresponding to hard exudates

OCT ANGIOGRAPHY[110]

OCT angiography is a relatively new and promising imaging technique which uses SD OCT or SS OCT for the 3D visualization of the retinal and choroidal microcirculation without the need for dye injection. While fluorescein angiography is still considered as the gold standard for imaging retinal vasculature, OCT angiography is a non-invasive, relatively fast imaging study that can be performed alongside routine OCT imaging. Several studies have demonstrated its applicability in diabetic patients, like providing a prognosis and assessing treatment effects. Currently, OCT angiography allows the study of the retinal vascular bed and discriminates between superficial and deep retinal vascular plexus, which cannot be demonstrated by conventional fluorescein angiography. In contrast, fluorescein angiography is still superior in identifying slow blood flow structures like microaneurysms. OCT angiography may thus provide images with greater detail regarding macular status and may become a novel imaging technique for the diagnosis of DMI in conjunction with fluorescein angiography in the management of DR.

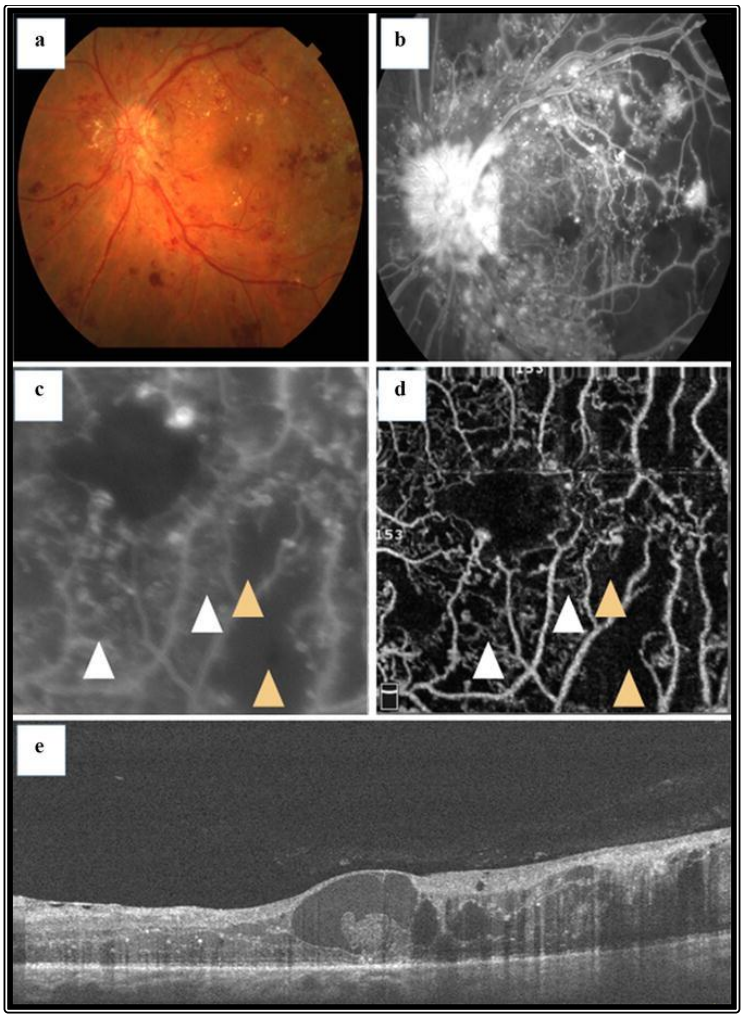


Figure 18- Intraretinal microcirculation and capillary nonperfusion. This patient was diagnosed with PDR. Fundus photograph and fluorescein angiography (a**,** b**). Images of 3 × 3-mm fluorescein angiography (**c**) and OCT angiography were obtained (**d**). The *white arrowheads* indicate superficial collaterals with adjacent nonperfusion areas (*orange arrowheads*) (**c**). The corresponding SD OCT B-scan is also shown (**e**)**

TREATMENT

MEDICAL MANAGEMENT [110]

Medical management constitutes an important part of diabetic retinopathy management in a diabetic patient. Numerous studies have shown the importance of control of the three major systemic associations namely; blood sugar, hypertension and hyperlipidemia.

A. Glycemic Control: [111,112,113,114]

The Wisconsin Epidemiological Study of Diabetic Retinopathy (WESDR) demonstrated a statistically significant relationship between HbA1c and incidence & progression of retinopathy[111]. The Diabetes Control and Complications Trial (DCCT) showed that intensive treatment of Type I diabetes decreases the frequency and severity of retinopathy, nephropathy and neuropathy[112]. The United Kingdom Prospective Diabetes Study (UKPDS) demonstrated that strict glycemic control in Type II diabetic patients decreased all diabetic complications including retinopathy[113,114].

B. Blood Pressure Control [115,116]:

It was concluded that elevation of both systolic and diastolic blood pressure was associated with increased risk of development of PDR in younger onset diabetics by WESDR[115].The UKPDS showed that patient's assigned to tight control had 37% risk reduction of microvascular complications[116].

C. Lipid Control [88,89] :

The data from WESDR and ETDRS shows that there was a significant increase in the

severity of diabetic retinopathy and the presence of retinal hard exudates with increasing cholesterol.

PHOTOCOAGULATION[117,118]

The recommendations for laser treatment for diabetic retinopathy are based on the results of two randomized clinical trials of laser photocoagulation which are, the Diabetic Retinopathy Study (DRS)[117] and the Early Treatment Diabetic Retinopathy Study (ETDRS)[118]. The DRS study showed a 50% reduction in severe visual loss in eyes with severe NPDR or PDR and a visual acuity of 20/100 or better in those treated with photocoagulation compared with the untreated eyes. The risk of severe visual loss with PDR at the end of 2-year follow-up examination was 6% in the treated eyes when compared to 16% in the control group. The risk of vision loss due to diabetic macular edema was reduced by laser treatment by 50–70%. In about 17% of laser-treated eyes experienced a three-line improvement in visual acuity in 5 years.

According to the results of a study conducted by Wei et al., complete resolution of neovascularization was found in 67% of eyes, and a partial resolution in 33% which needed additional photocoagulation[119]. A study by Qian et al., reported that panretinal argon laser photocoagulation was effective in 85% of eyes with NPDR and in 77% with PDR[120]. An improvement in visual acuity was observed in 23% and was unchanged in 61%. Study by Rema et al., showed the outcome of patients with Type II diabetes and PDR after panretinal laser photocoagulation.[121]About 73% of patients with visual acuity of 6/9 or better maintained their vision.

Scatter laser photocoagulation should be considered in patients with severe and very severe NPDR, especially in those with poor compliance, proliferative disease in the fellow eye, pending cataract surgery, advanced renal disease and extensive capillary

closure [122.]

In eyes with severe and very severe NPDR with clinically significant macular edema, the macular edema has to be treated first and panretinal scatter photocoagulation can be delayed until the macular edema has improved. In mild and moderate PDR, especially in Type II diabetes, scatter laser photocoagulation should be performed because it reduces the risk of severe visual loss.

In patients with PDR with high-risk characteristics, extensive scatter laser photocoagulation should be performed immediately because of increase in the risk of visual loss. Panretinal photocoagulation significantly reduces the risk of severe visual loss, but in some patients, vision loss may be there in spite of laser treatment. [123]

Diabetic macular edema may be present at any stage of NPDR and PDR. It is due to focal or diffuse leakage. As the proportion of eyes with macular edema increases with the severity of diabetic retinopathy, ranging from 38% in eyes with moderate to severe NPDR to 71% in eyes with PDR[125]. After the diagnosis of diabetic macular edema is made, a fluorescein angiography should be performed to rule out the presence of ischemic maculopathy. Grid laser treatment is the recommended and the gold standard for treatment of diffuse macular edema.

A study by Zaninetti et al., concluded that eyes that require vitrectomy because of vitreous hemorrhage or retinal detachment in PDR after panretinal laser photocoagulation is usually due to incomplete photocoagulation. Thus, sufficient panretinal laser treatment is mandatory[124].

The most common side effects due to panretinal laser treatment are pain during the treatment, moderate visual loss, restriction of the field of vision and nyctalopia. Other

side effects include glare, exudative retinal detachment, ciliochoroidal effusion, elevated intraocular pressure, angle-closure glaucoma and subretinal or epiretinal fibrosis. The most common side effects due to macular laser treatment are central scotoma in laser burns close to the border of the foveal avascular zone [123].

VITRECTOMY

The advent of pars plana vitrectomy in 1995 by Robert Machemer considerably improved the prognosis of advanced stages of diabetic retinopathy.

Indications for surgery in diabetic eyes can be attributed to: ischemia, media opacities and tractional forces[126]:

(A) Ischemia and complications:

- Active proliferative retinopathy and its consequences
- Neovascularisation of the anterior segment in association with secondary glaucoma

(B) Media Opacities:

- Persistent vitreous opacities
- Persistent sub hyaloid fibrosis
- Neovascularisation of anterior segment in association with vitreous opacities
- Vitreous hemorrhage in combination with "ghost cell glaucoma"

(C) Traction-related complications:

- Progressive fibrovascular proliferation
- Tractional macular detachment
- Combined tractional and rhegmatogenous detachment
- Macular edema with a "taut posterior hyaloid"
- Neovascularised epiretinal membranes

One of the most frequent causes for vitrectomy is persistent vitreous and preretinal hemorrhage. Vitreous hemorrhage occurs as a result of ruptured neovascularization at the vitreoretinal interface secondary to a partial posterior vitreous detachment. Early vitrectomy should be done in eyes with vitreous hemorrhage, precluding laser treatment, not resolving within 4–8 weeks.

A study was conducted by Helbig et al., on 389 eyes that underwent vitrectomy between 1990 and 1994. In this study the indications for vitrectomy were vitreous hemorrhages in 39%, tractional detachment of the macula in 13%, tractional and rhegmatogenous retinal detachment in 12%, and severe progressing proliferative retinopathy in 36%[127,128].

INTRAVITREAL INJECTIONS

Triamcinolone Acetonide [129,130,131]

Robert Machemer, Yasuo Tano, Gholam Peyman, Stephan Ryan and other researchers were the pioneers to consider and use the vitreous cavity as drug reservoir for treatment of intraocular diseases such as proliferative vitreoretinopathy. A recent randomized trial by Sutter et al., gives one of the most convincing evidence of the effect of IVTA as a treatment for diabetic macular edema , in which they concluded

that 55% of eyes treated with triamcinolone gained 5 or more letters of best corrected visual acuity.

IVTA has been used for its anti-inflammatory and anti angiogenic effects in combination with pars plana vitrectomy for patients with diffuse diabetic macular edema with proliferative diabetic retinopathy. Neovascular glaucoma which is a typical end-stage complication of proliferative diabetic retinopathy has recently been treated with IVTA, using the antiangiogenic effect of triamcinolone acetonide[132].

ANTI-VEGF therapy[38,133,134,135,136]:

The important role of VEGF in vascular permeability and its up regulation in patients with diabetic retinopathy is the rationale for use of anti-VEGF drugs.

Specific Intravitreal anti-VEGF therapy given at frequent intervals may temporarily blunt the effects of VEGF and decrease macular edema.

Anti-VEGF drugs are [133]

- Bevacizumab (Avastin : off label)
- Pegaptanib (Macugen)
- Ranibizumab (Lucentis)

Newer drugs: (Under trial)[134]

- VEGF Trap – Eye
- Bevasiranib
- Rapamycin.
- Route of administration: Intravitreal injection

1. Bevacizumab: [134,135,136,137]

It was the first systemic anti-angiogenic drug therapy approved by the FDA in the treatment of colorectal cancer. It is a full length, recombinant, humanized monoclonal antibody that is active against all VEGF isomers.

Dosage: 1.25 mg

- Improvement of visual acuity with decrease in central retinal thickening is observed with a single injection, which lasts for about 4-6 weeks.

- Hence repeated injections which is required for prolonged effect is one of its main disadvantages

- It is used in combination with IVTA in patients with DME who are unresponsive to laser therapy.

- It is an effective adjunct to PRP in the treatment of PDR: it results in greater and rapid regression of the new vessels compared with PRP alone. It can also prevent (or) decrease PRP associated macular edema.

- It can be used as a pre-operative adjunct to vitrectomy for eyes with dense vitreous haemorrhage, to enhance the clearance of the vitreous cavity, reduce the intra-operative time and bleeding.

2. Ranibizumab: [138,139]

It is a recombinant humanized monoclonal antibody fragment that is active against all

VEGF isomers. It has a specific antigen binding region (Fab) that binds to VEGF.

Dosage : 0.5 mg

- It is used for DME and PDR.
- It reduces the foveal thickness and improves visual outcome in patients with DME.

3. Pegaptanib: [140]

It is an RNA aptamer that is engineered to selectively bind only to the VEGF 165 isoform.

Dosage : 0.3 mg

- It is administered at 6 weekly intervals.
- It decreases the central retinal thickness with good visual outcome and also decreases the need for additional photocoagulation in DME patients.

There is regression of new vessels in patients with PDR. As it selectively blocks VEGF– 125, it plays an essential role in pathologic neovascularization, but not physiological neovascularization.

Newer Drugs[133,136]:

1. VEGF trap – eye :

- It is a recombinant fusion protein consisting of VEGF binding domains of human VEGF receptors 1 and 2 fused to the Fc fragment of human IgG. Single Intravitreal injection was found to be effective in DME.

2. Bevasiranib:

- It is a sRNA molecule which inactivates VEGF mRNA and essentially silences the gene responsible for production of all VEGF isomers.

- Advantages: it has a longer duration of VEGF blockage, thus requiring less frequent injections.

3. Rapamycin:

- It is an inhibitor of mammalian target of Rapamycin (mTOR) which is a regulatory protein kinase. mTOR on inhibition, down regulates hypoxia inducible factor 1, which moderates production of VEGF.

Complications of anti-VEGF during local injection[38]:

- Pain and bleeding at the site of Injection (mild)

- Damage to globe with a needle causes – retinal tears, cataract, vitreous hemorrhage.

- Endophthalmitis (rare)

COMBINATION THERAPIES:

1. IVTA in combination with laser photocoagulation[141, 142]:

Laser photocoagulation is done a few weeks after IVTA will improve its efficacy and safety. The short term benefit from IVTA will be combined with the long term benefit of laser photocoagulation.

2. IVTA and anti VEGF[141, 142]:

The combination showed an improvement in visual acuity with a decrease in central retinal thickness and decrease in the likelihood of needing additional photocoagulation at follow up.

Protein kinase inhibitors: RUBOXISTAURIN[38,143]:

It is a specific PKC-B inhibitor which decreases the abnormal vascular permeability and blood flow.

The route of administration: – Oral.

Dosage: 32 mg/day

It reduces the moderate visual loss and need for laser treatment in DME. Currently, it is under regulatory review for the treatment of early DME.

METHODOLOGY

MATERIALS AND METHODS:

SOURCE OF DATA:

Diabetic eye camps conducted every month as per the schedule, with prior publicity, in Kolar district.

STUDY DESIGN:

Cross Sectional Study

SAMPLE SIZE: 250

At 5% absolute error and 95% Confidence interval sample size of 227 was obtained. Considering 10% non-response rate sample size of $227 + 22.7 \approx 250$ diabetic case will be evaluated for diabetic retinopathy in the study.

INCLUSION CRITERIA:

- Patients diagnosed with Type 1 and Type 2 DM.

EXCLUSION CRITERIA:

- Known Hypertensive patients.
- Patients with Gestational DM.

METHODOLOGY

All diabetic patients fulfilling the inclusion criteria were interviewed about their awareness of DR with the help of a questionnaire.

These patients were then subjected to an ophthalmic examination including visual acuity with Snellens chart kept at a distance of 6m in a naturally illuminated room, anterior segment examination was performed with torch light and fundus examination

was done at the end of 45mins after dilatation of the pupils with the instillation of Tropicamide plus (tropicamide 0.8% and phenylephrine 5%) with a direct and/or indirect

ophthalmoscope in the camp and those patients who are diagnosed to have diabetic retinopathy changes who need further evaluation with a slit lamp, applanation tonometer, gonioscopy, fundus fluorescein angiography and B-scan ultrasonography were then referred to the hospital. The patients diagnosed to have diabetic retinopathy were further classified according to ETDRS classification into Non Proliferative DR, Proliferative DR, Advanced diabetic eye disease and Macular edema.

	NON PROLIFERATIVE DR (NPDR)
NO DR	
Very Mild	Only Microaneurysms
Mild	Any or all of: microaneurysms , retinal haemorrhages, hard exudates, cotton wool spots. No IRMA changes or venous beading.
Moderate	-Severe retinal hemorrhages in 1-3 quadrants or mild IRMA changes -significant venous beading in no more than one quadrant -cotton wool spots maybe present.
Severe	4-2-1 rule -severe hemorrhages in all 4 quadrants -significant venous beading in 2 or more

	quadrants -moderate IRMA in one or more quadrants
Very Severe	2 or more criteria in severe
	PROLIFERATIVE DR
Mild – Moderate	New vessels on the disc(NVD) or new vessels elsewhere(NVE)
High risk	-NVD with vitreous hemorrhage -NVE greater than disc area with vitreous or preretinal hemorrhage
Advanced Diabetic Eye Disease	-Tractional retinal detachment -Tractional Retinoschisis -Rubeosis iridis-NVG
Macular Eye Disease	-CSME -Macular ischemia

A questionnaire in both English and Kannada, was used to collect the responses. It comprised of six questions on awareness of the disease, five questions on the awareness on treatment, 6 questions on attitude of the patients towards the disease and 4 questions on the practice patterns of the patients about diabetes and eye care. Without prompting from the investigator or relative accompanying the participant, responses were collected. Personal information like age, sex, education status, duration of diabetes and treatment were collected. Two or three graded responses were used for each question. The correct responses of each question were determined by the study investigators prior to the study. If the response of participants to these

questions matched with gold standard, it was considered as correct and 10 points for that response was awarded. For wrong answer, minus 10 points were given. For equivocal response ’0’ point was designated.

The total points of awareness, attitude and practice related questions were regrouped in four categories. Person with 75% to 100% score was considered to have ’excellent’ grade of response. If the score was 50% to 74%, it was considered as good. Persons scoring 25% to 49% and 0% to 24% were grouped into poor and very poor grades respectively.

We used pretested data collection form. The data from these forms was transformed on spreadsheet using EPI Data software. We used Statistical Package for Social Studies (SPSS) for the analysis. Univariate analysis was conducted by parametric method. We calculated frequencies and percentage proportions.

Awareness was then created among all the patients attending the eye camp with the help of audio visual aids and through lectures regarding the effects of diabetes on the eye and the necessity for regular eye check-up to prevent ocular morbidity.

RESULTS

STATISTICAL ANALYSIS:

Data was entered into Microsoft excel data sheet and was analysed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi square test or Fischer's exact test** (for 2x2 tables only) was used as test of significance for qualitative data. **Yates correction** was applied were ever chi-square rules were not fulfilled (for 2x2 tables only).

Continuous data was represented as mean and standard deviation. **Independent t test or Mann Whitney U test** was used as test of significance to identify the mean difference between two quantitative variables and qualitative variables respectively.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram, Pie diagram and Scatter plots.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

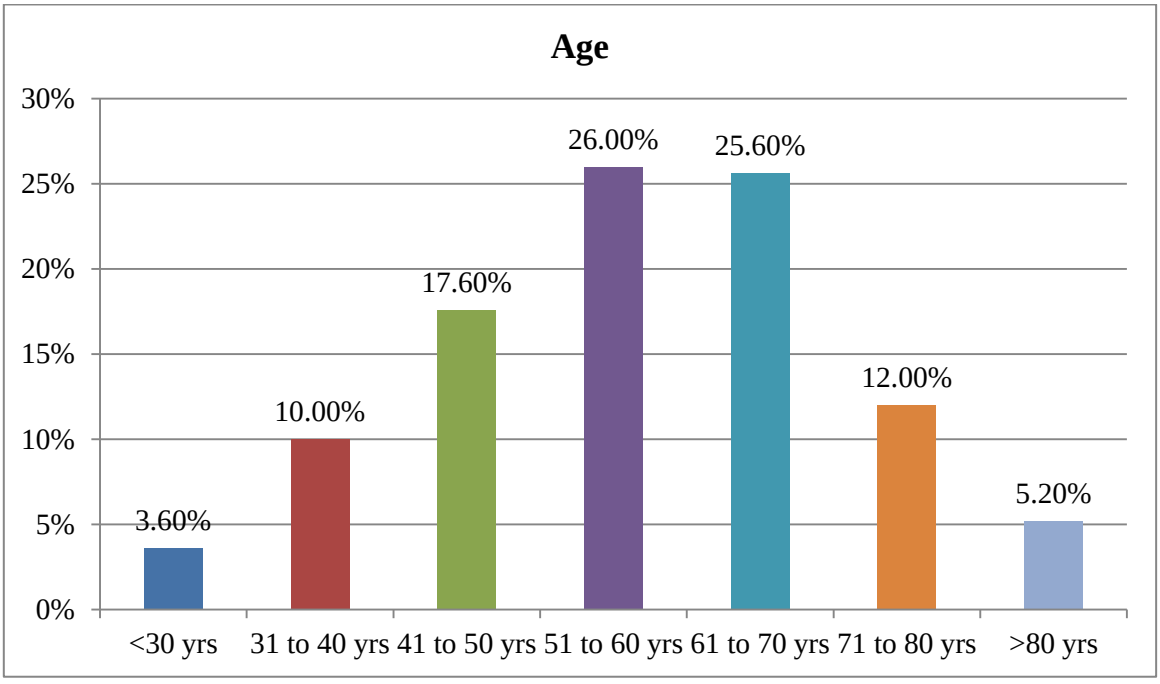
Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data. EPI Info (CDC Atlanta), Open Epi, Med calc and Medley's desktop were used to estimate sample size, odds ratio and reference management in the study.

RESULTS:

Table 1: Age distribution of subjects in the study

Age	Number	Percentage (%)
<30 years	9	3.6
31 to 40 years	25	10.0
41 to 50 years	44	17.6
51 to 60 years	65	26.0
61 to 70 years	64	25.6
71 to 80 years	30	12.0
>80 years	13	5.2
Total	250	100

In our study, majority of subjects were in the age group 51 to 60 years (26%) and 25.6% of the subjects were in the age group 61 to 70 years. Mean age of subjects was 58.01 ± 14.28 years.



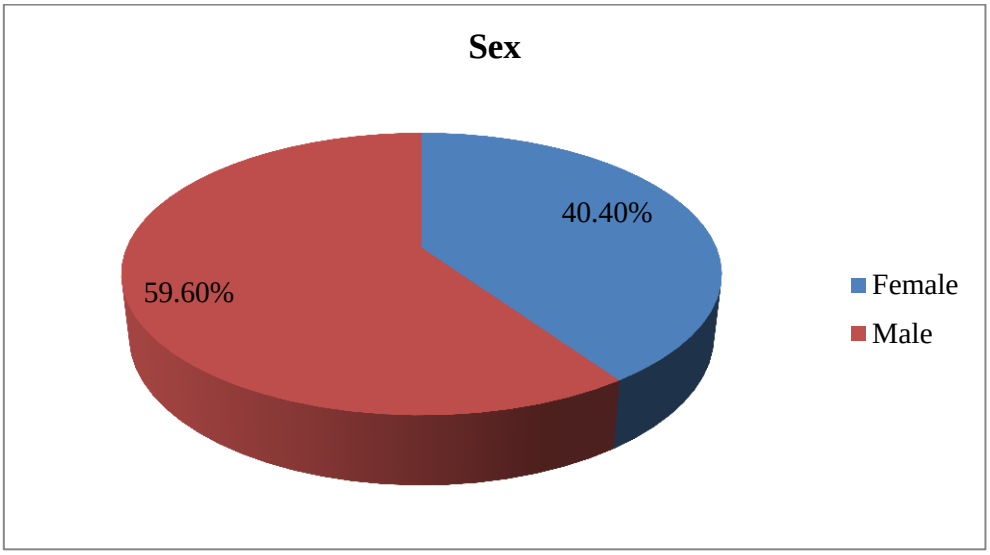
Graph 1: Bar diagram showing Age distribution of subjects in the study

Table 2: Sex distribution among the subjects in the study.

Sex	Number	Percentage (%)
Female	101	40.4
Male	149	59.6

In this study, 59.6% of the subjects were males and 40.4% were females.

Mean age of females was 56.3 ± 13.2 years and males were 59.2 ± 14.9 years.

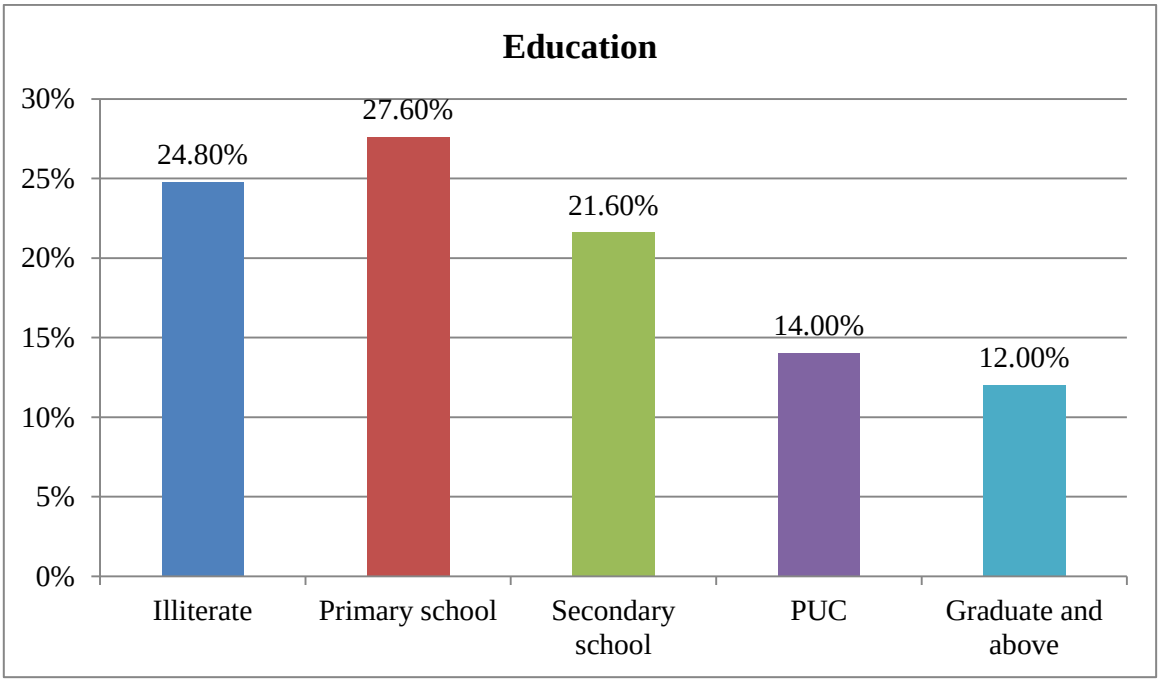


Graph 2: Pie diagram showing Sex distribution among the patients.

Table 3: Education status of subjects in the study

Education	Number	Percentage (%)
Illiterate	62	24.8
Primary school	69	27.6
Secondary school	54	21.6
PUC	35	14.0
Graduate and above	30	12.0

In this study, majority, i.e., 27.6% of the subjects had education till primary school, 24.8% of the subjects were illiterate and 21.6% of the subjects till secondary school.

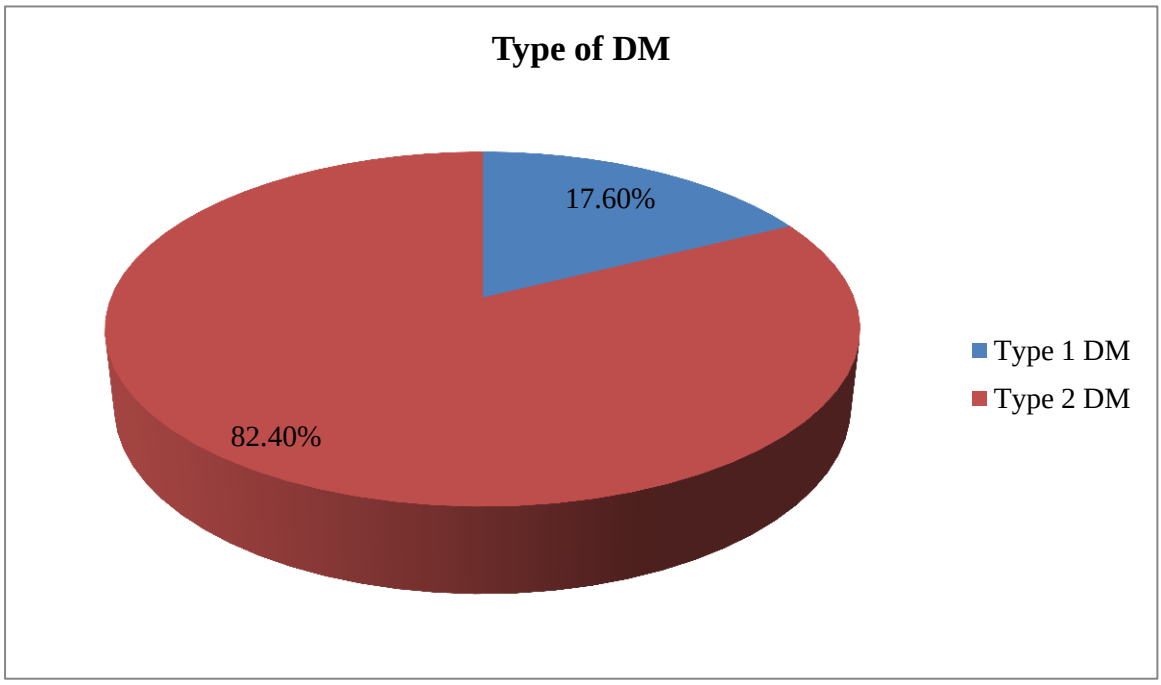


Graph 3: Bar diagram showing Education status of subjects in the study

Table 4: Type of Diabetes mellitus

Type of DM	Number	Percentage (%)
Type 1 DM	44	17.6
Type 2 DM	206	82.4

In our study, 17.6% of the subjects had type 1 DM and 82.4% of the subjects had Type 2 DM.

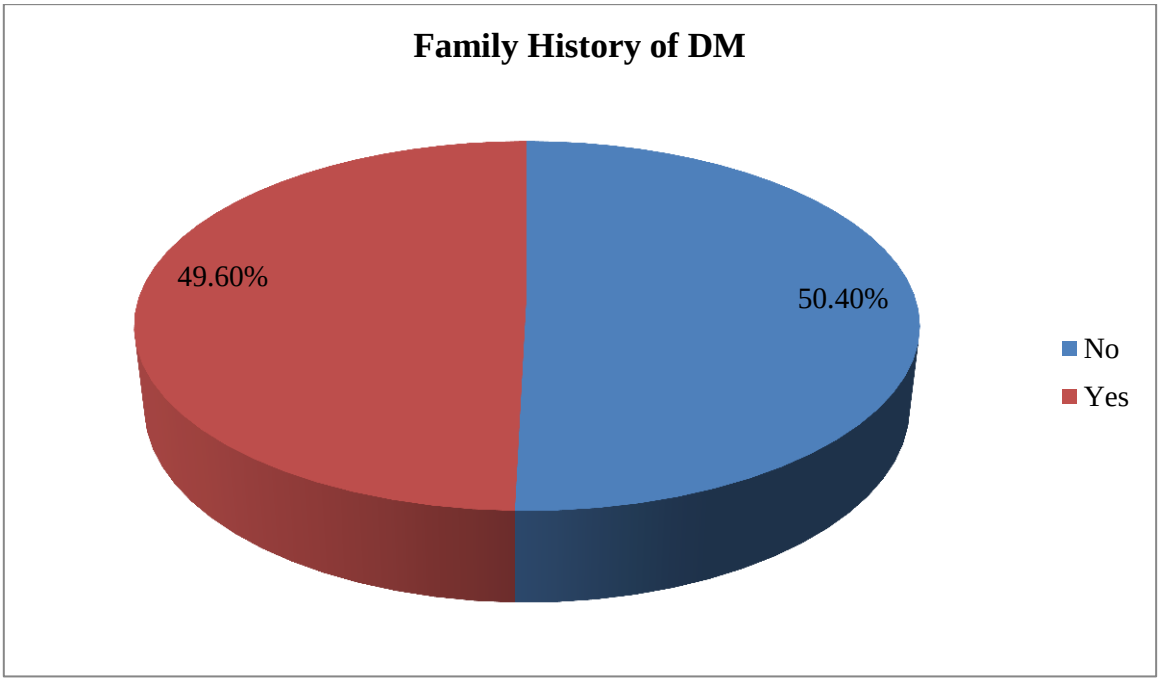


Graph 4: Pie diagram showing Type of Diabetes mellitus

Table 5: Family history of DM among subjects

Family History of DM	Number	Percentage (%)
No	126	50.4
Yes	124	49.6

In this study, 49.6% of subjects had family history of DM.

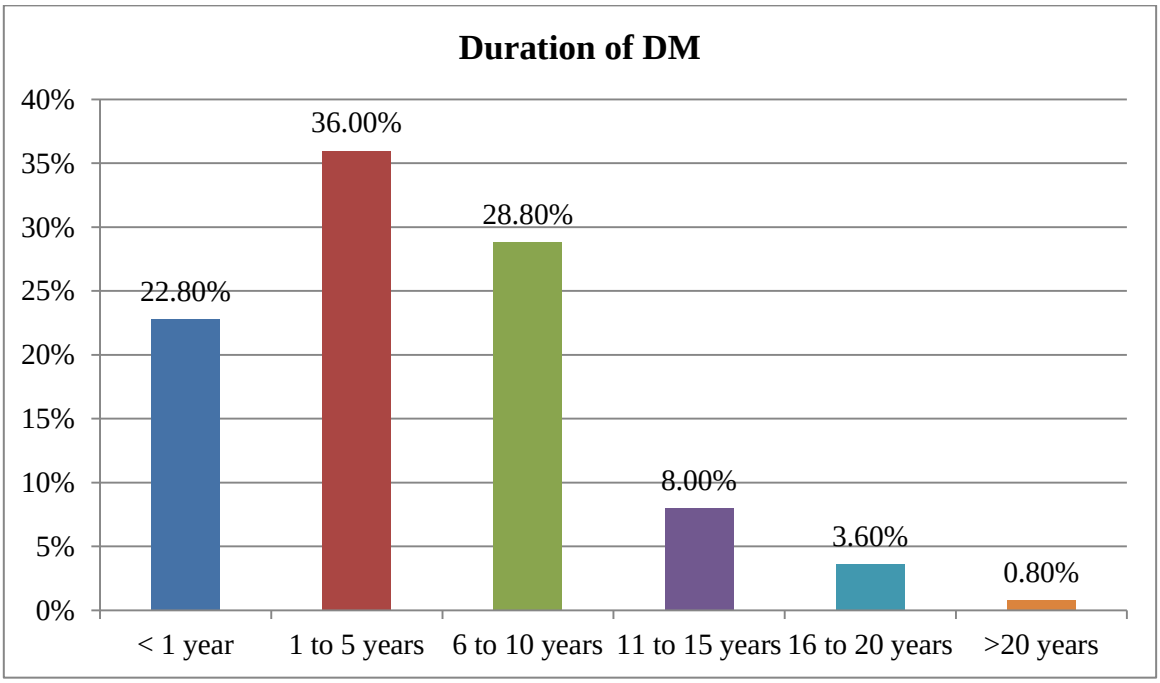


Graph 5: Pie diagram showing Family history of DM among subjects

Table 6: Duration of DM among patients

Duration of DM	Number	Percentage (%)
< 1 year	57	22.8
1 to 5 years	90	36.0
6 to 10 years	72	28.8
11 to 15 years	20	8.0
16 to 20 years	9	3.6
>20 years	2	0.8

In our study, 36% of the subjects had DM for 1 to 5 years, 28.8% of the subjects had DM for 6 to 10 years and 22.8% of the subjects had DM <1 year.

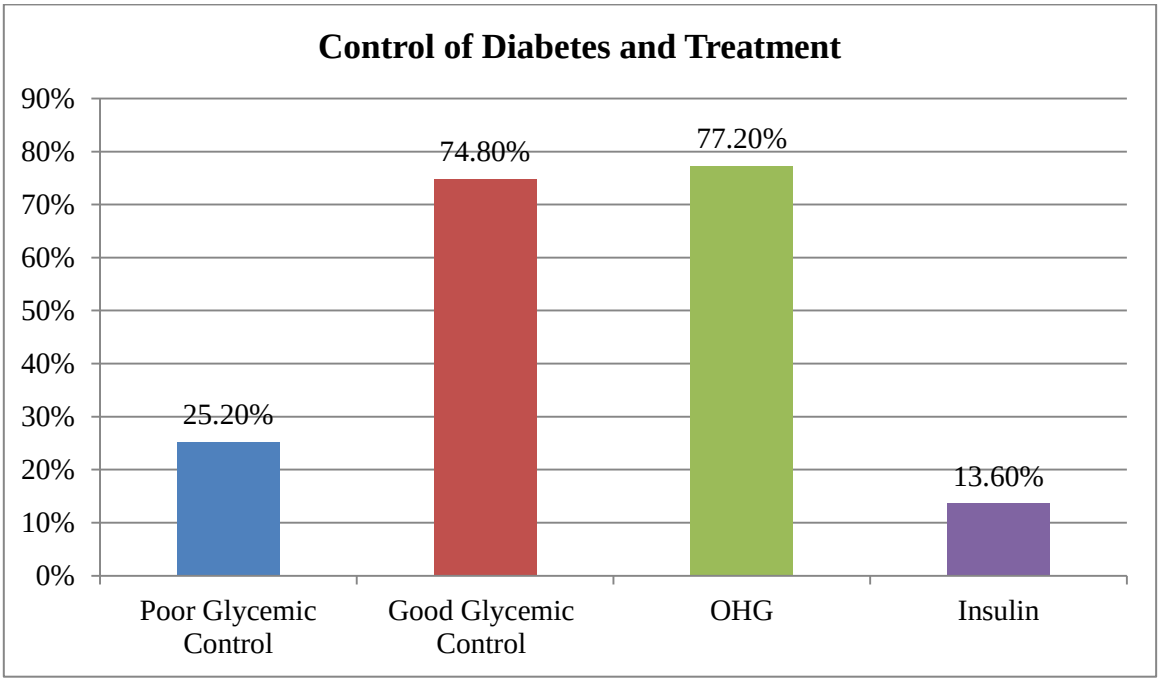


Graph 6: Bar diagram showing Duration of DM among subjects

Table 7: Control of Diabetes and Treatment taken by Diabetics

		Number	Percentage (%)
Control	No	63	25.2
	Yes	187	74.8
Treatment	OHA	193	77.2
	Insulin	34	13.6
	Nil	23	9.2

In this study, 74.8% of subjects had good glycaemic control. 77.2% of the subjects were on oral hypoglycaemic drugs, 13.6% were on insulin and 9.2% were not on any treatment.

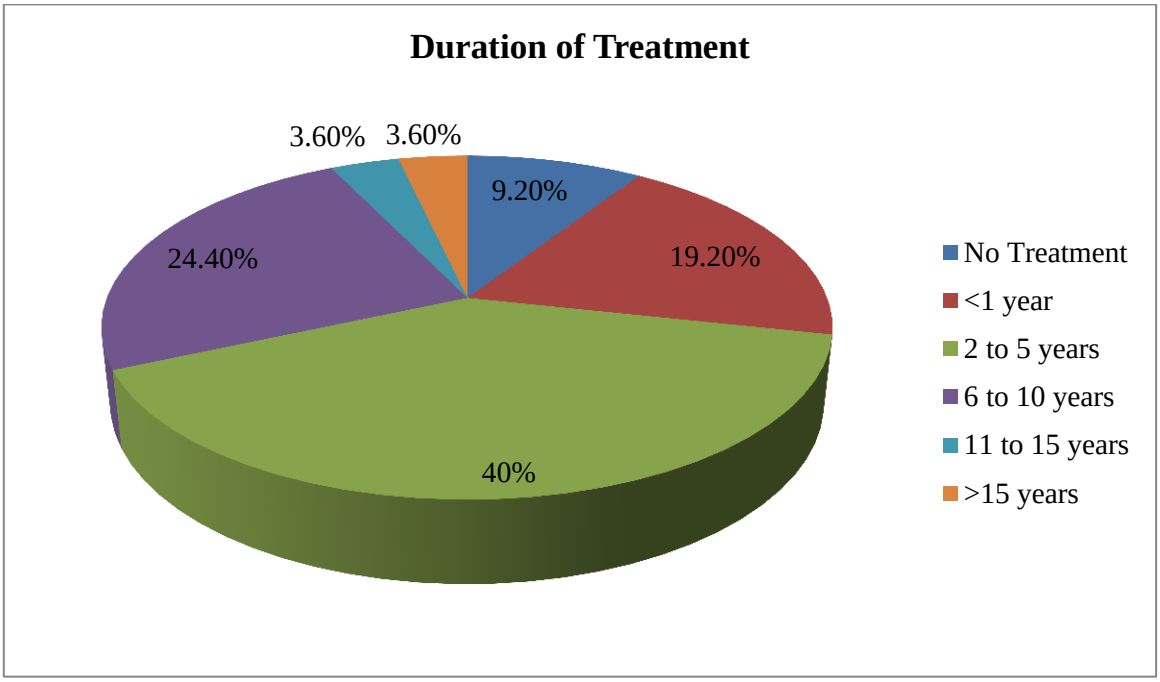


Graph 7: Bar diagram showing Control of Diabetes and Treatment taken by Diabetics

Table 8: Duration of Treatment among subjects

Duration of Treatment	Number	Percentage (%)
No Treatment	23	9.2
<1 year	48	19.2
2 to 5 years	100	40
6 to 10 years	61	24.4
11 to 15 years	9	3.6
>15 years	9	3.6

In our study, 40% of the subjects were on treatment for 2 to 5 years, 24.4% of the subjects for 6 to 10 years and 19.2% for < 1 year.

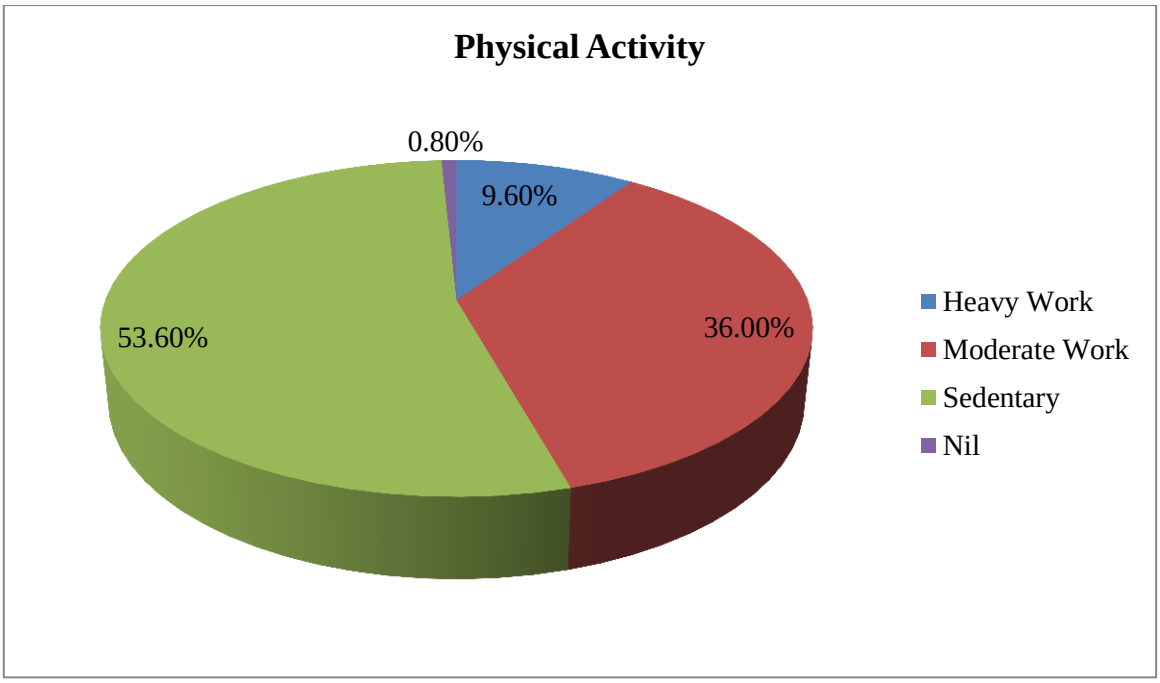


Graph 8: Pie diagram showing Duration of Treatment among subjects

Table 9: Physical activity among subjects

Physical Activity	Number	Percentage (%)
Heavy Work	24	9.6
Moderate Work	90	36.0
Sedentary	134	53.6
Nil	2	0.8

In this study, 9.6% of the subjects were heavy workers, 36% of the subjects were doing moderate work, 53.6% of the subjects were sedentary workers and 0.8% did no physical activity.

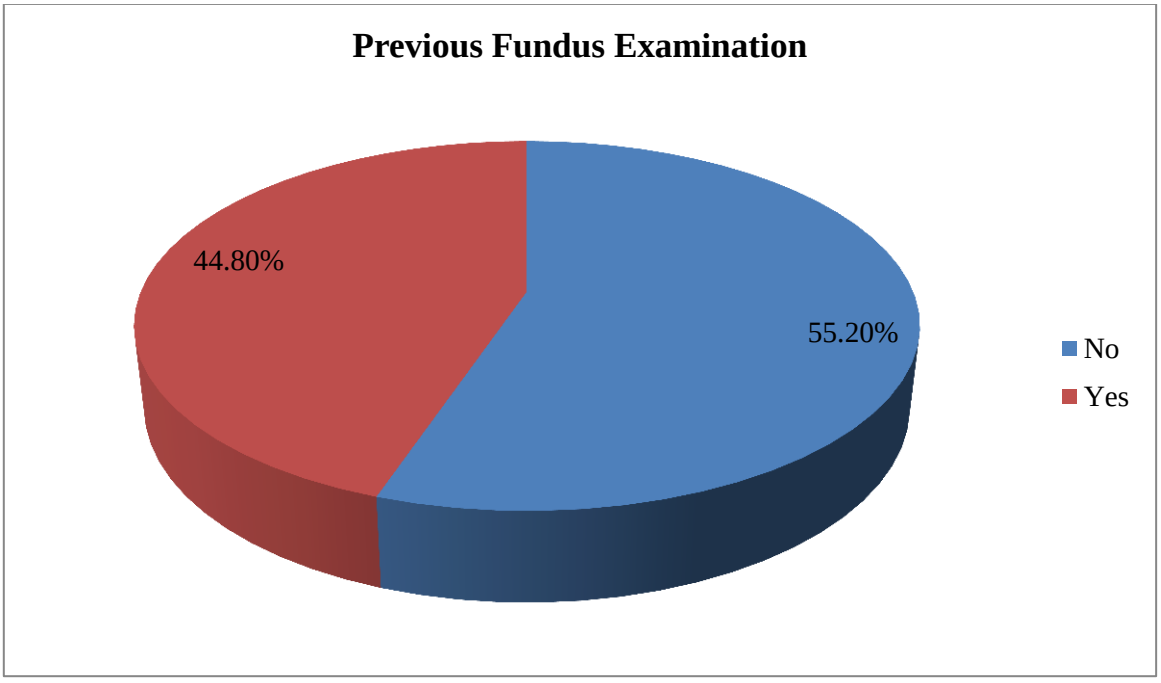


Graph 9: Pie diagram showing Physical activity among subjects

Table 10: Previous Fundus examination among Diabetics

Previous Fundus Examination	Number	Percentage (%)
No	138	55.2
Yes	112	44.8

In the study 44.8% of subjects had history of previous fundus examination.

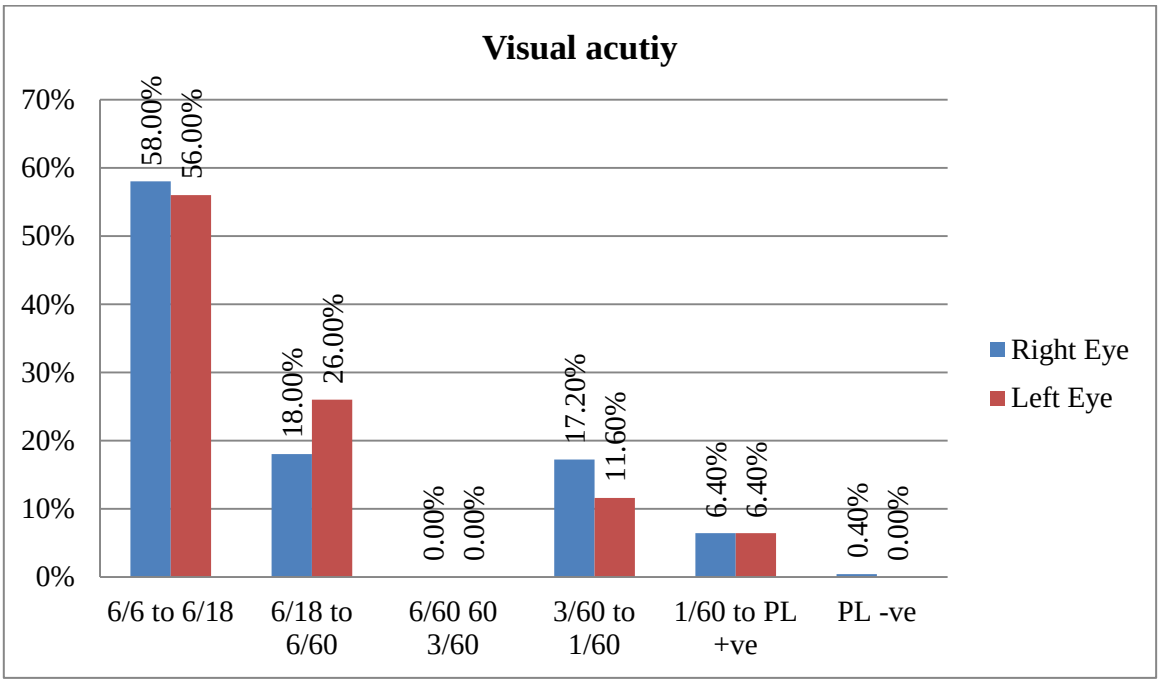


Graph 10: Pie diagram showing previous fundus examination

Table 11: Visual acuity of subjects in right and left eye

	RE Vision	Number	Percentage (%)
Normal or Mild Visual Impairment	6/6 to 6/18	145	58
Moderate Impairment	6/24 to 6/60	45	18
Severe Impairment	5/60 - 3/60	23	9.2
Blindness	2/60 to 1/60	20	8
	<1/60 to PL +ve	16	6.4
	PL -ve	1	0.4
	LE Vision	Number	Percentage (%)
Normal or Mild Visual Impairment	6/6 to 6/18	140	56
Moderate Impairment	6/24 to 6/60	65	26
Severe Impairment	5/60 - 3/60	15	6
Blindness	2/60 to 1/60	14	5.6
	<1/60 to PL +ve	16	6.4
	PL -ve	-	-

In the study on right side 58% of the subjects had 6/6 to 6/18 vision, 18% of the subjects had 6/24 to 6/60 vision, 9.2% of the subjects had 5/60 to 3/60, 8% of the subjects had 2/60 to 1/60 vision, 6.4% of the subjects had 1/60 to PL +ve and 0.4% of the subjects had PL – ve. On left side 56% of the subjects had 6/6 to 6/18 vision, 26% of the subjects had 6/24 to 6/60 vision, 6% of the subjects 5/60 to 3/60, 5.6% of the subjects had 2/60 to 1/60 vision and 6.4% had 1/60 to PL +ve.

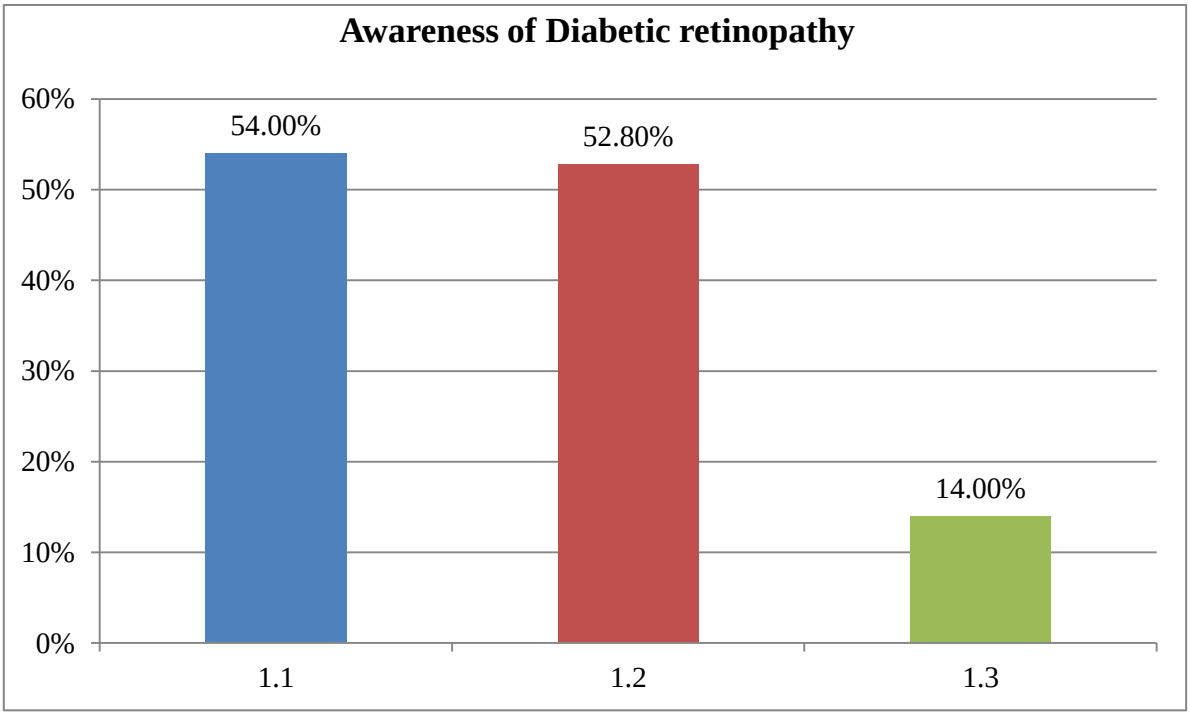


Graph 11: Bar diagram showing Visual acuity of subjects in right and left eye

Table 12: Awareness of Diabetic Retinopathy among patients

		No		Yes	
		Number	Percentage (%)	Number	Percentage (%)
1.1	Are you aware that Diabetes affects the eye	115	46.0	135	54.0
1.2	Did you know that Diabetes can damage eyesight leading to blindness	118	47.2	132	52.8
1.3	Have you heard that it affects the nerve layer of the eye (Diabetic Retinopathy)	215	86.0	35	14.0

In the study 54% of the subjects were aware that Diabetes affects eye, 52.8% of the subjects were aware that Diabetes can damage eyesight leading to blindness and 14% of the subjects were aware that it affects the nerve layer of the eye.



Graph 12: Bar diagram showing Knowledge of Diabetic Retinopathy among subjects

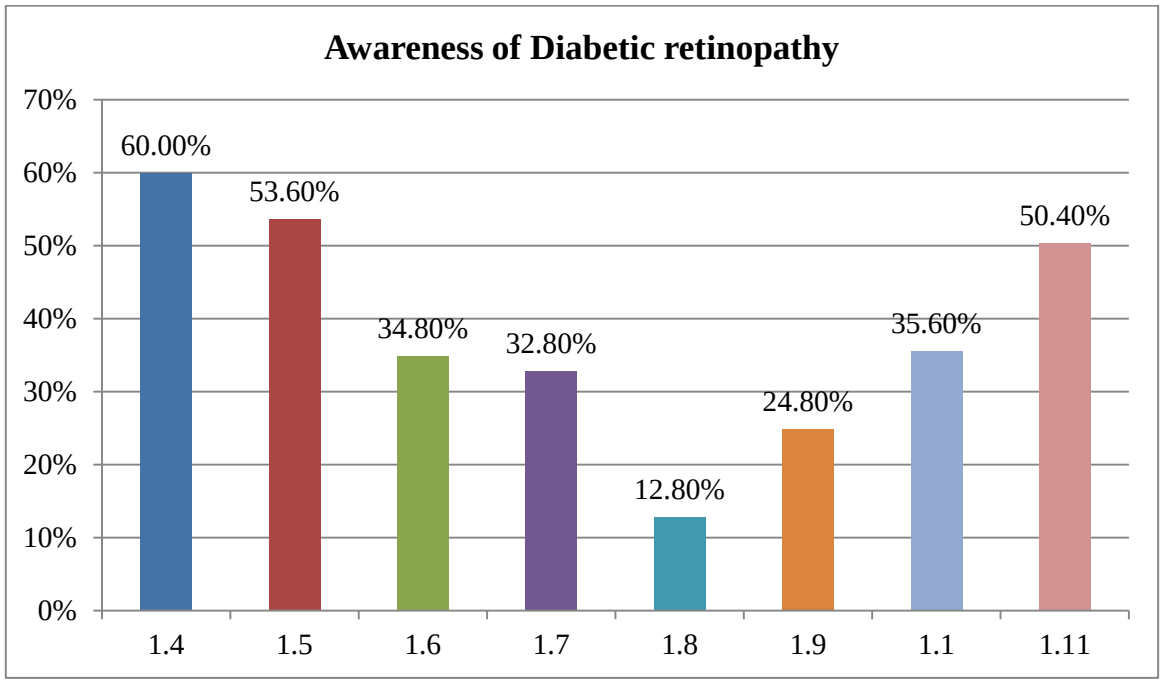
Table 12: Awareness of Diabetic Retinopathy among subjects

		Don't Know		No		Yes	
		Number	Percentage (%)	Number	Percentage (%)	Number	Percentage (%)
1.4	Regular checkup and treatment can prevent / delay damage due to Diabetic Retinopathy in eye	77	30.8	23	9.2	150	60.0
1.5	Control of blood sugar and lipids makes eye / Diabetic Retinopathy treatment effective	85	34.0	31	12.4	134	53.6
1.6	If vision is damaged due to Diabetic Retinopathy, use of ' Low Vision Aids'(Spectacles) helps in daily work	126	50.4	37	14.8	87	34.8
1.7	In Diabetic Retinopathy, one eye may be affected first followed by the other eye.	116	46.4	52	20.8	82	32.8
1.8	Knowledge of modalities of treatment for Diabetic Retinopathy	143	57.2	75	30.0	32	12.8

1.9	An eye with Diabetic Retinopathy, if successfully treated with laser, doesn't need laser treatment again	151	60.4	37	14.8	62	24.8
1.10	Laser treatment of Diabetic Retinopathy is painful	112	44.8	49	19.6	89	35.6
1.11	Frequency of follow up visits needed	83	33.2	41	16.4	126	50.4

In this study 60% of the subjects were of the opinion that regular check-up and treatment can prevent / delay damage due to Diabetic Retinopathy in the eye, 53.6% of the subjects opined that control of blood sugar and lipids makes Diabetic Retinopathy treatment effective, 34.8% of the subjects felt that if vision is damaged due to Diabetic Retinopathy, use of 'low vision aids' (spectacles) helps in daily work, 32.8% of the subjects were of the opinion that in Diabetic Retinopathy, one eye may be affected first followed by the other eye.

12.8% of the subjects were aware of the modalities of treatment for Diabetic Retinopathy, 24.8% of the subjects opined that an eye with Diabetic Retinopathy, if successfully treated with laser, does not need laser treatment again, 35.6% of the subjects were of the opinion that laser treatment of Diabetic Retinopathy is painful and 50.4% of the subjects opined that frequency of follow up visits is needed.



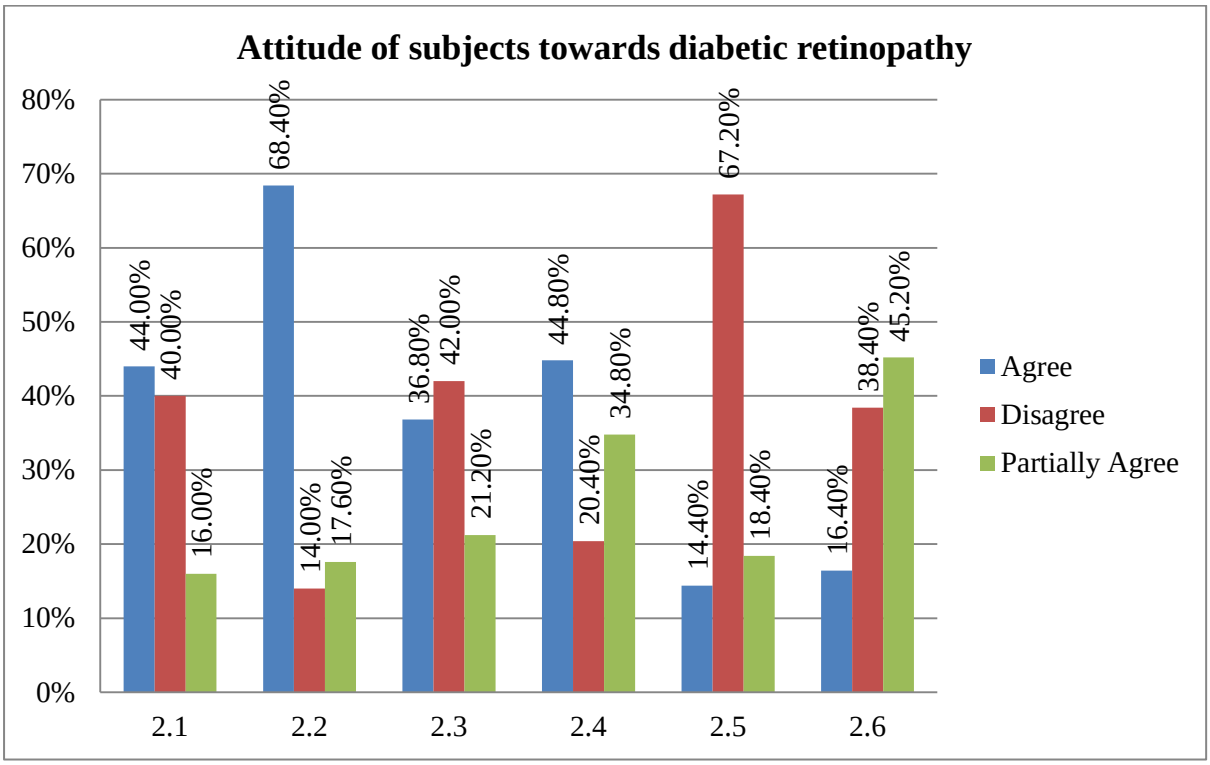
Graph 12 : Bar diagram showing Knowledge of Diabetic Retinopathy among subjects

Table 13: Attitude of subjects towards Diabetic Retinopathy

		Agree		Disagree		Partially Agree	
		Number	Percentage (%)	Number	Percentage (%)	Number	Percentage (%)
2.1	If my vision is good my eyes are not affected in Diabetes, so I do not need annual eye testing	110	44.0	100	40.0	40	16.0
2.2	The information on eye problems due to Diabetic Retinopathy should be given by eye doctor only	171	68.4	35	14.0	44	17.6
2.3	If im taking eye treatment, I need not worry about controlling my sugar and lipids	92	36.8	105	42.0	53	21.2
2.4	If my eye is treated with laser once, I don't need laser treatment again in the same eye	112	44.8	51	20.4	87	34.8

2.5	Patients with Diabetes often waste their time and money in eye check up as most of the time eyes of Diabetics are normal	36	14.4	168	67.2	46	18.4
2.6	One should not be treated with laser as its painful	41	16.4	96	38.4	113	45.2

44% of the subjects agreed that 'if my vision is good my eyes are not affected in Diabetes, so I do not need annual eye testing' , 68.4% of the subjects were of the opinion that the information on eye problems due to Diabetic Retinopathy should be given by eye doctor only, 36.8% of the subjects agreed that 'if I'm taking eye treatment, I need not worry about controlling my sugar and lipids' , 44.8% of the subjects agreed that 'if my eye is treated with laser once, I don't need laser treatment again in the same eye' , 14.4% of the subjects were of the opinion that patients with Diabetes often waste their time and money in eye check-up as most of the time eyes of Diabetics are normal and 16.4% of the subjects felt that one should not be treated with laser as its painful.

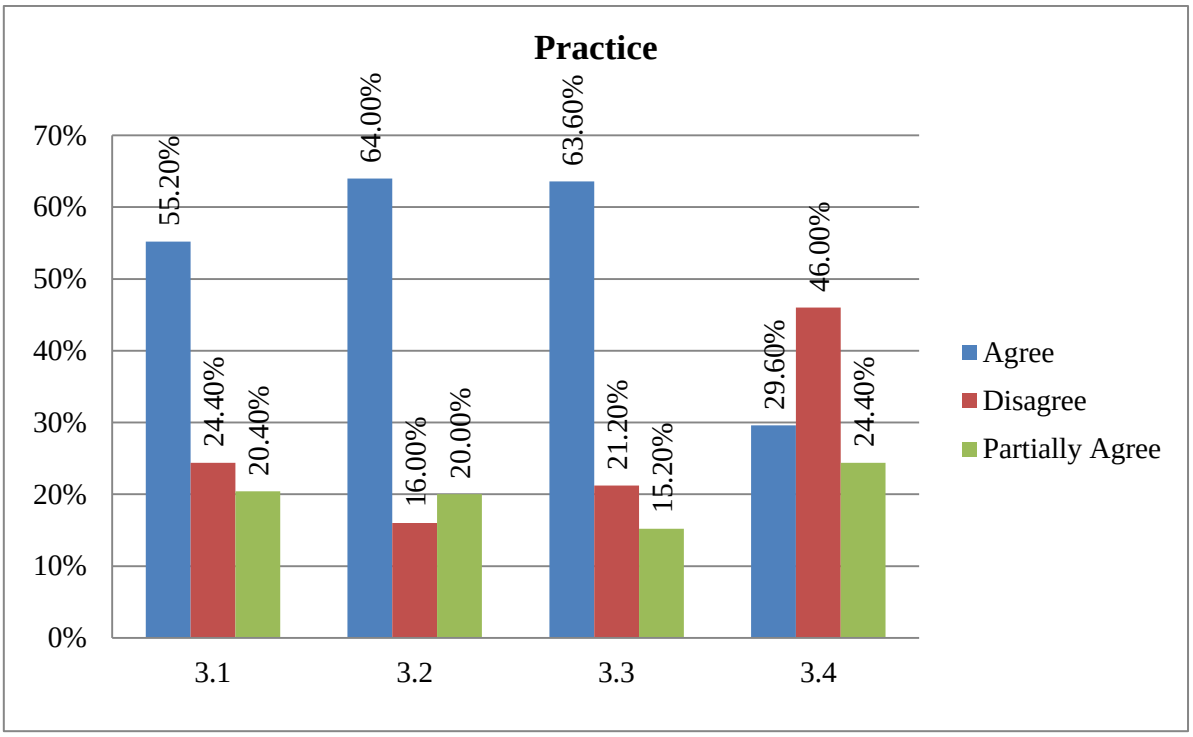


Graph 13: Bar diagram showing Attitude of subjects towards Diabetic Retinopathy

Table 14: Practices of subjects towards Diabetic Retinopathy

		Agree		Disagree		Partially Agree	
		Number	Percentage (%)	Number	Percentage (%)	Number	Percentage (%)
3.1	I go to the eye doctor regularly as advised by my family doctor, even if my vision is good	138	55.2	61	24.4	51	20.4
3.2	I control my blood sugar and lipid even if Diabetic Retinopathy is being treated	160	64.0	40	16.0	50	20.0
3.3	Staff in Eye unit counselled me about prevention and treatment for Diabetic Retinopathy and its complications	159	63.6	53	21.2	38	15.2
3.4	My vision due to Diabetic Retinopathy is less. Hence I'm using low vision devices	74	29.6	115	46.0	61	24.4

55.2% of the subjects agreed that 'I go to the eye doctor regularly as advised by my family doctor, even if my vision is good', 64% of the subjects were of the opinion that 'I control my blood sugar and lipid even if Diabetic Retinopathy is being treated', 63.6% of the subjects agreed that the staff in the eye unit counselled about prevention and treatment for Diabetic Retinopathy and its complications and 29.6% of the subjects felt that 'My vision due to Diabetic Retinopathy is less. Hence I'm using low vision devices'.

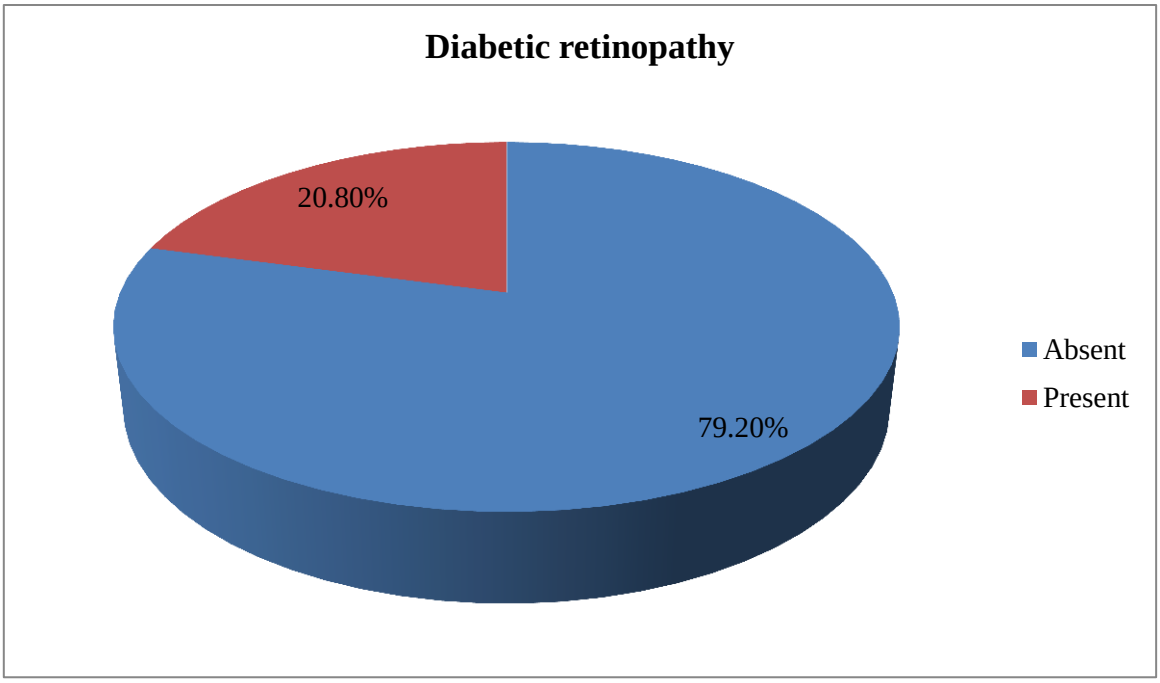


Graph 14: Bar diagram showing Practices of subjects towards Diabetic Retinopathy

Table 15: Prevalence of Diabetic Retinopathy among Diabetic subjects

Diabetic Retinopathy	Number	Percentage (%)
Absent	198	79.2
Present	52	20.8

In this study, 20.8% of the subjects of Diabetics had Diabetic Retinopathy.



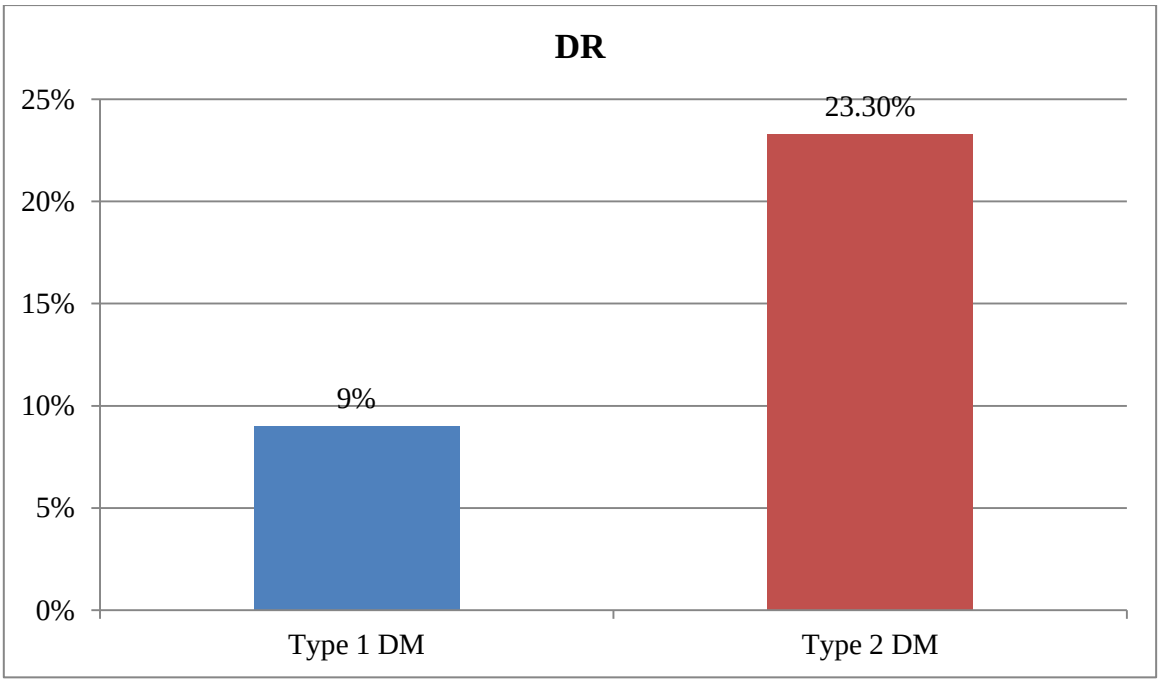
Graph 15: Pie diagram showing Prevalence of Diabetic Retinopathy among Diabetic subjects

Table 16: Association between Type of DM and Diabetic Retinopathy

DR	Type of DM			
	Type 1 DM		Type 2 DM	
	Number	Percentage (%)	Number	Percentage (%)
Absent	40	91	162	76.7
Present	4	9	48	23.3

$\chi 2 = 4.234$, df = 1, p = 0.03*

Among Type 1 DM subjects, 9% of the subjects had DR and among Type 2 DM, 23.3% of the subjects had DR. This difference in Diabetic Retinopathy between type 1 and type 2 DM was statistically significant.



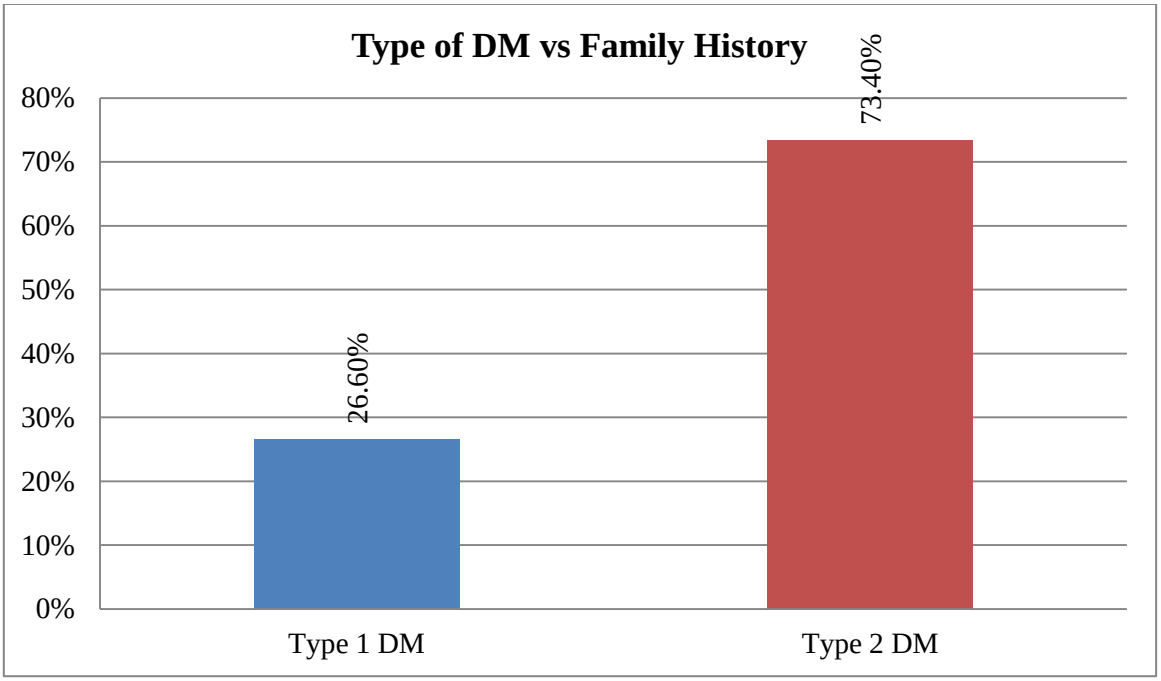
Graph 16: Bar diagram showing Association between Type of DM and Diabetic Retinopathy

Table 17: Association between Type of DM and Family History

		Type of DM			
		Type 1 DM		Type 2 DM	
		Number	Percentage (%)	Number	Percentage (%)
Family History	No	11	8.7	115	91.3
	Yes	33	26.6	91	73.4

$\chi 2 = 13.78$, df = 1, p <0.001*

Among type 1 DM, 26.6% of the subjects had a positive family history and among type 2 DM 73.4% of the subjects had a positive family history. This difference in family history with respect to type of DM was statistically significant.(p <0.001)



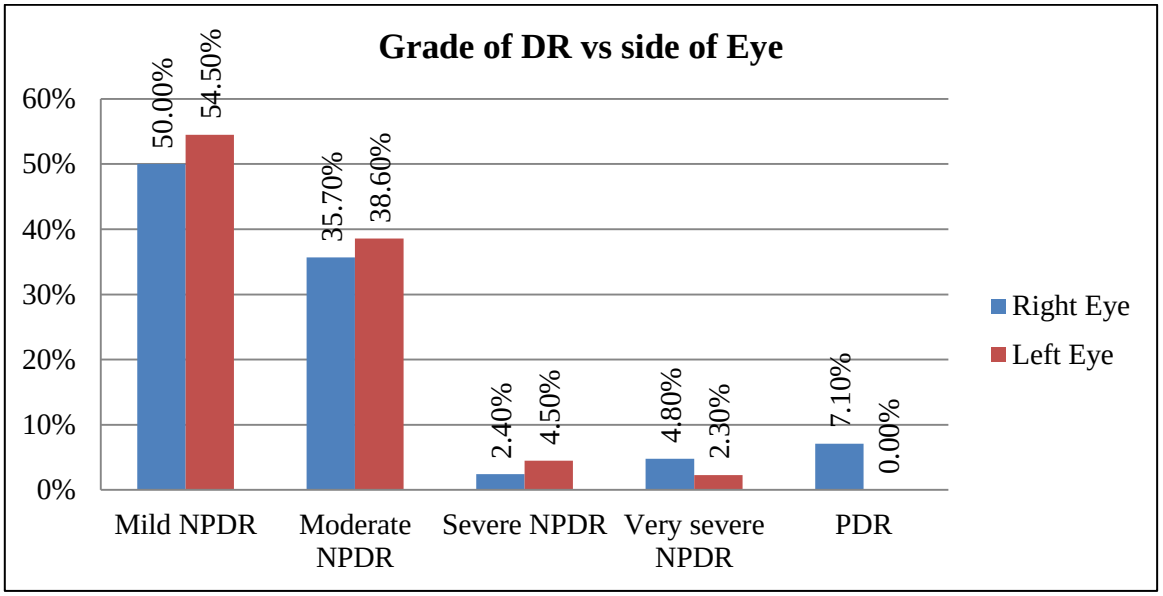
Graph 17: Bar diagram showing Association between Type of DM and Family History

Table 18: Grade of DR with respect to side of Eye

		Side				Total	
		Right Eye		Left Eye			
		Count	%	Count	%	Count	%
Grade of DR	Mild NPDR	21	50.0	24	54.5	45	52.33
	Moderate NPDR	15	35.7	17	38.6	32	37.21
	Severe NPDR	1	2.4	2	4.5	3	3.49
	Very severe NPDR	2	4.8	1	2.3	3	3.49
	PDR	3	7.1	0	0.0	3	3.49
	CSME	11		6			

In the study majority of subjects on both sides had Mild NPDR (50% in the right eye and 54.5% on left eye). 35.7% of the subjects on right side and 38.6% of the subjects on left side had Moderate NPDR, 2.4% of the subjects on right and 4.5% of the subjects on left side had Severe NPDR, 4.8% of the subjects on right and 2.3% of the subjects on left side had very severe NPDR and 7.1% of the subjects on right and no one on left side had PDR.

52.33% of the subjects eyes had mild NPDR, 37.21% of the subjects had moderate NPDR, 3.49% of the subjects had severe NPDR, Very severe NPDR and PDR respectively.



Graph 18: Bar diagram showing Grade of DR with respect to side of Eye

Table 19: Association between profile of Diabetics with respect to Diabetic Retinopathy

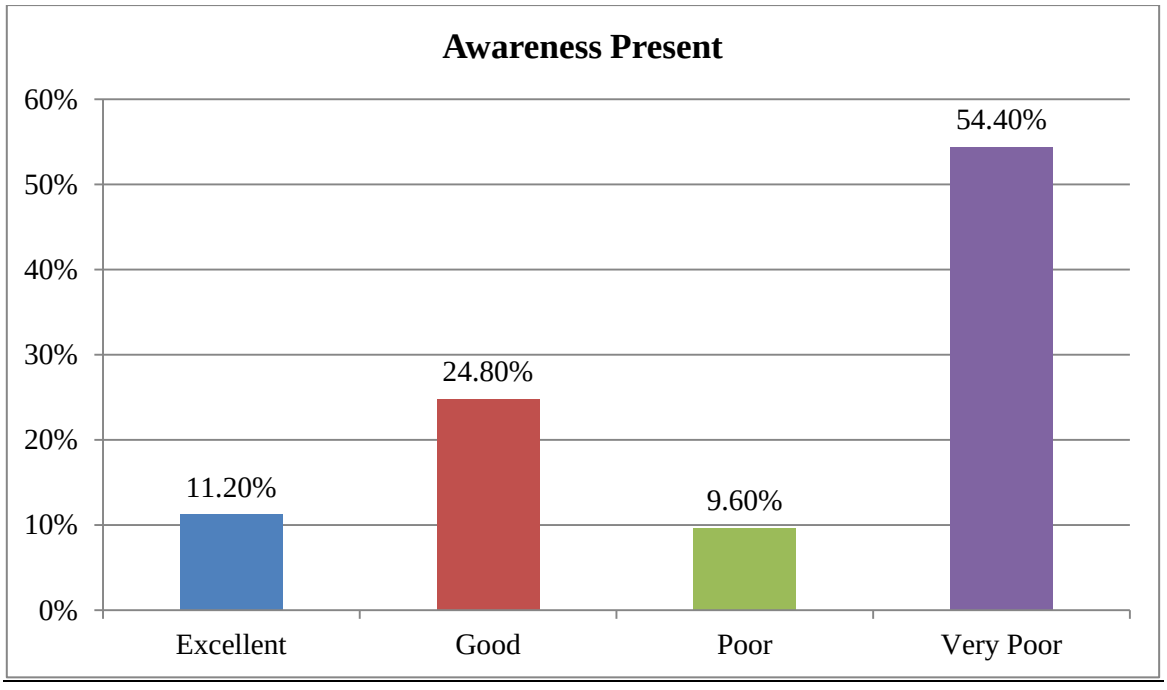
		Diabetic Retinopathy				P value
		Present		Absent		
		Number	Percentage (%)	Number	Percentage (%)	
Age	<30 years	0	0.0	9	4.5	0.346
	31 to 40 years	4	7.7	21	10.6	
	41 to 50 years	7	13.5	37	18.7	
	51 to 60 years	17	32.7	48	24.2	
	61 to 70 years	15	28.8	49	24.7	
	71 to 80 years	8	15.4	22	11.1	
	>80 years	1	1.9	12	6.1	
Gender	Female	18	34.6	83	41.9	0.339
	Male	34	65.4	115	58.1	
Family History	No	29	55.8	97	49.0	0.384
	Yes	23	44.2	101	51.0	
Duration of DM	< 1 year	4	7.7	53	26.8	0.003*
	1 to 5 years	20	38.5	70	35.4	
	6 to 10 years	14	26.9	58	29.3	
	11 to 15 years	9	17.3	11	5.6	
	16 to 20 years	4	7.7	5	2.5	
	>20 years	1	1.9	1	0.5	
Treatment	Insulin	14	26.9	20	10.1	0.004*
	Nil	2	3.8	21	10.6	
	OHA	36	69.2	157	79.3	

In our study, it showed that duration of Diabetes and duration of treatment were significantly associated with the development of Diabetic Retinopathy.

Table 20: Overall Awareness Regarding the Disease (DR)

Awareness	Number	Percentage (%)
Excellent	28	11.2
Good	62	24.8
Poor	24	9.6
Very Poor	136	54.4

In this study, 11.2% of the subjects had excellent, 24.8% of the subjects had good, 9.6% had poor and 54.4% of the subjects had very poor awareness regarding DR.

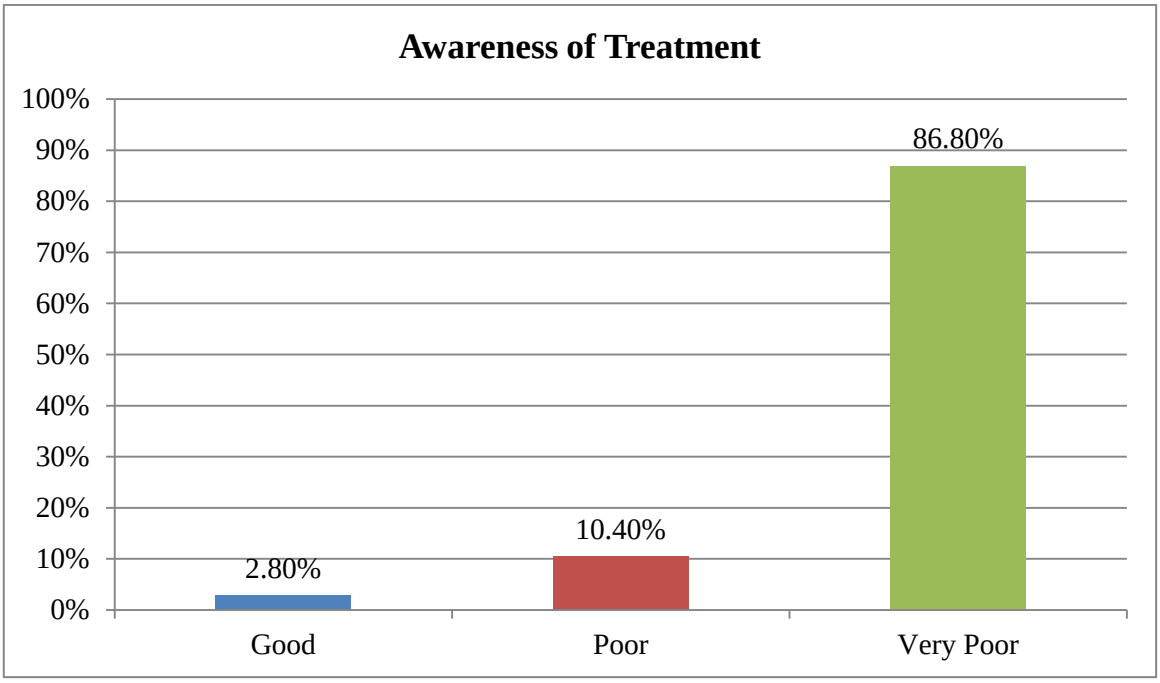


Graph 19: Bar diagram showing Overall awareness of disease among subjects

Table 21: Awareness Regarding Treatment

Awareness of Treatment	Number	Percentage (%)
Good	7	2.8
Poor	26	10.4
Very Poor	217	86.8

In our study, 2.8% of the subjects had good, 10.4% of the subjects had poor and 86.8% of the subjects had very poor awareness regarding treatment of Diabetic Retinopathy.

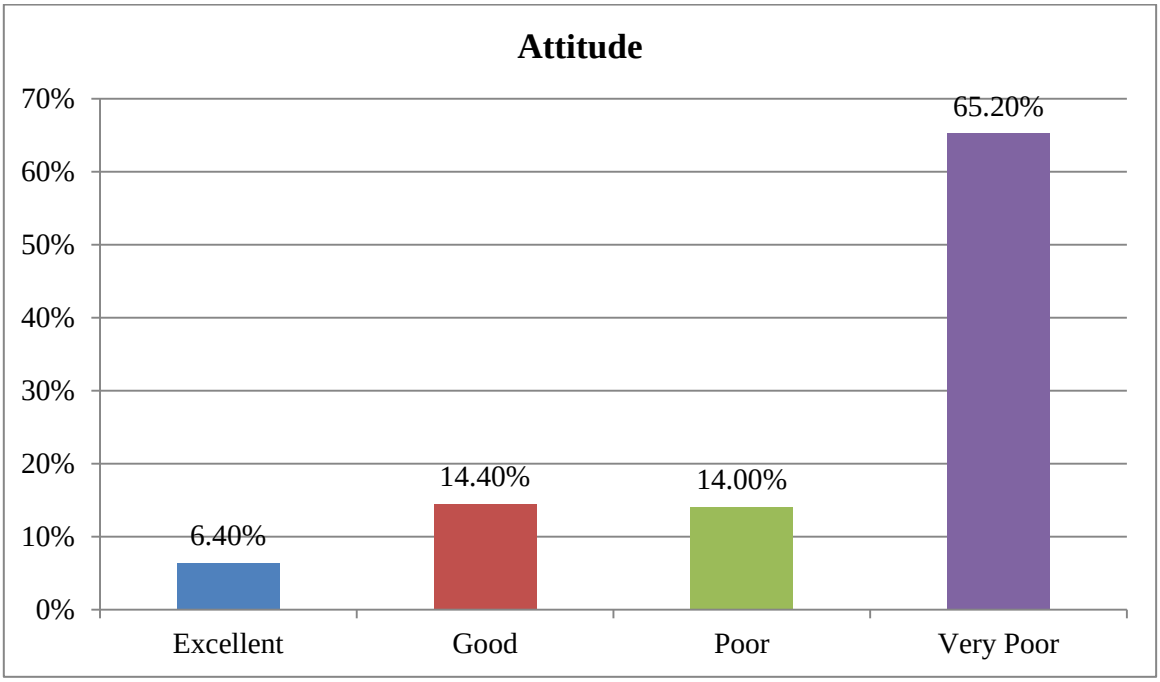


Graph 20: Bar diagram showing Awareness Regarding Treatment

Table 22: Attitude towards the Disease

Attitude	Number	Percentage (%)
Excellent	16	6.4
Good	36	14.4
Poor	35	14.0
Very Poor	163	65.2

In this study, 6.4% of the subjects had excellent, 14.4% of the subjects had good, 14% of the subjects had poor and 65.2% had very poor attitude.

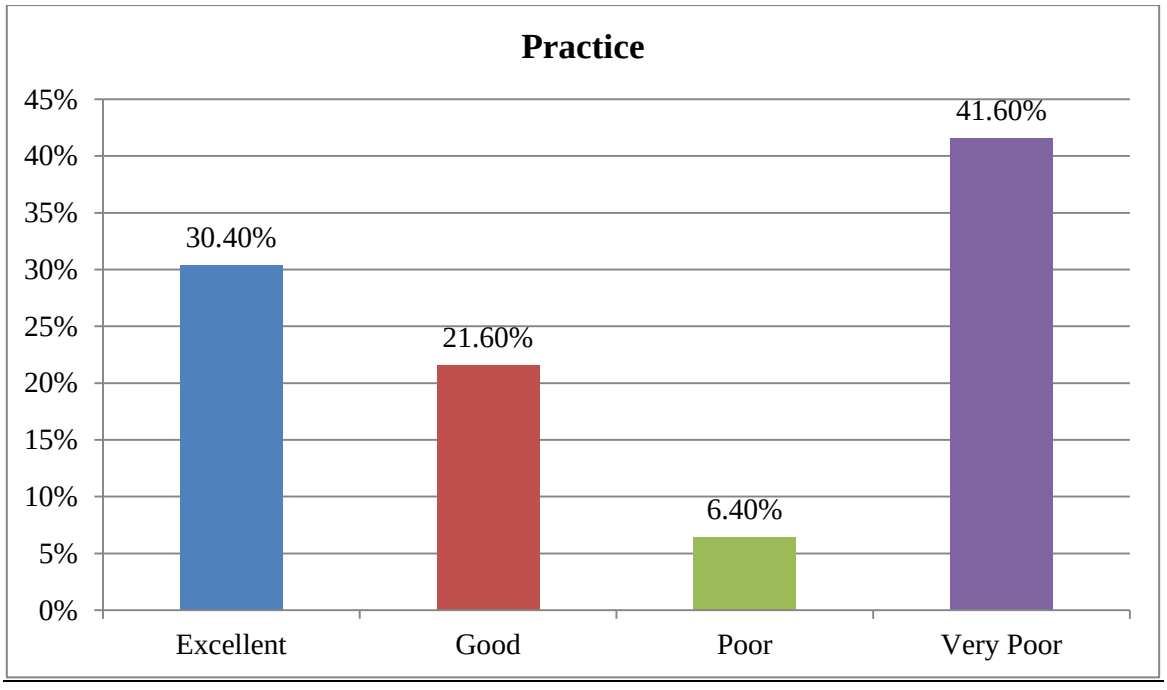


Graph 21: Bar diagram showing Attitude towards the Disease

Table 23: Practice among the subjects towards the disease

Practice	Number	Percentage (%)
Excellent	76	30.4
Good	54	21.6
Poor	16	6.4
Very Poor	104	41.6

In this study, 30.4% of the subjects had excellent, 21.6% of the subjects had good , 6.4% of the subjects had poor and 41.6% of the subjects had very poor practices regarding Diabetic Retinopathy.



Graph 22: Bar diagram showing Practice among the subjects towards the disease

Table 24: Association between overall Awareness and various parameters among Diabetic subjects

		Awareness								P value
		Excellent		Good		Poor		Very Poor		
		Number	%	Number	%	Number	%	Number	%	
Age	<30 years	3	10.7	3	4.8	1	4.2	2	1.5	0.001*
	31 to 40 years	3	10.7	11	17.7	1	4.2	10	7.4	
	41 to 50 years	4	14.3	13	21.0	1	4.2	26	19.1	
	51 to 60 years	10	35.7	18	29.0	5	20.8	32	23.5	
	61 to 70 years	6	21.4	14	22.6	13	54.2	31	22.8	
	71 to 80 years	1	3.6	1	1.6	1	4.2	27	19.9	
	>80 years	1	3.6	2	3.2	2	8.3	8	5.9	
Gender	Female	10	35.7	30	48.4	10	41.7	51	37.5	0.496
	Male	18	64.3	32	51.6	14	58.3	85	62.5	
Education	Illiterate	5	17.9	9	14.5	9	37.5	39	28.7	<0.001*
	Primary school	2	7.1	11	17.7	5	20.8	51	37.5	
	Secondary school	9	32.1	15	24.2	6	25.0	24	17.6	
	PUC	4	14.3	16	25.8	3	12.5	12	8.8	
	Graduate and above	8	28.6	11	17.7	1	4.2	10	7.4	
Type of DM	Type 1 DM	5	17.9	16	25.8	7	29.2	16	11.8	0.04*

	Type 2 DM	23	82.1	46	74.2	17	70.8	120	88.2	
Duration of DM	< 1 year	7	25.0	13	21.0	5	20.8	32	23.5	0.104
	1 to 5 years	13	46.4	26	41.9	7	29.2	44	32.4	
	6 to 10 years	5	17.9	14	22.6	6	25.0	47	34.6	
	11 to 15 years	2	7.1	7	11.3	2	8.3	9	6.6	
	16 to 20 years	1	3.6	2	3.2	4	16.7	2	1.5	
	>20 years	0	0.0	0	0.0	0	0.0	2	1.5	
Treatment	Insulin	5	17.9	13	21.0	4	16.7	12	8.8	0.231
	Nil	1	3.6	6	9.7	1	4.2	15	11.0	
	OHA	22	78.6	43	69.4	19	79.2	109	80.1	
Diabetic Retinopathy	Absent	25	89.3	52	83.9	21	87.5	110	80.9	
	Present	3	10.7	10	16.1	3	12.5	26	19.1	

In our study, there was significant association between overall awareness and age, education and type of DM. With other parameters there was no significant association with overall awareness.

Table 25: Association between Awareness regarding treatment and various parameters among Diabetic subjects

		Awareness of Treatment						P value
		Good		Poor		Very Poor		
		Number	%	Number	%	Number	%	
Age	<30 years	0	0.0	0	0.0	9	4.1	0.484
	31 to 40 years	1	14.3	3	11.5	21	9.7	
	41 to 50 years	1	14.3	2	7.7	41	18.9	
	51 to 60 years	2	28.6	5	19.2	58	26.7	
	61 to 70 years	3	42.9	8	30.8	53	24.4	
	71 to 80 years	0	0.0	7	26.9	23	10.6	
	>80 years	0	0.0	1	3.8	12	5.5	
Gender	Female	4	57.1	8	30.8	89	41.0	0.396
	Male	3	42.9	18	69.2	128	59.0	
Education	Illiterate	3	42.9	6	23.1	53	24.4	0.377
	Primary school	4	57.1	9	34.6	56	25.8	
	Secondary school	0	0.0	5	19.2	49	22.6	

	PUC	0	0.0	2	7.7	33	15.2	
	Graduate and above	0	0.0	4	15.4	26	12.0	
Type of DM	Type 1 DM	3	42.9	5	19.2	36	16.6	0.194
	Type 2 DM	4	57.1	21	80.8	181	83.4	
Duration of DM	< 1 year	3	42.9	7	26.9	47	21.7	0.027*
	1 to 5 years	0	0.0	8	30.8	82	37.8	
	6 to 10 years	1	14.3	4	15.4	67	30.9	
	11 to 15 years	2	28.6	4	15.4	14	6.5	
	16 to 20 years	1	14.3	2	7.7	6	2.8	
	>20 years	0	0.0	1	3.8	1	0.5	
Treatment	Insulin	2	28.6	6	23.1	26	12.0	0.363
	Nil	0	0.0	2	7.7	21	9.7	
	OHA	5	71.4	18	69.2	170	78.3	
Diabetic Retinopathy	Absent	5	71.4	19	73.1	184	84.8	0.224
	Present	2	28.6	7	26.9	33	15.2	

In this study, there was significant association between duration of DM and awareness of treatment. No significant association was observed between awareness of treatment and other parameters.

Table 26: Association between attitude and various parameters among Diabetic subjects

		Attitude								P value
		Excellent		Good		Poor		Very Poor		
		Number	%	Number	%	Number	%	Number	%	
Age	<30 years	1	6.2	2	5.6	3	8.6	3	1.8	0.082
	31 to 40 years	2	12.5	3	8.3	6	17.1	14	8.6	
	41 to 50 years	4	25.0	8	22.2	8	22.9	24	14.7	
	51 to 60 years	6	37.5	11	30.6	7	20.0	41	25.2	
	61 to 70 years	0	0.0	11	30.6	7	20.0	46	28.2	
	71 to 80 years	3	18.8	1	2.8	1	2.9	25	15.3	
	>80 years	0	0.0	0	0.0	3	8.6	10	6.1	
Gender	Female	5	31.2	14	38.9	12	34.3	70	42.9	0.666
	Male	11	68.8	22	61.1	23	65.7	93	57.1	
Education	Illiterate	4	25.0	2	5.6	5	14.3	51	31.3	<0.001*
	Primary school	5	31.2	7	19.4	6	17.1	51	31.3	
	Secondary school	2	12.5	13	36.1	9	25.7	30	18.4	

	PUC	2	12.5	5	13.9	12	34.3	16	9.8	
	Graduate and above	3	18.8	9	25.0	3	8.6	15	9.2	
Type of DM	Type 1 DM	2	12.5	8	22.2	9	25.7	25	15.3	0.394
	Type 2 DM	14	87.5	28	77.8	26	74.3	138	84.7	
Duration of DM	< 1 year	3	18.8	10	27.8	7	20.0	37	22.7	0.452
	1 to 5 years	4	25.0	13	36.1	19	54.3	54	33.1	
	6 to 10 years	8	50.0	8	22.2	9	25.7	47	28.8	
	11 to 15 years	1	6.2	3	8.3	0	0.0	16	9.8	
	16 to 20 years	0	0.0	2	5.6	0	0.0	7	4.3	
	>20 years	0	0.0	0	0.0	0	0.0	2	1.2	
Treatment	Insulin	1	6.2	7	19.4	5	14.3	21	12.9	0.359
	Nil	0	0.0	3	8.3	1	2.9	19	11.7	
	OHA	15	93.8	26	72.2	29	82.9	123	75.5	
Diabetic Retinopathy	Absent	14	87.5	31	86.1	32	91.4	131	80.4	0.382
	Present	2	12.5	5	13.9	3	8.6	32	19.6	

In our study, there was significant association between education status and attitude.

No significant association was observed attitude and other parameters.

Table 27: Association between practice and various parameters among Diabetic subjects

		Practice								P value
		Excellent		Good		Poor		Very Poor		
		Number	%	Number	%	Number	%	Number	%	
Age	<30 years	5	6.6	1	1.9	1	6.2	2	1.9	0.097
	31 to 40 years	8	10.5	5	9.3	4	25.0	8	7.7	
	41 to 50 years	16	21.1	6	11.1	3	18.8	19	18.3	
	51 to 60 years	27	35.5	12	22.2	3	18.8	23	22.1	
	61 to 70 years	16	21.1	17	31.5	3	18.8	28	26.9	
	71 to 80 years	2	2.6	10	18.5	1	6.2	17	16.3	
	>80 years	2	2.6	3	5.6	1	6.2	7	6.7	
Gender	Female	31	40.8	23	42.6	11	68.8	36	34.6	0.075
	Male	45	59.2	31	57.4	5	31.2	68	65.4	
Education	Illiterate	12	15.8	13	24.1	2	12.5	35	33.7	0.009*
	Primary school	14	18.4	16	29.6	4	25.0	35	33.7	
	Secondary school	20	26.3	13	24.1	3	18.8	18	17.3	
	PUC	18	23.7	5	9.3	3	18.8	9	8.7	
	Graduate and above	12	15.8	7	13.0	4	25.0	7	6.7	
Type of DM	Type 1 DM	16	21.1	8	14.8	1	6.2	19	18.3	0.500
	Type 2 DM	60	78.9	46	85.2	15	93.8	85	81.7	

Duration of DM	< 1 year	18	23.7	11	20.4	7	43.8	21	20.2	0.066
	1 to 5 years	35	46.1	16	29.6	5	31.2	34	32.7	
	6 to 10 years	17	22.4	17	31.5	2	12.5	36	34.6	
	11 to 15 years	6	7.9	6	11.1	0	0.0	8	7.7	
	16 to 20 years	0	0.0	4	7.4	2	12.5	3	2.9	
	>20 years	0	0.0	0	0.0	0	0.0	2	1.9	
Treatment	Insulin	12	15.8	9	16.7	1	6.2	12	11.5	0.246
	Nil	6	7.9	1	1.9	3	18.8	13	12.5	
	OHA	58	76.3	44	81.5	12	75.0	79	76.0	
Diabetic Retinopathy	Absent	67	88.2	42	77.8	14	87.5	85	81.7	0.416
	Present	9	11.8	12	22.2	2	12.5	19	18.3	

In this study, there was significant association between education status and positive practice. No significant association was observed practice and other parameters.

OTHER CO-EXISTING PATHOLOGIES FOUND:

RIGHT EYE – 61 subjects had lenticular opacity, 5 subjects with pseudophakia with posterior capsular opacity, 3 subjects with ARMD, 3 subjects with corneal opacity,2 subjects with glaucoma , 2 patients with aphakia, 1 subject with Optic Neuritis , traumatic optic neuropathy and phthisis bulbi, resopectively.

LEFT EYE - 65 subjects had lenticular opacity, 5 subjects with pseudophakia with posterior capsular opacity, 2 subjects with ARMD, 2 subjects with glaucoma and 1 subject with pseudophakic bullous keratopathy.

DISCUSSION

DISCUSSION

Diabetic retinopathy is an emerging public health problem with both medical and economic considerations involved. It is now considered as one of the most common causes of legal blindness among the working age individuals both in the developing and developed countries.

This study presents the number of diabetic retinopathy patients in a cross sectional study conducted on all diabetic patients attending the diabetic retinopathy eye camps conducted by a tertiary care hospital in Kolar district.

SOCIO-DEMOGRAPHIC CHARACTERISTICS OF THE STUDY:

1. **Age and Sex Distribution in study population**

In our study, majority of subjects were in the age group of 51 to 60 years (26%), 25.6% were in the age group 61 to 70 years. Mean age of subjects was 58.01 ± 14.28 years. Mean age of males were 59.2 ± 14.9 years and that of females was 56.3 ± 13.2 years. Prevalence was higher among those individuals of age >50years. Masalikoshimura et al., in their study observed that the mean age was 58.8 years.[149] P Namperumalsamy et al., found that the mean age was 47 years and found higher prevalence of Diabetic Retinopathy among patients >45 years of age.[146]

Out of the total study population of 250 cases, 149 cases were males (59.6%) while 101 cases (40.4%) were females. DR was more in males than females but was not statistically significant. P Namperumalsamy et al., found higher rates of diabetic retinopathy in the males compared to females (sex ratio 2: 1)[146] . Similar results have

been reported from the CURES Eye study[22], UKPDS study[147] and the Hyderabad study[148] thus supporting our results.

2. **Prevalence of Diabetic Retinopathy**

We observed different grades of diabetic retinopathy in 52 patients out of 250, i.e, prevalence of diabetic retinopathy in our study was 20.80% (65.40% were males and 34.60% were females). Among Type 1 DM subjects, 9% had DR and among Type 2 DM, 23.3% had DR. This difference in diabetic retinopathy between type 1 and type 2 DM was statistically significant (p = 0.03*). Our results are consistent with various other studies[145].

A study that was conducted by Himanshu K Nayak et al.[144] In Gujarat estimated that the prevalence of type 2 Diabetes was about 13.8%.

The National Urban Diabetes Survey (NUDS) [145] conducted a population based prevalence study in six metropolitan cities across India as shown below:

PLACE	**PREVALENCE (%)**
CHENNAI	13.5
BANGALORE	12.4
HYDERABAD	16.6
KOLKATA	11.7
NEW DELHI	11.6
MUMBAI	9.3

Table 28: Prevelance of diabetic retinopathy in India

Ramchandran et al., in 1999 observed retinopathy in 714 i.e. 23.7% cases out of 3010 patients of type 2 diabetes[150]. In various South Indian studies conducted to calculate prevalence by Raman et al.[151] showed a prevalence of 18.1%, another study conducted by Rema et al.,[22] showed a prevalence of 17.6%, a study by P Namperumalsamy et al.,[146] showed a prevalence of 10.6%, Narendran et al.,[152] showed a prevalence of 26.2% and by Dandona et al.,[148] a prevalence of 22.58%.

M.W. Knuiman reported prevalence of retinopathy at 28% in Perth, Western Australia[153].

Caird et al found a diabetic retinopathy prevalence rate of 36.8% in a survey that involved 4076 diabetic patients with over ten years duration of diabetes[154].

Following table showing studies on Prevalence of Diabetic Retinopathy in different populations[155]

POPULATIONS	**PLACE**	**PREVALENCE (%)**
CHENNAI URBAN RURAL EPIDEMOLOGY STUDY (CURES) - 1	CHENNAI, INDIA	17.6
LOS ANGELES LATIONO EYE STUDY (LALES)	LOS ANGELES, USA	46.9
LIVERPOOL DIABETIC EYE STUDY	LIVERPOOL , UK	33.6
BARBADOS EYE STUDY	BARBADOS, WESTINDIES	28.8
BLUE MOUNTAIN EYE STUDY (BMES)	AUSTRALIA	29
BEAVER DAM EYE STUDY (BCES)	WISCONSIN , USA	35.1
TAIWAN	TAIWAN, CHINA	35
WISCONSIN EPIDEMIOLOGIC STUDY OF DIABETIC RETINOPATHY	WISCONSIN,USA	50.3

Table 29 : Prevalence of Diabetic Retinopathy in different populations

3. **Family History and Diabetic Retinopathy**

In our study, 124 (49.6%) patients with diabetes had a positive family history. Of the patients with diabetic retinopathy, 23 patients (44.2%) had a positive family history of diabetes whereas 29 patients (55.8%) had a negative family history.

However, there was no statistical significance between family history and diabetic retinopathy in our study (P= 0.384) . The results in the Diabetes Control and Complications trial (DCCT) showed that family history of either diabetic nephropathy or retinopathy is associated with an increase in the risk of diabetic micro vascular complications among relatives with Type 1 Diabetes mellitus.[156] A study done by Reema M et al.,[22] concluded that the siblings of Type 2 diabetic patient with DR are 3.5 times more prone to develop retinopathy.

4. **Duration of Diabetes and Diabetic Retinopathy**

In our study 36% had DM for 1 to 5 years, 28.8% had DM for 6 to 10 years, 22.8% had DM <1 year.

In our study, 17.35 % of those with diabetes of duration less than 10 years had DR and 45.16 % of those with duration of more than 10 years had DR.

Dandona et al., reported that 87.5 % of those with diabetes for >15 year duration had DR when compared to 18.9% of those who had <15 year duration[148].

In the CURES Eye study, 41.8% had DR after 15 years of diabetes and the severity of DR proportionally increased with the duration of diabetes.[22] Also, it has been demonstrated that for every five year increase in duration of diabetes, the risk for DR increases by 1.89 times[22].

5. **Types of Diabetic Retinopathy**

Out of 52 (86 eyes) diabetic retinopathy patients, 83 eyes (96.48%) had Non Proliferative Diabetic Retinopathy (NPDR) while 3 eyes (3.49%) had Proliferative Diabetic Retinopathy (PDR).

NPDR Patients consisted of 45 eyes (52.33%) with Mild NPDR, 32 eyes (37.21%) with Moderate NPDR, 3 eyes (3.49%) with Severe NPDR and 3 eyes (3.49%) with Very severe NPDR. CSME was seen in 17 eyes (20.48%). Our results were in accordance to other studies.

	NPDR	PDR	MACULOPATHY
R.P Agrawal et al	79.8%	14.6%	5.8%
Narendran V et al.	94.1	5.9	29.4%
OUR STUDY	96.48%	3.49%	20.48%

Table 30 : Different grades of DR in different studies

6. **Treatment Modality and Diabetic Retinopathy**

Our study showed a higher prevalence of diabetic retinopathy in patients on Insulin and Oral hypoglycemic Agents (OHA) (26.90% and 69.20% respectively) in comparison to those patients not on any medication (3.80%) probably because of longer duration of diabetes or poor glycemic control prompting the use of Insulin/ Insulin combination with OHAs.

A similar correlation between the type of treatment i.e. insulin therapy (alone or in combination with OHA) was seen in a study conducted by R.A Agrawal et al.[21.]

In a study conducted by the AIOS, it was found that nearly 94.4% of the patients were taking treatment under the allopathic system of medicine for their DM: 81.1% with oral hypoglycaemic agents[157].

7. **Overall Awareness Regarding the DR**

In our study, it was found that 11.2% had excellent, 24.8% had good, 9.6% had poor and 54.4% had very poor awareness regarding diabetic retinopathy.

A study conducted in Oman by Rajiv et al., involving 750 participants, 'excellent', grade of knowledge about diabetes and eye care was present in 72.9% and 18% respectively. There was an 'excellent' grade in attitude, regarding the eye involvement and eye care was seen in 18% and 29.9% respectively. The practice for undergoing regular eye check-up and accepting timely treatment was of 'excellent' grade in 52% and 79.2% respectively.

In our study, 54% (135) were aware that diabetes affects the eye and 52.8% were aware that diabetes can damage eyesight leading to blindness.

In a study conducted by Srinivasan NK, et al., about KAP of diabetes and Diabetic Retinopathy in A Tertiary Eye Care Centre, it was found that, among the 288 subjects, 207 (71.9%) were 'aware' that eyes could be affected by diabetes, but only 49 patients (17.01%) were 'aware' of diabetic retinopathy as an ocular complication of diabetes[159].

This is similar to the results obtained in other studies conducted in South India by Hussain R[160] et al., and Rani PK et al.,[28] who reported good knowledge in 40.7% and 49.9% respectively, in the subjects of their studies .

However, in another study done in South India by Babu N et al., only 28% of the population was 'aware' of diabetes [156].

Another study that was conducted in South India involved assessing awareness of diabetic retinopathy before and after DR awareness campaign, showed that only 55% of paramedical personnel and 7% of community members were aware of diabetic retinopathy in the beginning which increased to 79% and 68% respectively.[161].

In a study conducted by Rajani Kadri, awareness of DR was found in 27.9% patients whereas awareness of treatment for diabetic retinopathy was seen in 9% patients[162].

However, in a study done in South India by Mahesh G et al.,[163] 36.31% felt that they were aware about retinopathy, while 30.9% of the patients in a study done in North India by Koshy J et al.,[29] were aware that diabetes could lead to retinal disease .

Das T et al., also reported poor knowledge of retinopathy among the patients in their study conducted in Eastern India [164].

A study done in Singapore[165], a developed country, showed an awareness rate of about 70 – 80%. This state of decreased awareness of diabetic retinopathy in developing countries demands for many more health programmes for educating the public.

In a study conducted to assess the awareness of diabetic retinopathy among diabetic patients in Nigeria, it was found that the awareness rate amongst the patients was quite high (84.3%) [166].

Another study which was conducted in Malaysia to assess the awareness of eye complications and the prevalence of diabetic retinopathy in their first visit to eye clinic among diabetic patients. 86% of the

participants were aware of diabetic eye complications, especially in patients who had tertiary level of education (96.3%) [167].

The impact of literacy on increased awareness of eye diseases was a known fact in earlier studies. Similar results were seen in our study as well and it was found to be statistically significant.

Many studies have showed that the duration of the disease has an important role to play in the awareness of retinopathy. The awareness about diabetic retinopathy and its complications increases with the increase in the duration of illness.[29]

In our study, no such correlation was found between duration and awareness.

AWARENESS ABOUT TREATMENT

In our study, a mere 2.8% of patients were aware of treatment options for DR. This shows how important the awareness creation is in rural areas. Only 12.8% of the patients had an idea about the various modalities of treatment available in the treatment of DR. 35.5% thought that treatment with laser is painful and 16.4% believed that it should therefore be avoided.

In a study done by Koshy J,et al., only 32.6% of patients knew the various treatment modalities for diabetic retinopathy and out of these 7.7% had heard or knew anything about laser treatment for diabetic retinopathy. This demonstrated that there is a great need for health education to increase the level of awareness and knowledge of systemic disease related eye problems[29].

ATTITUDE TOWARDS THE DISEASE

We found that only 52 patients (20.8%) in our study had a positive attitude towards diabetes.

Hussain R et al.,[160] found a positive attitude towards diabetes in 53.8% of the diabetic patients in their study. In contrast to this Srinivasan NK et al.,[159] found that only 84 patients (29.2%) in their study had a positive attitude towards diabetes.

PRACTICE PATTERNS

We found that 130 patients (52%) in our study had good practice patterns with respect to diabetes.

Srinivasan NK et al.,[159] found that good practice was found in 158 patients (54.9%) when compared to 57.6% and 48.45% of the subjects in the studies by Hussain R et al., and Rani PK et al.,[28] respectively, were reported to have good practice patterns .

In our study 44% of patients believed that if their vision is good, then their eyes are not affected in diabetes, so they do not need annual eye testing. A huge, 68.4% believed that the information on eye problems due to diabetic retinopathy should be given by an eye doctor only. 36.8% believed that if they are taking eye treatment, they need not worry about controlling their sugar and lipids.

The Chengamanad Diabetic Retinopathy Awareness Study (CDRAS), tried to determine the level of awareness of diabetic population regarding the blinding complications of diabetic retinopathy. This study showed that 63.7% felt they were not educated about the complications of diabetic retinopathy, and 61.5% believed that they should have been informed about eye related problems by their treating physicians[163].

67.2% of subjects disagreed that people with diabetes often waste their time and money showing that a majority of the people are aware that regular checkup and evaluation is beneficial for them.

CONCLUSION

CONCLUSION

Diabetic retinopathy is a major health problem in patients with diabetes.

Risk factors such as age, duration of diabetes, hypertension, hyperglycaemia, hyperlipidaemia etc., should be taken into consideration for regular check-ups and early detection of diabetic retinopathy.

Awareness and knowledge about diabetic retinopathy was poor among the patients in our study. Lack of knowledge regarding the need for screening for diabetic retinopathy was found to be a major barrier to compliance with regular screening. Good knowledge about diabetic retinopathy was associated with a positive attitude towards diabetes and good practice patterns with respect to retinopathy. Therefore, there is an urgent need to develop strategies to educate diabetic patients about this potentially blinding complication of diabetes.

As new therapies for diabetic retinopathy and its complications emerge, the need to collect and monitor new epidemiological data becomes increasingly important to be able to evaluate the impact and effectiveness of these therapies.

Given the large number of diabetic subjects in India, even with the low prevalence rates, the actual number of patients with diabetic retinopathy would be large. This highlights the need for routine retinal screening of diabetic individuals to detect DR and prevent visual impairment.

SUMMARY

SUMMARY

Our study was conducted on 250 diabetic patients comprising of 149 male (59.6%) and 101 female (40.4%) patients attending diabetic eye camps conducted in Kolar district. Prevalence of diabetes among the study population was found to be 20.80 %. Among Type 1 DM subjects, 9% had DR and among Type 2 DM, 23.3% had DR. This difference in diabetic retinopathy between type 1 and type 2 DM was statistically significant.

$p = 0.03$*

Mean age of the patients was found to be 58.01± 14.28 years. Mean age among males was 59.2 ± 14.9 years and among females was 56.3 ± 13.2 years. . Majority of the patients having Diabetic retinopathy (32.7%) were in the age group 51-60 years followed by patients of 61-70 years (28.8%). In our study, 17.35 % of those with diabetes of duration less than 10years had DR and 45.16 % of those with duration of more than 10years had DR.

Diabetic retinopathy patients comprised of 34 males and 18 females i.e. 65.4% of the patients were males, showing male preponderance. Prevalence of NPDR was 96.40% and PDR was 3.49%.

Among NPDR Patients, NPDR consisted of 45 eyes (52.33%) with Mild NPDR, 32 eyes (37.21%) with Moderate NPDR, 3 eyes (3.49%) with Severe NPDR and 3 eyes (3.49%) with Very severe NPDR. PDR was present in 3eyes (3.49%). CSME was seen in 17 eyes (20.48%). Our study showed a higher prevalence of Diabetic retinopathy in patients on Insulin and Oral hypoglycemic Agents (OHA) (26.90% and 69.20% respectively) in comparison to those patients not on any medication (3.80%)

Among type 1 DM, 26.6% had a positive family history and among type 2 DM 73.4% had family history. This difference in family history with respect to type of DM was statistically significant. ($p < 0.001$).

Duration of diabetes proved to be an important association of Diabetic Retinopathy in our study ($p = 0.003$). Age, gender and family history were not statistically significant risk factors of diabetic retinopathy in our study.

In this study 11.2% had excellent, 24.8% had good, 9.6% had poor and 54.4% had very poor awareness regarding DR. There was significant association between overall awareness and age, education and type of DM (Type 2). With other parameters there was no significant association seen.

In this study 2.8% had good, 10.4% had poor and 86.8% had very poor awareness regarding treatment of diabetic retinopathy. Significant association was observed between duration of DM and awareness of treatment. No significant association was observed between awareness of treatment and other parameters.

In our study 6.4% had excellent, 14.4% had good, 14% had poor and 65.2% had very poor attitude towards the disease. There was significant association between education status and attitude. No significant association was observed attitude and other parameters.

In our study 30.4% had excellent, 21.6% had good, 6.4% had poor and 41.6% had very poor practices regarding diabetic retinopathy. In the study there was significant association between education status and positive practice. No significant association was observed in practice and other parameters.

BIBLIOGRAPHY

BIBLIOGRAPHY

1) Wild S, Roglic G, Green A, Sicree R, King H. Global Prevalence of diabetes: estimates for the year d 2000 and projections for 2030. Diabetes Care. 2004; 27:1047-53.

2) International Diabetes Federation. IDF Diabetes Atlas, 2nd edn. Brussels, Belgium: International Diabetes Federation, 2003.

3) International Diabetes Federation. IDF Diabetes Atlas, 3rd edn. Brussels, Belgium: International Diabetes Federation, 2007.

4) Mohan V, Ranchandran A, Snehalata C, Mohan R, Bharani G, Viswanathan M. High prevalence of maturity onset diabetes of the young (MODY) among Indians. Diabetes Care. 1985;8:371-74.

5) Joshi SR. Metabolic Syndrome: emerging clusters of the Indian phenotype. J Assoc Physicians. 2003; 51:445-56.

6) Vishvanathan M, Mohan V, Snehalata C, Ramchandran A. High prelavence of Type II (noninsulin dependent) diabetes among the offspring of conjugal Type II diabetic parents in India. Diabetologia. 1985; 28:907-10

7) Mohan V, Shanthirani CS, Deepa R. Glucose intolerance (diabetes and IGT) in a selected South Indian population with special reference to family history, obesity and lifestyle factors: The Chennai Urban Population Study (CUPS 14). J Assoc Physicians India. 2003; 51:771-77.

8) Ramchandran A. Epidemiology of diabetes research in India – Three decades of research. J Assoc Physicians. 2005; 53:34-38.

9) Health situation in the South-East Asia Region 1998-2000. WHO Regional Office for South East Asia 2002; New Delhi.

10) Ramchandran A, Snehalata C, Vijay V. Explosion of type II diabetes in Indian subcontinent. International Diabetes Monitor. 2003;15:1-6.

11) Klein R, Klein BEK, Moss SE, Davis MD, DeMets DL. The Wisconsin Epidemiologic Study of Diabetic Retinopathy. II. Prevalence and risk of diabetic retinopathy when age at diagnosis is less than 30 years. Arch Ophthalmol. 1984;102:520-6.

12) Corrêa, Silva ZMS, Moraes FA, Mundialino MI. Risk factors related to the severity of diabetic retinopathy. Arq Bras Oftalmol. 2003; 66: 739-743.

13) Klein R, Klein B. Epidemiology of Proliferative Diabetic Retinopathy. Diabetes Care. 1992;15: 1875-91.

14) Klein R, Knudtson MD, Lee KE, Gangnon R, Klein BEK. The Wisconsin Epidemiologic Study of Diabetic Retinopathy XXII. The Twenty-Five-Year Progression of Retinopathy in Persons with Type 1 Diabetes. Ophthalmology. 2008; 115 :1859-68.

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

16) Dandona R, Dandona L, John RK, McCarty CA, Rao GN. Awareness of eye diseases in an urban population in southern India.Bulletin of the World Health Organisation. 2001; 79: 96-102.

17) Bollu M, Nalluri KK, Prakash AS, Lohith MN, Venkataramarao N. Study of knowledge, attitude, and practice of general population of Guntur toward silent killer diseases: hypertension and diabetes. Asian J Pharm Clin Res. 2015; 8:74-78.

18) Murugesan N, Snehalatha C, Shobhana R, Roglic G, Ramachandran A. Awareness about diabetes and its complications in the general and diabetic population in a city in southern India. Diabetes Res Clin Pract. 2007; 77: 433-7.

19) Ahuja MMS. Epidemiological studies on diabetes mellitus in India. Epidemiology of diabetes in developing countries . New Delhi. 1979; 29-38.

20) Mohan V, Mathur P, Deepa R, Deepa M, Shukla DK. Urban rural differences in prevalence of self-reported diabetes in India- The WHO-ICMR Indian NCD risk factor surveillance. Diabetes Res Clin Pract .2008; 80: 159–68.

21) Agrawal RP, Ranka M, Beniwal R, Gothwal SR, Jain GC, Kochar DK, Kothari RP. Prevalence of Diabetic Retinopathy in Type 2 Diabetes in Relation to Risk Factors: Hospital Based Study. Int J Diab Dev Countries. 2003; 23: 16-19.

22) Rema M, Premkumar S, Anitha B, Deepa R, Pradeepa R, Mohan V. Prevalence of diabetic retinopathy in Urban India: The Chennai Urban Rural Epidemiology Study (CURES) Eye Study - 1. Invest Ophthal Vis Sci. 2005; 46:2328-33.

23) Gupta S, Ambade A. Prevalence of Diabetic Retinopathy and influencing factors amongst Type II diabetics from Central India. Int J Diab Dev Countries. 2004; 24:75-78.

24) Nirmalan PK, Katz J, Robin AL, Tielsch JM, Namperumalsamy P, Kim R et al. Prevalence of vitreoretinal disorders in a rural population of southern India: the Aravind Comprehensive Eye Study. Arch Ophthalmol .2004;122:581– 6.

25) Ramachandran A, Snehalatha C, Kapur A, Vijay V,Mohan V, Das AK, et al.High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. Diabetologia. 2001;44:1094 –101.

26) Agarwal S, Raman R, Paul PG, Rani PK, Uthra S, Gayathree R , et al. Sankara Nethralaya-Diabetic Retinopathy Epidemiology and Molecular Genetic Study

(SN-DREAMS 1): study design and research methodology. Ophthalmic Epidemiol. 2005;12:143–53.

27) Rao CR, Kamath VG, Shetty A, Kamath A. A study on the prevalence of type 2 diabetes in coastal Karnataka. Int J Diab Dev Countries. 2010; 30:80-85.

28) Rani PK, Raman R, Subramani S, Perumal G, Kumaramanickavel G, Sharma T. Knowledge of diabetes and diabetic retinopathy among rural populations in India, and the influence of knowledge of diabetic retinopathy on attitude and practice. Rural Remote Health. 2008;8:838.

29) Koshy J, Varghese DL, Mathew T, Kaur G, Thomas S, Bhatti SM. Study of KAP of ocular complications due to diabetes among type II diabetics visiting a tertiary teaching hospital. Indian J Community Health. 2012; 24: 27–31.

30) Stults-Kolehmainen MA, Sinha R. The Effects of Stress on Physical Activity and Exercise. Sports Med. 2014; 44:81-121.

31) Satyanarayana TB, Mahendrappa SK. A cross sectional study of knowledge, attitude and practice among patients with type 2 diabetes mellitus at a tertiary care hospital. J of Evolution of Med and Dent Sci. 2014;3:5317-21.

32) Francis A, L'esperance, Jr., Willim A, James. . Diabetic Retinopathy: Clinical Evaluation and Management. London: CV Mosby and Company; 1981.

33) Wu L, Fernandez-Loaiza P, Sauma J, Hernandez-Bogantes E, Masis M. Classification of diabetic retinopathy and diabetic macular edema. World J Diabetes. 2013;4 :290-94.

34) Abe H, Hoshi M. Diabetic Microangiopathy. International Symposium on Epidemiology of Diabetic Microangiopathy. Osaka :Karger; 1982.p.39-45.

35) Haut J, Redor JY, Abboud E, van Effenterre G, Moulin F. Classification of diabetic retinopathy.Ophthalmologica. 1987;195:145-55.

36) Dubey AK, Nagpal P, Chawla S, Dubey B. A proposed new classification for diabetic retinopathy: The concept of primary and secondary vitreopathy. Indian J Ophthalmol . 2008;56:23-9.

37) Sir Stewart Duke Elder. System of Ophthalmology. 1st ed.St. Louis: Mosby ; 1965. p. 123-60.

38) Albert, Jakobiec. Principles and practice of ophthalmology. 3rd ed. Netherlands : Elsevier; 2008. p. 1743 -1800.

39) Grading diabetic retinopathy for stereoscopic colour fundus photographs an extension of the modified airline house classification, ETDRS report number 10. Early treatment diabetic retinopthy study research group. Ophthalmology. 1991;98:786-806

40) Kanski JJ.Clinical Ophthalmology:A systemic approach. 6th ed. Netherlands: Elsevier; 2009. p.566-584.

41) Classification of diabetic retinopathy from fluorescein angiograms: ETDRS report number 11. Ophthalmology. 1991;98:807-22.

42) Wilkinson CP, Ferris FL, Klein RE, Lee PP, Agardh CD, Davis M, et al. Proposed international clinical diabetic retinopathy and diabetic macular edema disease severity scales. Ophthalmology. 2003;110:1677-82.

43) The Diabetic Study Research Group: A modification of the Airlie House Classification of Diabetic Retinopathy (DRS report no. 7). Invest Ophthalmol Vis Sci. 1981;21:210-26.

44) David J. Browning. Diabetic Retinopathy: Evidence- Based Management. 1st ed. New York: Springer;1982. p.10-17.

45) Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001; 414:813–20.

46) Guigliano D, Ceriello A, Paolisso G. Oxidative stress and diabetic vascular complications. Diabetes Care. 1996; 19:257–67.

47) Robinson . Diabetic Eye Disease. Oxford: Butterworth Heimann.2000; p.40.

48) Agaki Y, Kador PF, Kuwabara T, Kinoshita JH .Aldose reductase localization in human mural cells. Invest Ophthalmol Visc Sci.1983 ; 24: 1516-19.

49) No authors listed.A Randomized Trial of Sorbinil, an Aldose Reductase Inhibitor, in Diabetic Retinopathy. Arch Ophthalmol.1990;108:1234–44.

50) Singh VP, Bali A, Singh N, Jaggi AS. Advanced Glycation End Products and Diabetic Complications. Korean J Physiol Pharmacol. 2014;18:1-14.

51) Huijberts MSP, Wolffenbuttel BH, Boudier HA, Crijns FR, Kruseman AC, Poitevin P, et al . Aminoguanidine treatment increases elasticity and decreases fluid filtration of large arteries from diabetic rats. J Clin Invest. 1993;92:1407–11.

52) Kalfa TA, Gerristen ME, Carlson, EC, Binstock AJ, Tsilibary EC. Altered proliferation of retinal vascular cells on a glycated matrix .Inv Ophthalmol Vis Sci. 1995; 36:2358-67.

53) Hammes HP, Strodler D, Weiss A. Secondary intervention with aminoguanidine retards the progression of diabetic retinopathy in rat model. Diabetologica. 1995;38:656-60.

54) Lu M, Kuroki M, Amano S, Tolentino M, Keough K, Kim I. Advanced glycation end products increase retinal vascular endothelial growth factor expression. J Clin Invest. 1998;101:1219–24.

55) Koya D, King GL. Protein kinase C activation and the development of diabetic complications. Diabetes. 1998;47: 859–66.

56) Portilla D, Dai G, Peters JM, Gonzalez FJ, Crew MD, Proia AD. Etomoxir-induced PPARalpha-modulated enzymes protect during acute renal failure. Am J Physiol Renal Physiol. 2000;278: 66-75.

57) Keough RJ, Dunlop ME, Larkins RG. Effect of inhibition of aldose reductase on glucose flux, diacylglycerol formation, protein kinase C, and phospholipase A2 activation. Metabolism. 1997;46:41–47.

58) Ishii H, Jirousek MR, Koya D, Takagi C, Xia P, Clermont A. Amelioration of vascular dysfunctions in diabetic rats by an oral PKC beta inhibitor. Science. 1996;272:728–31.

59) Stephen J Ryan. Retina . 5th ed. Baltimore: CV Mosby and Company; 2012.

60) Sayin N, Kara N, Pekel G. Ocular complications of diabetes mellitus. World J Diabetes. 2015;6:92-108

61) Barot M, Gokulgandhi MR, Patel S, Mitra AK.Microvascular complications and diabetic retinopathy: recent advances and future implications. Future Med Chem.2013;5:301–14.

62) Durham JT, Herman IM. Microvascular modifications in diabetic retinopathy. Curr Diab Rep.2011; 11: 253–64.

63) Forbes JM, Cooper ME. Mechanisms of diabetic complications. Physiol Rev. 2013;93:137–88.

64) Goldberg RB.Cytokine and cytokine-like inflammation markers, endothelial dysfunction, and imbalanced coagulation in development of diabetes and its complications.J Clin Endocrinol Metab. 2009 ;94:3171-82 .

65) de Gooyer TE, Stevenson KA, Humphries P, Simpson DA, Gardiner TA, Stitt AW. Retinopathy is reduced during experimental diabetes in a mouse model of outer retinal degeneration. Invest Ophthalmol Vis Sci. 2006;47:5561–8

66) Ellis TP, Choudhury RH, Kaul K, Chopra M, Kohner EM, Tarr JM, Chibber R. Diabetic retinopathy and atherosclerosis: is there a link?.Curr Diabetes Rev. 2013;9:146-60.

67) Zhang W, Liu H, Al-Shabrawey M, Caldwell RW, Caldwell RB. Inflammation and diabetic retinal microvascular complications. J Cardiovasc Dis Res. 2011;2:96–103.

68) Patel JI, Saleh GM, Hykin PG, Gregor ZJ, Cree IA. Concentration of haemodynamic and inflammatory related cytokines in diabetic retinopathy.Eye . 2008 ;22:223-8.

69) Hernández C, Segura RM, Fonollosa A, Carrasco E, Francisco G, Simó R. Interleukin-8, monocyte chemoattractant protein-1 and IL-10 in the vitreous fluid of patients with proliferative diabetic retinopathy.Diabet Med. 2005;22:719-22.

70) Murugeswari P, Shukla D, Kim R, Namperumalsamy P, Stitt AW, Muthukkaruppan Angiogenic potential of vitreous from Proliferative Diabetic Retinopathy and Eales' Disease patients.VPLoS One. 2014;9:107-51.

71) Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001;414:813-20.

72) Semeraro F, Cancarini A, dell’ Omo R, Rezzola S, Romano MR, Costagliola C. Diabetic Retinopathy: Vascular and Inflammatory Disease. J Diabetes Res. 2015;2015:582060.

73) Klein R, Klein BEK, Moss SE, Davis MD, DeMets DL. The Wisconsin Epidemiologic Study of Diabetic Retinopathy. II. Prevalence and risk of diabetic retinopathy when age at diagnosis is less than 30 years. Arch Ophthalmol .1984;102:520-6.

74) Lang GE .Diabetic Retinopathy. Dev Ophthalmol. 2007; 39:1-12.

75) Bhagat N, Grigorian RA, Tutela A, Zarbin MA. Diabetic macular edema: pathogenesis and treatment. Surv Ophthalmol. 2009;54:1-32.

76) Aiello LP, Avery RL, Arrigg PG, Keyt BA, Jampel HD, Shah ST, et al. Vascular endothelial growth factor in ocular fluids of patients with diabetic retinopathy and other retinal disorders. N Engl J Med .1994;331:1980-7.

77) Murata T, Ishibashi T, Khalil A , Hata Y, Yoshikawa H, Inomata H. Vascular endothelial growth factor plays a role in hyperpermiability of diabetic retinal vessels. Ophthalmic Res. 1995;27:48- 52.

78) Collins PD, Connolly DT, Williams TJ. Characterization of the increased in vascular permeability induced by vascular permeability factor in vivo. Br J Pharmacol .1993;109:195-9.

79) Aiello LP The potential role of PKC beta in diabetic retinopathy and macular edema. Surv Ophthalmol. 2002;47:263-9

80) Bhagat N, Grigorian RA, Tutela A, Zarbin MA.Diabetic macular edema: pathogenesis and treatment. Surv Ophthalmol. 2009;54:1-32.

81) Klein R, Klein Be, Moss SE, Davis MD, DeMets DL. The Wisconsin epidemiologic study of diabetic retinopathy III. Prevalence and risk of diabetic retinopathy when age at diagnosis is 30 or more years. Arch Ophthalmol.1984;102:520-26.

82) Fong DS, Aiello LP, Gardner TW, King GL, Blankenship G, Cavallerano JD . Retinopathy in diabetes. Diabetes Care.2004;27 : 84-87.

83) Albert DM, Jakobie FA. Diagnosis, management and treatment of macular edema, principles and practice of ophthalmology. Christchurch : Saunders and company. 1994; p. 747-82.

84) Klein R, Klein BE. Relation of glycemic control of diabetic complication and health outcomes. Diabetes Care.1998;21:39-43.

85) UK Prospective diabetes study (UKPDS) group. Intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type-2 diabetes (UKPDS 33). Lancet.1998;352:837-53.

86) Das A, Shah VO, Dorin RI, Zager PG. Biochemical and molecular mechanisms of diabetic retinopathy.Invest Ophthalmol Vis Sci.1996;3:105.

87) Van Leiden HA, Dekker JM, Moll AC, Nijpels G, Heine RJ, Bouter LM, et al. Blood pressure, lipids, and obesity are associated with retinopathy : the hoorn study. Diabetes Care.2002;25:1320.

88) Chew EY, Klein ML, Ferris FL III, Remaley NA, Murphy RP, Chantry K . Association of elevated serum lipid levels with retinal hard exudates in diabetic retinopathy : Early treatment diabetic retinopathy study (ETDRS) report 22. Arch Ophthalmol. 1996;114:1079-84.

89) Klein Be, Moss SE, Klein R, Surawicz TS. The Wisconsin epidemiologic study of diabetic retinopathy, XIII : relationship of serum cholesterol to retinopathy and hard exudates. Ophthalmology. 1991; 98: 1261-65.

90) Sheth HG, Aslam S, Davies N. Lipid lowering drugs in diabetes : lipid lowering has ophthalmic benefits. Br Med J. 2006;332 : 1272-73.

91) Van Leiden HA, Dekker JM, Moll AC, Nijpels G, Heine RJ, Bouter LM, Stehouwer CD, Polak BC. Blood pressure, lipids, and obesity are associated with retinopathy : the hoorn study. Diabetes Care. 2002;25:1320.

92) Moloney JBM, Duruy MI. The effect of pregnancy on the natural course if diabetic retinopathy. Am J Ophthalmol. 1982 ; 93: 745-75.

93) Moss SE, Klein R, Klein BE. The association of alcohol consumption with the incidence and progression of diabetic retinopathy. Ophthalmology. 1994;101:1962- 68.

94) Rudnicka A, Birch J. Diabetic Eye Disease: Identification and Management. (2000). Butterworth Heinemann.

95) Klein R, Klein BE, Moss SE. The epidemiology of ocular problems in diabetes mellitus in SSF : Ocular problems in diabetes mellitus. Boston : Blackwell Publications; 1991.p.1-51.

96) Salz DA, Witkin AJ. Imaging in Diabetic Retinopathy.Middle East Afr J Ophthalmol.2015; 22 : 145-50.

97) Patrick JS, Tyler ME. Ophthalmic Photography: Retinal Photography, Angiography, and Electronic Imaging. 2nd ed. New York: Butterworth-Heinemann Medical; 2001.

98) Treatment techniques and clinical guidelines for photocoagulation of diabetic macular edema. Early Treatment Diabetic Retinopathy Study Report Number 2. Early Treatment Diabetic Retinopathy Study Research Group. Ophthalmology. 1987;94:761–74.

99) Witmer MT, Kiss S. Wide-field imaging of the retina. Surv Ophthalmol. 2013;58:143–54.

100) Tyler ME. Stereo fundus photography: Principles and technique. J Ophthalmic Photogr. 1996;18:68–89

101) Gass JD, Sever RJ, Sparks D, Goren J. A combined technique of fluorescein funduscopy and angiography of the eye. Arch Ophthalmol. 1967;78:455–61

102) Ciardella A, Brown D. Wide field imaging. In: Agarwal A, editor. Fundus Fluorescein and Indocyanine Green Angiography: A Textbook and Atlas. New York: Slack Inc; 2007.p. 79–83

103) Wessel MM, Nair N, Aaker GD, Ehrlich JR, D'Amico DJ, Kiss S. Peripheral retinal ischaemia, as evaluated by ultra-wide field fluorescein angiography, is associated with diabetic macular oedema. Br J Ophthalmol. 2012;96:694–8.

104) Friberg TR, Gupta A, Yu J, Huang L, Suner I, Puliafito CA, et al. Ultra wide angle fluorescein angiographic imaging: A comparison to conventional digital acquisition systems. Ophthalmic Surg Lasers Imaging. 2008;39:304–10.

105) Oliver SC, Schwartz SD. Ultra-wide field fluorescein angiography, Retinal Angiography and Optical Coherence Tomography. New York: Springer Science and Business Media; 2009.p . 407–17.

106) Muqit MM, Marcellino GR, Henson DB, Young LB, Patton N, Charles SJ, et al. Optos-guided pattern scan laser (Pascal)-targeted retinal photocoagulation in proliferative diabetic retinopathy. Acta Ophthalmol. 2013;91:251–8.

107) Coleman DJ,Silverman RH, Lizzi FL, Rondeau MJ. Ultrasonography of the Eye and Orbit. 2nd ed. Philadelphia: Lippincott Williams and Wilkins; 2006. p64-68.

108) McLeod D, Restori M. Ultrasonic examination in severe diabetic eye disease. Br J Ophthalmol. 1979;63:533–8.

109) Sikorski BL, Malukiewicz G, Stafiej J, Lesiewska-Junk H, Raczynska D. The diagnostic function of OCT in diabetic maculopathy.Mediators Inflamm. 2013;2013:434560.

110) De Barros Garcia JMB, Isaac DLC, Avila M. Diabetic retinopathy and OCT angiography: clinical findings and future perspectives. Int J Retina and Vitreous. 2017;3:14.

111) Klein R, Klein BE, Moss SE, Cruickshanks KJOphthalmology. The Wisconsin Epidemiologic Study of Diabetic Retinopathy. XV. The long-term incidence of macular edema .1995;102:7-16.

112) Diabetes Control and Complications Trial Research group. The effect of intensive treatment of diabetes on the development and progression of long-term complications insulin dependent diabetes mellitus.N Eng J Med. 1993;329:977-86

113) UK Prospective Diabetes Research Study Group. Intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment and risk complications in patients with Type 2 diabetes (UKPDS 33). Lancet. 1998;352:837-53

114) UK Prospective Diabetes Research Study Group. Intensive blood glucose control with metformin on complications in overweight patients with Type 2 diabetes (UKPDS 34). Lancet. 1998;352:854-65

115) Klein R, Klein BE, Moss SE, Palta M. A cohort study of the relationship of diabetic retinopathy to blood pressure. Arch Ophthalmol. 1995;113:601-6

116) UK Prospective Diabetes Study Group. Tight blood pressure and risk of macrovascular and microvascular complications in type 2 diabetes (UKPDS 38). Br Med J. 1998;317:703-13

117) Diabetic Retinopathy Study Research Group: Indications for photocoagulation treatment of diabetic retinopathy. DRS report No 14. Int Ophthalmol Clin. 1987;27:239–53.

118) Early Treatment Diabetic Retinopathy Study Research Group: Treatment techniques and clinical guidelines for photocoagulation of diabetic macular edema. ETDRS report number 2. Ophthalmology. 1987;94:761–74.

119) Wei ZY, Hu SX, Tang N, Wu J, Wang J.Effect of argon laser photocoagulation on diabetic retinopathy. Ci Yi Jun Yi Da Xue Xue Bao. 2004;24:1313–15.

120) Qian Z, Zhu L, Zhao C. Observation on clinical effects of panretinal coagulation for diabetic retinopathy. Yan Ke Xue Bao. 2002;18:99–101.

121) Rema M, Sujatha P, Pradeepa R.Visual outcomes of pan-retinal photocoagulation in diabetic retinopathy at one-year follow-up and associated risk factors. Indian J Ophthalmol. 2005;53:93–99.

122) Ferris F. Early photocoagulation in patients with either type I or type II diabetes. Trans Am Ophthalmol Soc. 1996;94:505–37.

123) Lang GE. Diabetic Retinopathy: Laser Treatment of Diabetic Retinopathy. Dev Ophthalmol.2007; 39:48–68.

124) Zaninetti M, Petropoulos IK, Pournaras CJ. Proliferative diabetic retinopathy: vitreo-retinal complications are often related to insufficient retinal photocoagulation. J Fr Ophtalmol. 2005;28: 381–84.

125) Brenick GH. Diabetic macular edema, a review. Ophthalmology. 1986;93:989–97.

126) Lang GE . Diabetic Retinopathy: Benefits and limitations in Vitreoretinal surgery for Proliferative Diabetic Retinopathy and Macular Edema. Dev Ophthalmol. 2007; 39: 69-87.

127) Helbig H, Kellner U, Bornfeld N, Foerster MH. Vitrektomie bei diabetischer Retinopathie: Ergebnisse,Risikofaktoren, Komplikationen. Klin Monatsbl Augenheilkd. 1998;212:339–42.

128) Helbig H, Kellner U, Bornfeld N, Foerster MH.Rubeosis iridis after vitrectomy for diabetic retinopathy. Graefes Arch Clin Exp Ophthalmol. 1998;236:730–33.

129) Machemer R, Sugita G, Tano Y.Treatment of intraocular proliferations with intravitreal steroids. Trans Am Ophthalmol Soc. 1979;77:171–80.

(130) Tano Y, Sugita G, Abrams G, Machemer R. Inhibition of intraocular proliferation with intravitreal corticosteroid. Am J Ophthalmol. 1980;89:131–36.

131) McCuen BW 2nd, Bessler M, Tano Y, Chandler D, Machemer R.The lack of toxicity of intravitreally administered triamcinolone acetonide. Am J Ophthalmol. 1981;91:785–88.

(132) Sutter FK, Simpson JM, Gillies MC. Intravitreal triamcinolone for diabetic macular edema that persists after laser treatment: three-month efficacy and safety results of a prospective, randomized, double-masked, placebo-controlled clinical trial. Ophthalmology. 2004;111:2044–49.

133) Anant P, Yog RS, Vinith S, Anusha V. Current management of diabetic retinopathy. Ophthalmology Today.2010;11:42-51.

134) Grading diabetic retinopathy for stereoscopic colour fundus photographs an extension of the modified airline house classification, ETDRS report number 10. Early treatment diabetic retinopthy study research group. Ophthalmology. 1991;98:786-806.

(135) Doherty MD, Dooley I, Dwyer MH. Interventions for diabetic macular edema : A systemic review of the literature. Br J Ophthalmol .2008;92:1581-90

(136) Abdallah W, Fawzi AA. Anti-VEGF therapy in proliferative diabetic retinopathy. IntOphthalmolClin. 2009;49:95-107.

(137) Arevalo JF, Fromow-Guerra J, Quiroz-Mercado H, Sanchez JG, Wu L, Maia M, et al.Primary intravitreal bevacizumab (Avastin) for diabetic macular edema: results from the Pan-American Collaborative Retina Study Group at 6-month follow-up. Ophthalmology. 2007 ;114:743-50.

138) Haritogloc C, Kook D, Neubaver A, Wolf A, Priglinger S, Strauss R, et al. Intravitreal bevacizumab (Avastin) therapy for persistent diffuse diabetic macular edema. Retina. 2006;26:999-1005.

(139) Chum DW, Heier JS, Topping TM, Duker JS, Bankert JM. Pilot study of multiple intravitreal injections of ranibizumab in patients with centre involving clinically significant diabetic macular edema. Ophthalmology.2006;113:1706-12.

(140) Cunningham ET Jr, Adamis AP, Altaweel M, Aiello LP, Bressler NM, D'Amico DJ, et al. A phase II randomized double masked trial of pegaptanib, an anti-vascular endothelial growth factor aptamer, for diabetic macular edema. Ophthalmology. 2005;112:1747-57.

(141) Kang SW, Sa HS, Cho HY, Kim J I, et al. Macular grid photocoagulation after intravitreal triamcinolone acetonide for diffuse diabetic macular edema. Arch Ophthalmol.2006;124:653-8.

(142) Lam DS, Chan CK, Mohamed S,Lai TY, Lee VY, Liu DT, et al. Intravitreal triamcinolone plus sequential grid laser versus triamcinolone or laser alone for treating diabetic macular edema : six month outcomes. Ophthalmology.2007; 114 :2162-7.

143) PKC – DMES study group. Effect of Ruboxistaurin in patients with diabetic macular edema; Thirty month results of the randomized PKC-DMES clinical trial. Arch Ophthalmol 2007;125:318-24.

144) Nayak HK,Vyas S,Solanki A,Tiwari H.Prevalence of type 2 diabetes in urban population of Ahmedabad,Gujarat.Indian Journal of medical specialities. 2011; 2: 101-105.

145) Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das AK, 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.

146) Namperumalsamy P, Kim R, Vignesh TP, Nithya N, Royes J, Gijo T , et al.Prevalence and risk factors for diabetic retinopathy: a population-based assessment from Theni District, south India. Postgrad Med J. 2009 ;85:643-8.

(147) Harris MI, Klein R, Welborn TA, Knuiman MW. Onset of NIDDM occurs at least 4-7 years before clinical diagnosis. Diabetes Care. 1992; 15 : 815-9.

(148) Dandona L, Dandona R, Naduvilath TJ, McCarty CA, Rao GN. Population based assessment of diabetic retinopathy in an urban population in southern India. Br J Ophthal .1999; 83 : 937-40.

149) Shimura M, Yasuda K, Nakazawa T, Tamai M. Visual dysfunction after panretinal photocoagulation in patients with severe diabetic retinopathy and good vision. Am J Ophthalmol. 2005;140:8-15.

(150) Ramachandran A, Snehalata C, Satyavani K, Latha E, Sasikala R, Vijay V. Prevalence of vascular complications and their risk factors in type 2 diabetes. Jour Assoc Phy India. 1999; 47: 1152-62.

(151) Raman R, Rani PK, Reddi Rachepalle S, Gnanamoorthy P, Uthra S, Kumaramanickavel G, et al. Prevalence of diabetic retinopathy in India: Sankara Nethralaya diabetic retinopathy epidemiology and molecular genetics study report 2. Ophthalmology. 2009;116:311‑18.

152) Narendran V, John RK, Raghuram A, Ravindran RD, Nirmalan PK,Thulasiraj RD. Diabetic retinopathy among self reported diabetics in Southern India: A population based assessment. Br J Ophthalmol. 2002;86:101-8.

153) Knuiman MW, Welborn TA, McCann VJ, Stanton KG, Constable IJ. Prevalence of diabetic complications in relation to risk factors. Diabetes. 1986; 35: 1332-9.

154) Caird FI, Pirie A, Ramsell TG. The natural history of diabetic retinopathy : Diabetes and the eye. Oxford , Blackwell Publications; 1968. p. 72- 100.

155) Rema M, Pradeepa R. Diabetic retinopathy: an Indian perspective.Indian J Med Res. 2007 ;125:297-310.

(156) The Diabetes Control and Complications Trial Research Group. Clustering of long-term complications in families with diabetes in the diabetes control and complications trial. Diabetes. 1997;46:1829–39.

157) Gadkari SS, Maskati QB, Nayak BK. Prevalence of diabetic retinopathy in India: The All India Ophthalmological Society Diabetic Retinopathy Eye Screening Study 2014. Indian J Ophthalmol. 2016;64:38-44.

158) Rajiv K, Saleh A, Harith A, Jawad A. Knowledge, attitude and practice regarding eye complications and care among Omani persons with diabetes - A cross sectional study. Oman J Ophthalmol. 2010 ; 3:641.

159) Srinivasan NK, John D, Rebekah G, Kujur ES, Paul P, John SS. Diabetes and Diabetic Retinopathy: Knowledge, Attitude, Practice among Diabetic Patients in A Tertiary Eye Care Centre. J Clin Diagn Res. 2017;11:01-07.

160) Hussain R, Rajesh B, Giridhar A, Gopalakrishnan M, Sadasivan S, James J, et al. Knowledge and awareness about diabetes mellitus and diabetic retinopathy in suburban population of a South Indian state and its practice among the patients with diabetes mellitus: A population-based study. Indian J Ophthalmol. 2016;64:272-76

161) Babu N, Kim D, Ramchandani B, Sachin T. Diabetes and Diabetic Retinopathy: Knowledge,Attitude, Practice (KAP) Among Paramedical Personnel (PMPS) and Community Members (CMS) In Southern India. AIOC 2009 proceedings.

162) Kadri R. Awareness of Diabetic and Hypertensive eye disease in Public. International Journal of Biological and Medical Research. 2011; 2: 533-5.

163) Mahesh G, Elias A, Sandhya N, Giridhar A, Saikumar SJ, Sankaranarayanan, et al.Chengamanad Diabetic Retinopathy Awareness Study (CDRAS). Kerala J Ophthalmol. 2006;28:14-21.

164) Das T, Wallang B, Semwal P, Basu S, Padhi TR, Ali MH. Changing clinical presentation, current knowledge-attitude-practice, and current vision related quality of life in self-reported type 2 diabetes patients with retinopathy in eastern India: The LVPEI eye and diabetes study. J Ophthalmol. 2016;2016:3423814.

165) Wee HL, Ho HK, Li SC. Public awareness of diabetes mellitus in Singapore. Singapore Med J. 2002; 43: 128-34.

166) Mohammed I, Waziri A. Awareness of diabetic retinopathy amongst diabetic patients at the Murtala Mohammed Hospital, Kano, Nigeria. Nigeria Med J. 2009;50:38-41.

167) Tajunisah L, Wong P, Tan L, Rokiah P, Reddy S. Awareness of eye complications and prevalence of retinopathy in the first visit to eye clinic among type 2 diabetic patients Int J Ophthalmol. 2011; 4: 519–24.

ANNEXURES

ANNEXURE - 1

PROFORMA

1) Demographic Details:

- Name
- Age
- Sex
- Address
- Education
- Occupation

2) History:

- Type of DM
- Duration of disease
- Family history of Diabetes / DR
- Family history of loss of vision following DR
- How is the control of sugar levels since onset
- How is the compliance to treatment

3) Treatment

- OHA
- Insulin
- Duration

4) Physical Activity – Sedentary / Moderate / Heavy

5) Last dilated eye examination done?

QUESTIONNAIRE

1.1 Are you aware that diabetes affects the eye? YES/NO

1.2 Did you know that diabetes can damage eyesight leading to blindness?

YES/NO

1.3 Have you heard that it affects the nerve layer of the eye (diabetic retinopathy)?

YES/ NO

1.4 Regular checkup and treatment can prevent / delay damage due to diabetic retinopathy in eye.

YES/ NO /DON'T KNOW

1.5 Control of blood sugar and lipids makes eye / diabetic retinopathy treatment effective. YES/ NO /DON'T KNOW

1.6 If vision is damaged due to diabetic retinopathy, use of ' Low Vision Aids'(Spectacles) helps in daily work.

YES/ NO / DON'T KNOW

1.7 In diabetic retinopathy, one eye may be affected first followed by the other eye.

YES/ NO / DON'T KNOW

1.8 Knowledge of modalities of treatment for diabetic retinopathy.

YES/ NO / DON'T KNOW

1.9 An eye with diabetic retinopathy, if successfully treated with laser, doesn't need laser treatment again.

YES/ NO / DON'T KNOW

1.10 Laser treatment of diabetic retinopathy is painful.

YES/ NO / DON'T KNOW

1.11 Frequency of follow up visits needed.

YES/ NO / DON'T KNOW

2.1 If my vision is good my eyes are not affected in Diabetes, so I do not need annual eye testing.

AGREE / DISAGREE / PARTIALLY AGREE

2.2 The information on eye problems due to diabetic retinopathy should be given by eye doctor only.

AGREE / DISAGREE / PARTIALLY AGREE

2.3 If im taking eye treatment, I need not worry about controlling my sugar and lipids.

AGREE / DISAGREE / PARTIALLY AGREE

2.4 If my eye is treated with laser once, I don't need laser treatment again in the same eye.

AGREE / DISAGREE / PARTIALLY AGREE

2.5 Patients with diabetes often waste their time and money in eye checkup as most of the time eyes of diabetics are normal.

AGREE / DISAGREE / PARTIALLY AGREE

2.6 One should not be treated with laser as its painful.

AGREE / DISAGREE / PARTIALLY AGREE

3.1 I go to the eye doctor regularly as advised by my family doctor, even if my vision is good.

AGREE / DISAGREE / PARTIALLY AGREE

3.2 I control my blood sugar and lipid even if diabetic retinopathy is being treated.

AGREE / DISAGREE / PARTIALLY AGREE

3.3 Staff in Eye unit counseled me about prevention and treatment for diabetic retinopathy and its complications.

AGREE / DISAGREE / PARTIALLY AGREE

3.4 My vision due to diabetic retinopathy is less. Hence I'm using low vision devices.

AGREE / DISAGREE / PARTIALLY AGREE

OPHTHALMIC EXAMINATION :

1. Visual Acuity
2. Anterior Segment
3. IOP(Goldmans Applanation)
4. Dilated fundus Examination

	NON PROLIFERATIVE DR (NPDR)
NO DR	
Very Mild	
Mild	
Moderate	
Severe	
Very Severe	
	PROLIFERATIVE DR
Mild – Moderate	
High risk	
	ADVANCED DIABETIC EYE DISEASE
	MACULAR EYE DISEASE

5. Fundus Photography
6. B Scan

ಪ್ರಶ್ನಾವಳಿಗಳು

1.1ಮಧುಮೇಹದಿಂದ ದೃಷ್ಟಿಗೆ ತೊಂದರೆ ಆಗುತ್ತದೆಯೇ ? ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.2 ಅಕ್ಷಿಯ ಪಟಲದ ತೊಂದರೆ ಆಗುವ ಬಗ್ಗೆ ಮಾಹಿತಿ ಇದೆಯೇ? ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.3 ನೀವು ಮಧುಮೇಹ ರೆಟಿನೋಪತಿ ಬಗ್ಗೆ ಕೇಳಿದ್ದೀರ ? ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.4 ಮಧುಮೇಹ ರೆಟಿನೋಪತಿ ಕಾಯಿಲೆಯು ದೃಷ್ಟಿಯ ಮೇಲೆ ಹಾನಿಯುಂಟುಮಾಡಬಹುದು?

ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.5 ಕಣ್ಣಿನ ವೈದ್ಯರು ಕಣ್ಣಿನಲ್ಲಿ ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಪರಿಣಾಮಗಳನ್ನು ವಿಶೇಷ ಉಪಕರಣಗಳನ್ನು

ಬಳಸಿ ಕಣ್ಣುಗಳು ಪರೀಕ್ಷೆಗೆ ಒಳ ಪಡಿಸುತ್ತಾರೆ ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.6 ಸಕಾಲಿಕ ಚಿಕಿತ್ಸೆ ನೀಡುವುದರಿಂದ ಕಣ್ಣಿನಲ್ಲಿ ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಯಿಂದ ಆಗುವ ಹಾನಿ

ತಡೆಯಬಹುದು. ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.7 ರಕ್ತದಲ್ಲಿನ ಸಕ್ಕರೆ ಪ್ರಮಾಣವನ್ನು ನಿಯಂತ್ರಣದಲ್ಲಿ ಇಟ್ಟು ಕೊಳ್ಳುವದರಿಂದ / ಮಧುಮೇಹದ

ರೆಟಿನೋಪತಿ ಚಿಕಿತ್ಸೆ ಪರಿಣಾಮಕಾರಿಯಾಗಿ ಕೆಲಸ ಮಾಡುತ್ತದೆ.

ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.8 ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಒಂದು ಕಣ್ಣಿನ ಪರಿಣಾಮ ಇನ್ನೊಂದು ಮೊದಲ ಕಣ್ಣಿನ ಮೇಲು

ಪರಿಣಾಮ ಬೀರ ಬಹುದು .

ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.9 ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಒಂದು ಸಾರಿ ಯಶಸ್ವಿಯಾಗಿ ಲೇಸರ್ ಚಿಕಿತ್ಸೆ ಯನ್ನು ನೀಡಿದ್ದರೆ ,ಮತ್ತೆ

ಲೇಸರ್ ಚಿಕಿತ್ಸೆಯ ಅಗತ್ಯವಿಲ್ಲ. ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.10 ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಕಾರಣ ದೃಷ್ಟಿ ಮಂದ ಆಗಿದ್ದರೆ ಲೇಸರ್ ಚಿಕಿತ್ಸೆ ತುಂಬಾ

ನೋವಿನಿಂದ ಕೂಡಿರುತ್ತದೆ . ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.11, ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಚಿಕಿತ್ಸೆಯನ್ನು ಕನ್ನಡಕ ದಂತಹ ಸಾದನಗಳ ಬಳಕೆ ಮೂಲಕ

ದೈನಂದಿನ ಕೆಲಸದಲ್ಲಿ ಸಹಾಯ ವಾಗಬಲ್ಲದು . ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.12 ಮಧುಮೇಹ ರೆಟಿನೋಪತಿ ಕಾಯಿಲೆಯ ಬಗ್ಗೆ ನಿಮಗೆ ತಿಳುವಳಿಕೆ ಇದೆಯೇ?.

ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

1.13 ಪದೇ ಪದೇ ಬೇಟಿಯ ಅಗತ್ಯವಿದೆ. ಹೌದು / ಇಲ್ಲ / ಗೊತ್ತಿಲ್ಲ

ಧೋರಣೆ:

2.1 ನನ್ನ ದೃಷ್ಟಿ ಚೆನ್ನಾಗಿದ್ದು ಕಣ್ಣು ಗಳಿಗೆ ಮಧುಮೆಹದಿಂದ ಯಾವುದೇ ಪರಿಣಾಮವಿಲ್ಲಾ , ಆದ್ದರಿಂದ

ನನಗೆ ವಾರ್ಷಿಕ ಕಣ್ಣಿನ ಪರೀಕ್ಷೆ ಅಗತ್ಯವಿಲ್ಲ.

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಬಹುಷಃ ಒಪ್ಪಿಕೊಳ್ಳಬಹುದು

2.2 ಮಾಹಿತಿ ಕಣ್ಣಿನ ವೈದ್ಯರು ಮಾತ್ರ ಮೂಲಕ ನೀಡಬೇಕು.

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಬಹುಷಃ ಒಪ್ಪಿಕೊಳ್ಳಬಹುದು

2.3 ನನ್ನ ಕಣ್ಣಿನ ಚಿಕಿತ್ಸೆಯನ್ನು ಮಾಡಿಸಿಕೊಳ್ಳುವ ವೇಳೆ, ನನ್ನ ಮಧುಮೇಹದ ಮತ್ತು ಲಿಪಿಡ್ಗಳ

ನಿಯಂತ್ರಿಸುವ ಬಗ್ಗೆ ಚಿಂತೆ ಇಲ್ಲ.

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

2.4, ನಾನು ಮಧುಮೇಹದಿಂದ ಬಳತ್ತಿದ್ದು ನನ್ನ ಕಣ್ಣು ಗಳ ಮೇಲೆ ಒಮ್ಮೆ ಲೇಸರ್ ಚಿಕಿತ್ಸೆ ಆಗಿದೆ,ಅದೇ

ಕಣ್ಣಿಗೆ ಮತ್ತೆ ಲೇಸರ್ ಚಿಕಿತ್ಸೆ ಅಗತ್ಯವಿಲ್ಲ.

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

2.5 ಸಾಮಾನ್ಯವಾಗಿ ಮಧುಮೇಹಿ ರೋಗಿಗಳಿಗೆ ಸಮಯ ಮತ್ತು ಹಣ ವ್ಯರ್ಥ.

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

2.6 ಲೇಸರ್ ಆಪರೇಷನ್ ನೋವಿನಿಂದ ಕೂಡಿರುತ್ತದೆ.

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

ಆಚರಣೆಗಳು:

3.1 ನನ್ನ ದೃಷ್ಟಿ ಚನ್ನಾಗಿ ಇದ್ದಾಗಲೂ ನನ್ನ ಕುಟುಂಬ ವೈದ್ಯರು ಸೂಚಿಸಿದ ಹಾಗೆ ನಾನು ನಿಯಮಿತವಾಗಿ

ಕಣ್ಣಿನ ಚಿಕಿತ್ಸೆ ಮಾಡಿಸಿಕೊಳ್ಳುತ್ತೇನೆ ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

3.2 ನನಗೆ ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಕಣ್ಣಿನ ಚಿಕಿತ್ಸೆ ಮಾಡಿಸಿ ಕೊಳ್ಳುತ್ತಿದ್ದರು.ನನ್ನ ರಕ್ತದ ಸಕ್ಕರೆ

ಮತ್ತು ಲಿಪಿಡ್ ಪ್ರಮಾಣವನ್ನು ನಿಯಂತ್ರಣದಲ್ಲಿಟ್ಟು ಕೊಳ್ಳಬೇಕಾಗುತ್ತದೆ

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

3.3 ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಸಿಬ್ಬಂದಿ ನನಗೆ ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ ಚಿಕಿತ್ಸೆಯ ಬಗ್ಗೆ ನನಗೆ ಸಂಪೂರ್ಣ ಮಾಹಿತಿ ನೀಡಿದ್ದಾರೆ . ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

3.4 ನನ್ನ ದೃಷ್ಟಿ ಕಡಿಮೆ ಆಗಲು ಕಾರಣ ಮಧುಮೇಹದ ರೆಟಿನೋಪತಿ. ಆದ್ದರಿಂದ ಕನ್ನಡಕ ದಂತಹ ಸಾಧನಗಳನ್ನು ಬಳಸಿಕೊಂಡು ನೋಡ ಬಹುದು .

ಭಾಗಶಃ ಒಪ್ಪುತ್ತೇನೆ / ಅಸಮ್ಮತಿ / ಒಪ್ಪಿಕೊಳ್ಳದಿದ್ದರೆ

ANNEXURE - II

INFORMED CONSENT FORM

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of my personal information as outlined in this consent form.

I understand the purpose of this study, the risks and benefits of the procedure and the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

I have had the opportunity to ask questions regarding the various aspects of this study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw from this study at any time and this will not change my future care.

Participation in this study does not involve any extra cost to me.

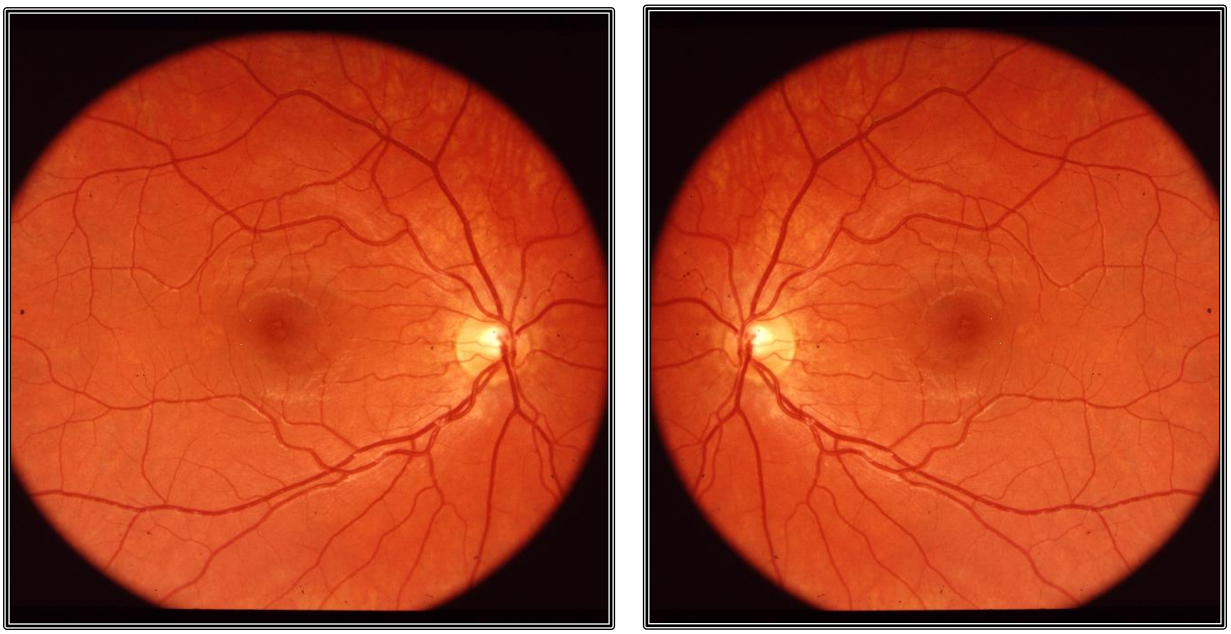
Subject's name and signature /thumb impression Date:

Name and signature of witness Date:

Name and signature of person obtaining consent Date:

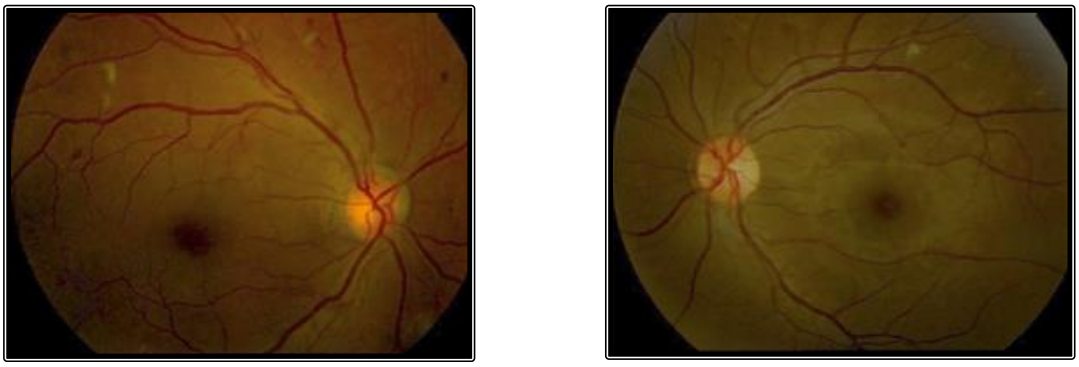
ANNEXURE – III

PHOTOGRAPHS



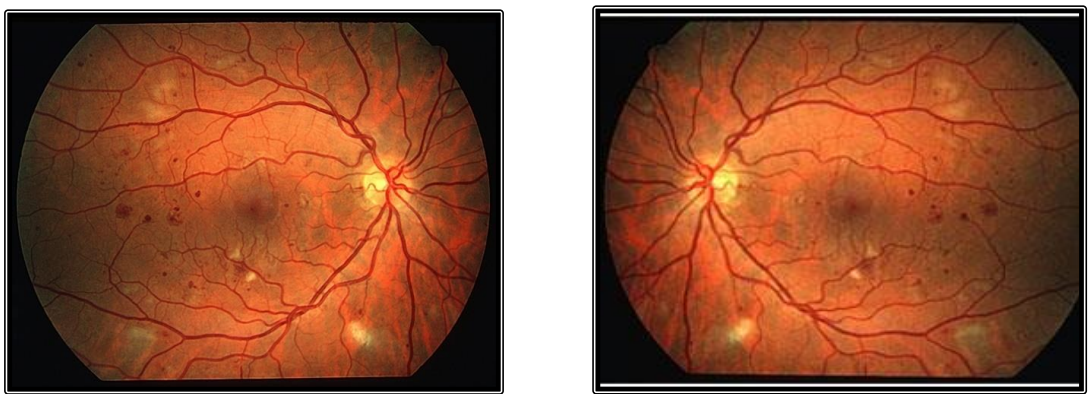
(a) (b)

Figure 19 (a & b): Normal Fundus picture



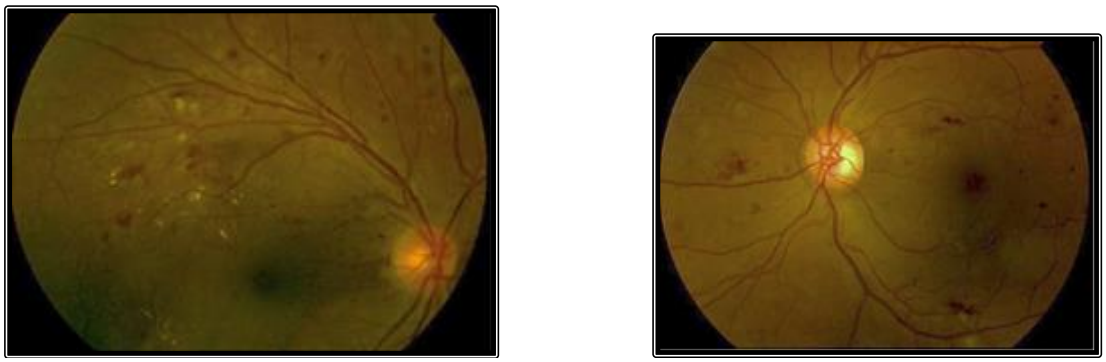
(a) (b)

Figure 20 (a&b): Fundus Photos showing Mild NPDR in a patient.



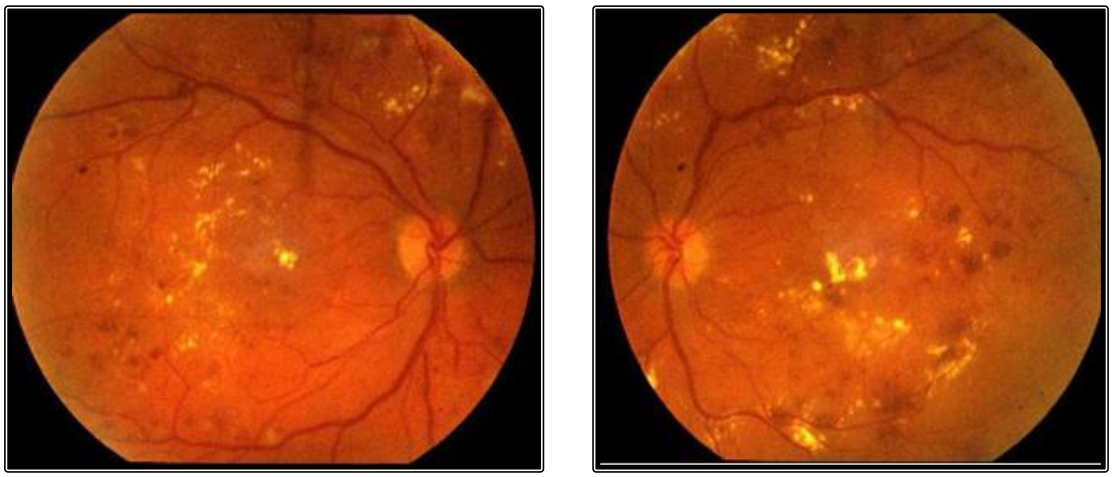
(a) (b)

Figure 21 (a & b): Fundus picture showing Moderate NPDR



(a) (b)

Figure 22 (a & b): Fundus Pictures showing severe NPDR (RE) and Moderate NPDR (LE). Venous beeding is seen along with superficial and dot-blot hemorrhages.

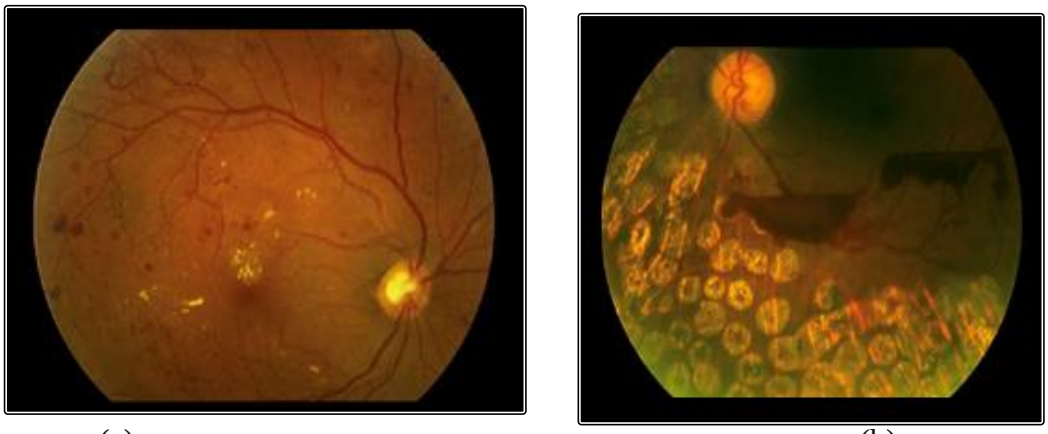


(a) (b)

Figure 23 (a& b): Diabetic retinopathy with severe NPDR with multiple microaneurysms, dot and blot hemorrhages, flame shaped hemorrhages and hard exudates with CSME.

(a) (b)

Figure 24 (a&b) : Proliferative Diabetic Retinopathy (both eye) with sub hyaloid hemorrhages in the inferior temporal quadrant in Right eye



(a) (b)

Figure 25 (a & b): Fundus examination showed Proliferative diabetic retinopathy (a) NVE in the superior temporal quadrant in RE and (b) laser spots with sub hyaloid hemorrhage and NVD in LE (High Risk PDR).

Figure 26 (a,b & c) : Diabetic Retinopathy Camp

(a) Camp Place

(b) Torch Light Examination

(c) Examination with a Direct Ophthalmoscope

ANNEXURE – IV

KEY TO MASTER CHART

- A: AGREE
- ARMD: AGE RELATED MACULAR DEGENERATION
- AS: ANTERIOR SEGMENT
- BCVA: BEST CORECTED VISUAL AQUITY (DISTANT)
- CDR: CUP DISC RATIO
- CF: COUNTING FINGERS
- CSME: CLINICALLY SIGNIFICANT MACULAR OEDEMA
- DA: DISAGREE
- DK : DON'T KNOW
- F: FEMALE
- H: HEAVY
- HM: HAND MOVEMENTS
- M: MALE
- m: METER
- Mo: MODERATE
- N: NO
- NPDR: NON PROLIFERATIVE DIABETIC RETINOPATHY
- OD: OCULUS DEXTER
- OHA : ORAL HYPOGLYCEMIC AGENTS
- OS: OCULUS SINISTER
- PA: PARTIALLY AGREE
- PCO: POSTERIOR CAPSULAR OPACIFICATION

- PDR: PROLIFERATIVE DIABETIC RETINOPATHY
- PEX: PSEUDOEXFOLIATION
- PSP: PSEUDOPHAKIA
- PUC: PRE – UNIVERSITY COURSE
- RAPD: RELATIVE AFFERENT PUPILLARY DEFECT
- S: SEDENTARY
- SIMC: SENILE IMMATURE CATARACT
- SL. NO.: SERIAL NUMBER
- SMC: SENILE MATURE CATARACT
- STD : STANDARD
- WNL : WITHIN NORMAL LIMITS
- Y: YES

SL. NO.	NAME	AGE	SEX	EDUCATION	OCCUPATION	TYPE OF DM	AMILY HISTOR	DURATION	CONTROL	TREATMENT	DURATION OF Rx	IYSICAL ACTIVI	ST FUNDUS EXAMINATI
1	Venkatamma	60 YEARS	F	7STD	FARMER	2	N	10YEARS	Y	OHA	10YEARS	H	N
2	Abdul Faeez	35 YEARS	M	GRADUATE	ENGINEER	2	Y	1MONTHS	Y	OHA	1MONTHS	S	N
3	Govindu	63 YEARS	M	PUC	BUSINESS	2	N	10YEARS	N	OHA	10YEARS	S	N
4	Ramappa	70 YEARS	M	10STD	FARMER	2	N	12YEARS	N	I	8YEARS	H	N
5	Subraya	80 YEARS	M	2STD	FARMER	2	N	7YEARS	Y	OHA	7YEARS	H	N
6	Gowardhan	29 YEARS	M	GRADUATE	LECTURER	2	Y	6MONTHS	Y	OHA	6MONTHS	Mo	N
7	Noor Mohammed	50 YEARS	M	NIL	FARMER	2	N	1YEARS	Y	OHA	1YEARS	H	N
8	Kedar Khan	30 YEARS	M	GRADUATE	TEACHER	2	Y	1YEARS	Y	OHA	1YEARS	Mo	Y
9	Kinnapa	50 YEARS	M	GRADUATE	BUSINESS	1	Y	15YEARS	Y	I	15YEARS	Mo	Y
10	Naryavshi	68 YEARS	M	6STD	BUSINESS	2	N	3MONTHS	Y	OHA	3MONTHS	Mo	N
11	Kamal Bhai	85 YEARS	F	NIL	UNOCCUPIED	2	N	8YEARS	N	OHA	8YEARS	S	N
12	Eshwara Rao	51 YEARS	M	GRADUATE	CLERK	2	Y	5YEARS	Y	OHA	5YEARS	Mo	Y
13	Anaulla	40 YEARS	M	PUC	AUTO DRIVER	1	Y	9YEARS	N	I	8YEARS	Mo	Y
14	Renuakka	60 YEARS	F	HOUSE WIFE	HOUSEWIFE	2	N	4YEARS	Y	OHA	4YEARS	Mo	N
15	Ramana	60 YEARS	F	NIL	FARMER	2	N	2YEARS	N	NO	2YEARS	H	N
16	Shivakasi	60 YEARS	M	NIL	COOLIE	2	Y	3MONTHS	Y	NO	3MONTHS	H	N
17	Srinivasa	30YEARS	M	GRADUATE	BUSINESS	2	N	1YEARS	Y	OHA	1YEARS	S	Y
18	Anjaneya	50 YEARS	M	10STD	DRIVER	1	N	6YEARS	Y	OHA	5YEARS	Mo	N
19	Shiva	26YEARS	M	GRADUATE	BUSINESS	1	Y	2YEARS	Y	OHA	2YEARS	S	Y
20	Karthi	40 YEARS	F	GRADUATE	LECTURER	2	N	6MONTHS	Y	OHA	3MONTHS	Mo	N
21	Srinivas	57 YEARS	M	NIL	FARMER	2	N	7YEARS	Y	OHA	7YEARS	Mo	N
22	Nagendra Kumar	60 YEARS	M	10STD	WAITER	2	Y	9YEARS	N	I	9YEARS	Mo	N
23	Ramachandrappa	82 YEARS	M	NIL	UNOCCUPIED	2	N	20YEARS	N	OHA	20YEARS	S	Y
24	Muniyappa	53 YEARS	M	PUC	DRIVER	2	Y	2YEARS	Y	OHA	2YEARS	S	Y
25	Adilukahmi	40 YEARS	F	GRADUATE	NURSE	2	N	3YEARS	Y	OHA	3YEARS	Mo	Y
26	Abdul Sattar	75 YEARS	M	10STD	FARMER	2	N	12YEARS	N	OHA	10YEARS	Mo	N
27	Shabana	31 YEARS	F	BSC	OPERATOR	1	Y	5YEARS	Y	I	5YEARS	Mo	Y
28	Shalsila	31 YEARS	F	PUC	HOUSEWIFE	2	N	2YEARS	Y	OHA	2YEARS	Mo	N
29	Safarabhi	62 YEARS	F	2STD	FARMER	2	Y	10YEARS	Y	OHA	10YEARS	H	Y
30	Babijaan	50 YEARS	F	7STD	HOUSE MAID	2	Y	5YEARS	N	OHA	5YEARS	H	Y
31	Nagray Naik	44 YEARS	M	GRADUATE	BUSINESS	2	N	2MONTHS	Y	OHA	2MONTHS	S	N
32	Pyari Jaan	71 YEARS	M	NIL	HOUSEWIFE	2	N	28YEARS	N	OHA	20YEARS	S	N
33	N M Narayanswamy	54 YEARS	M	2STD	DRIVER	2	N	5YEARS	Y	OHA	5YEARS	Mo	N
34	Munivenkatamma	48 YEARS	F	NIL	HOUSE MAID	2	N	1MONTHS	N	NIL	NIL	Mo	N
35	Nazeen Munnira	56 YEARS	F	GRADUATE	TEACHER	1	Y	20YEARS	Y	I	20YEARS	S	Y
36	Thameem	70 YEARS	M	NIL	FARMER	2	Y	7YEARS	Y	OHA	5YEARS	Mo	N
37	Lakshmamma	86 YEARS	F	NIL	HOUSE WIFE	2	N	12YEARS	N	NIL	NIL	S	N
38	Sharadha	35 YEARS	F	GRADUATE	HOUSE WIFE	2	Y	7YEARS	Y	OHA	7YEARS	Mo	Y
39	Balubai	45 YEARS	F	PUC	CLERK	2	N	2YEARS	Y	OHA	2YEARS	S	N
40	Narayanswamy	76 YEARS	M	10STD	FARMER	2	N	18YEARS	N	NIL	5YEARS	S	N
41	Venkateshappa	60 YEARS	M	PUC	BUSINESS	1	Y	15YEARS	Y	OHA	15YEARS	Mo	Y
42	Nalamma	62 YEARS	F	3STD	COOK	2	Y	8YEARS	Y	OHA	5YEARS	Mo	N
43	Babeethul	63 YEARS	M	NIL	HOUSE MAID	2	Y	3YEARS	Y	OHA	3YEARS	Mo	Y
44	Muniswamy	71 YEARS	M	9STD	DRIVER	2	N	5YEARS	Y	OHA	5YEARS	Mo	Y
45	Govindappa	61 YEARS	M	NIL	FARMER	1	N	6YEARS	Y	OHA	6YEARS	Mo	Y
46	Narayamma	60 YEARS	F	NIL	HOUSE WIFE	2	N	3MONTHS	Y	OHA	3MONTHS	Mo	N
47	Shamshal	44 YEARS	F	PUC	TAILOR	2	Y	1YEARS	Y	OHA	8MONTHS	S	Y
48	C Narayanappa	50 YEARS	M	5STD	COOLIE	2	N	3MONTHS	Y	NIL	NIL	H	N
49	Munivenkatamma	58 YEARS	F	10STD	HOUSE WIFE	2	Y	2YEARS	Y	OHA	2YEARS	Mo	Y
50	Muninarayanappa	90 YEARS	M	NIL	UNOCCUPIED	2	N	10YEARS	Y	OHA	8YEARS	S	N
51	Ayappa	80 YEARS	M	5STD	FARMER	2	Y	5YEARS	Y	OHA	5YEARS	S	N
52	Lakshmanna	60 YEARS	F	6STD	BUSINESS	2	Y	3YEARS	N	NIL	NIL	S	N
53	P S Babu	52 YEARS	M	PUC	DRIVER	1	Y	5YEARS	Y	I	5YEARS	Mo	Y
54	Suheda	40 YEARS	F	4STD	COOK	2	N	4MONTHS	N	NIL	NIL	Mo	N
55	Chandu	23 YEARS	M	STUDENT	STUDENT	1	Y	1YEARS	Y	I	1YEARS	S	Y
56	R Vittal Rao	67 YEARS	M	10STD	FARMER	2	Y	10YEARS	Y	OHA	10YEARS	H	Y
57	Marappa	50 YEARS	M	GRADUATE	EMPLOYEE	2	N	3YEARS	Y	OHA	3YEARS	S	Y
58	Munimathamma	40 YEARS	F	GRADUATE	RECEPTIONIST	2	N	6MONTHS	Y	OHA	6MONTHS	S	N
59	Ambresh	35 YEARS	M	GRADUATE	EMPLOYEE	2	N	9MONTHS	Y	OHA	9MONTHS	S	Y
60	Ramappa	68 YEARS	M	PUC	BUSINESS	1	Y	13YEARS	Y	I	13YEARS	S	Y
61	Suma	50 YEARS	F	2STD	COOK	2	Y	2YEARS	Y	OHA	2YEARS	Mo	N
62	Muniyamma	70 YEARS	F	NIL	HOUSEWIFE	2	Y	5YEARS	N	OHA	3YEARS	S	N
63	Chilekhakka	68 YEARS	F	NIL	HOUSEWIFE	2	Y	2YEARS	Y	OHA	2YEARS	S	N
64	Timayappa	76 YEARS	M	NIL	FARMER	2	Y	6YEARS	Y	OHA	4YEARS	Mo	N
65	Muniyamma	60 YEARS	F	3STD	FARMER	2	Y	5YEARS	Y	OHA	5YEARS	H	N
66	Chandrashekar	64 YEARS	M	10STD	UNOCCUPIED	2	Y	7YEARS	Y	OHA	4YEARS	Mo	N
67	Bramarambika	65 YEARS	F	NIL	HOUSE MAID	1	Y	18YEARS	Y	I	18YEARS	S	N
68	Prakash	54 YEARS	M	GRADUATE	SECRETARY	2	Y	7YEARS	Y	OHA	7YEARS	Mo	Y
69	Uma	60 YEARS	F	PUC	TAILOR	1	N	15YEARS	N	I	10YEARS	S	Y
70	Lakshmi	62 YEARS	F	10STD	AGARBATHI FACTOR	2	Y	2YEARS	Y	OHA	2YEARS	S	N
71	Krishnappa	42 YEARS	M	PUC	FACTORY	2	Y	7MONTHS	Y	OHA	2MONTHS	S	N
72	Babu	60 YEARS	M	7STD	BUSINESS	1	Y	11YEARS	Y	OHA	10YEARS	S	N
73	M G Vyasara Rao	58 YEARS	M	GRADUATE	BANK	2	Y	3MONTHSON	Y	OHA	3MONTHS	S	N
74	Krishna Kumar	59 YEARS	M	10STD	CLERK	2	Y	4YEARS	Y	OHA	4YEARS	S	Y
75	Nawadeep	32YEARS	M	GRADUATE	POLICE CONSTABLE	1	Y	7YEARS	N	I	7YEARS	Mo	Y
76	Abdullah	24 YEARS	M	STUDENT	STUDENT	1	N	1MONTHS	Y	NIL	1MONTHS	S	Y
77	Parvathamma	50 YEARS	F	10STD	FACTORY	2	N	2YEARS	Y	OHA	2YEARS	S	N
78	Jayamma	60 YEARS	F	5STD	COOK	2	N	6YEARS	Y	OHA	4YEARS	Mo	N
79	Venkatramappa	62 YEARS	M	NIL	FARMER	2	N	10YEARS	Y	OHA	10YEARS	S	N
80	Venkatramappa	62 YEARS	M	5STD	FARMER	2	N	11YEARS	Y	I	7YEARS	Mo	N
81	Jayaramappa	73 YEARS	M	10STD	UNOCCUPIED	2	N	5YEARS	Y	OHA	3YEARS	S	N
82	Sonappa	62 YEARS	M	10STD	TAILOR	2	N	3YEARS	N	NIL	NIL	S	N
83	Punit	35 YEARS	M	7STD	WAITER	2	N	6MONTHS	Y	OHA	6MONTHS	Mo	Y
84	Gulam Rasood	58 YEARS	M	10STD	MECHANIC	2	Y	5YEARS	Y	OHA	5YEARS	Mo	Y
85	Roshan	27 YEARS	M	GRADUATE	BANK	2	Y	5MONTHS	Y	OHA	5MONTHS	S	N
86	Muniyamma	60 YEARS	F	NIL	HOUSE WIFE	1	Y	19YEARS	Y	I	19YEARS	S	N
87	Shamalamma	45 YEARS	F	PUC	TAILOR	2	Y	3YEARS	Y	OHA	3YEARS	S	Y
88	Pranilamma	45 YEARS	F	GRADUATE	RECEPTIONIST	2	N	5YEARS	Y	OHA	5YEARS	S	N
89	M C Rangappa	66 YEARS	M	PUC	BUSINESS	2	N	7YEARS	N	OHA	2YEARS	S	N
90	Nissral Ahmed	48 YEARS	M	GRADUATE	DRIVER	2	N	2YEARS	Y	OHA	2YEARS	S	N
91	Gurura Reddy	72 YEARS	M	NIL	UNOCCUPIED	2	N	8YEARS	Y	OHA	8YEARS	S	N
92	Sadashivanna	66 YEARS	M	4STD	FARMER	1	N	4YEARS	Y	OHA	4YEARS	S	Y
93	Chellamma	60 YEARS	F	3STD	HOUSE MAID	2	Y	7YEARS	Y	I	7YEARS	Mo	Y
94	Gopalappa	65 YEARS	M	10STD	DRIVER	2	Y	7MONTHS	Y	OHA	7MONTHS	S	Y
95	Anand	37 YEARS	M	GRADUATE	OFFICER	2	Y	9MONTHS	Y	OHA	9MONTHS	S	Y
96	Chotibu	50 YEARS	F	GRADUATE	CONSTABLE	2	Y	5YEARS	Y	OHA	5YEARS	Mo	N
97	Lakshmaiah	55 YEARS	M	PUC	WAITER	2	N	6YEARS	N	OHA	6YEARS	Mo	N
98	Muniyappa	62 YEARS	M	NIL	BUSINESS	2	Y	7YEARS	N	OHA	2YEARS	S	Y
99	Narayanswamy	68 YEARS	M	NIL	FARMER	1	Y	11YEARS	N	I	7YEARS	H	Y
100	Devraj	32 YEARS	M	PUC	DRIVER	2	N	3MONTHS	Y	NIL	NIL	Mo	N
101	Shamanna	80 YEARS	M	NIL	UNOCCUPIED	2	N	9YEARS	N	OHA	7YEARS	S	N
102	Venkatramappa	70 YEARS	M	3STD	FARMER	2	N	4YEARS	Y	OHA	5YEARS	Mo	N
103	Muniyamma	65 YEARS	F	9STD	BUSINESS	1	Y	7YEARS	Y	OHA	7YEARS	S	Y
104	Ajjappa	64 YEARS	M	2STD	FARMER	2	N	3YEARS	Y	OHA	3YEARS	Mo	Y
105	Rathna	36 YEARS	F	PUC	FACTORY WORKER	2	N	6YEARS	Y	OHA	6YEARS	N	Y
106	Malligamma	45 YEARS	F	5STD	TAILOR	2	N	3MONTHS	Y	OHA	3MONTHS	S	N
107	Gayathri	30 YEARS	F	DEGREE	CLERK	2	N	2YEARS	Y	OHA	2YEARS	S	N
108	Amith	65 YEARS	M	NIL	MECHANIC	2	Y	5YEARS	N	OHA	3YEARS	S	Y
109	Parvathamma	45 YEARS	F	5STD	HOUSE MAID	1	Y	12YEARS	N	I	12YEARS	S	Y
110	Nagarathamma	80 YEARS	F	NIL	UNOCCUPIED	2	N	8YEARS	Y	OHA	8YEARS	S	N
111	Sushellamma	63 YEARS	F	3STD	HOUSE WIFE	2	N	1YEARS	Y	OHA	1YEARS	S	Y
112	Narayanappa	70 YEARS	M	7STD	FARMER	2	Y	6YEARS	N	OHA	3YEARS	S	Y
113	Vangoba Rao	68 YEARS	M	6STD	WATCHMAN	2	Y	7YEARS	N	OHA	7YEARS	S	N
114	Krishnappa	67 YEARS	M	9STD	WAITER	2	N	5YEARS	N	OHA	5YEARS	Mo	Y
115	Srinivasmurthy	49 YEARS	M	10STD	WARD BOY	2	N	4YEARS	Y	OHA	4YEARS	S	Y
116	Muniyappa	78 YEARS	M	NIL	UNOCCUPIED	1	Y	8YEARS	N	I	3YEARS	S	Y
117	Narayanappa	56 YEARS	M	PUC	CONDUCTOR	2	N	1YEARS	Y	OHA	6MONTHS	S	N
118	Narayanappa	64 YEARS	M	5STD	AUTO DRIVER	2	N	9MONTHS	Y	OHA	6MONTHS	S	N
119	Sundhmetha Bai	55 YEARS	F	PUC	HOUSE WIFE	2	N	3YEARS	Y	OHA	3YEARS	S	Y
120	Venkatappa	60 YEARS	M	9STD	FACTORY	2	Y	7YEARS	Y	OHA	5YEARS	S	Y
121	Manjunathappa	48 YEARS	M	10STD	TAILOR	2	Y	2YEARS	Y	OHA	2YEARS	S	Y
122	Anand	37 YEARS	M	DEGREE	MALE NURSE	2	Y	3MONTHS	Y	NIL	NIL	S	Y
123	Isha Ahmed	75 YEARS	M	3STD	HOUSEWIFE	2	Y	9YEARS	N	OHA	9YEARS	S	Y
124	Prameela	46 YEARS	F	PUC	FACTORY	2	Y	1YEARS	Y	OHA	1YEARS	S	N
125	Neelamma	70 YEARS	F	8STD	HOUSE WIFE	1	N	17YEARS	N	I	17YEARS	S	Y
126	Vellappa	82 YEARS	M	NIL	UNOCCUPIED	2	N	5YEARS	N	OHA	5YEARS	S	Y
127	Ramappa	53 YEARS	M	DEGREE	CONSTABLE	2	Y	6YEARS	Y	OHA	6YEARS	Mo	Y
128	Muniyappa	72 YEARS	M	NIL	FARMER	2	Y	4YEARS	Y	OHA	4YEARS	H	N
129	Tamappa	65 YEARS	M	7STD	FARMER	2	N	10YEARS	Y	OHA	10YEARS	H	N
130	Shanappa	86 YEARS	F	NIL	UNOCCUPIED	2	Y	1MONTHS	Y	OHA	1MONTHS	S	N
131	Sadiya	76 YEARS	F	NIL	HOUSE WIFE	2	N	10YEARS	N	OHA	10YEARS	S	N
132	Nadia	72 YEARS	M	5STD	HOUSE WIFE	2	N	12YEARS	N	I	8YEARS	H	N
133	Beguam	66 YEARS	M	NIL	HOUSE WIFE	2	N	7YEARS	Y	OHA	7YEARS	H	N
134	Nawazzuddin	73 YEARS	F	6STD	UNOCCUPIED	2	Y	4YEARS	Y	OHA	4YEARS	Mo	N
135	Sidique	86 YEARS	M	NIL	UNOCCUPIED	2	N	7YEARS	Y	OHA	7YEARS	H	N
136	Muniyappa	83 YEARS	M	NIL	UNOCCUPIED	2	Y	3YEARS	Y	OHA	3YEARS	Mo	Y
137	Nanjegowda	80 YEARS	M	2STD	FARMER	1	Y	15YEARS	Y	I	15YEARS	Mo	Y
138	Sarojamma	50 YEARS	F	4STD	FACTORY	2	N	3MONTHS	Y	OHA	3MONTHS	Mo	N
139	Lakshman Reddy	65 YEARS	M	6STD	BUSINESS	2	N	8YEARS	N	OHA	8YEARS	S	N
140	Jayamma	60 YEARS	F	9STD	HOUSE WIFE	2	Y	5YEARS	Y	OHA	5YEARS	Mo	Y
141	M T A Drasappa	76 YEARS	M	NIL	UNOCCUPIED	1	Y	9YEARS	N	I	8YEARS	Mo	Y
142	Krishnamurthy A	74 YEARS	M	NIL	FARMER	2	N	4YEARS	Y	OHA	4YEARS	Mo	N
143	Krishnamurthy S	51 YEARS	M	8STD	TICKET COLLECTOR	2	N	2YEARS	N	NO	2YEARS	H	N
144	Chandra	47 YEARS	M	10STD	DRIVER	2	Y	3MONTHS	Y	NO	3MONTHS	H	N
145	Trnasayappa Swamy	92 YEARS	M	NIL	UNOCCUPIED	2	N	10YEARS	Y	OHA	10YEARS	S	Y
146	Muniyappa	65 YEARS	M	7STD	SUPERVISOR	1	N	6YEARS	Y	OHA	5YEARS	Mo	N
147	Prameela	74 YEARS	F	10STD	HOUSE WIFE	1	Y	2YEARS	Y	OHA	2YEARS	S	Y
148	Muniswamy	50 YEARS	M	8STD	CARPENTER	2	N	6MONTHS	Y	OHA	3MONTHS	Mo	N
149	Thimappa Swamy	65 YEARS	M	10STD	BUSINESS	2	N	7YEARS	Y	OHA	7YEARS	Mo	N
150	Ramanayak	70 YEARS	M	NIL	FARMER	2	Y	9YEARS	N	I	9YEARS	Mo	N
151	Syed Ammen	65 YEARS	M	9STD	SECURITY GUARD	2	N	20YEARS	N	OHA	20YEARS	S	Y
152	Babu Jaan	45 YEARS	M	NIL	COOLIE	2	Y	2YEARS	Y	OHA	2YEARS	S	Y
153	Aleem	45 YEARS	M	9STD	CONTRACTOR	2	N	3YEARS	Y	OHA	3YEARS	Mo	Y
154	Ramana Rao	55 YEARS	M	3STD	MASON	2	N	12YEARS	N	OHA	10YEARS	Mo	N
155	Surya	15 YEARS	M	8STD	STUDENT	1	Y	5MONTHS	Y	I	5MONTHS	Mo	Y

SL. NO.	1.1	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	1.1	1.11	2.1	2.2	2.3	2.4	2.5	2.6	3.1	3.2	3.3	3.4
1	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	A	PA	A	DA	DA
2	N	N	N	Y	DK	DK	Y	DK	DK	N	DK	A	PA	DA	A	PA	DA	PA	PA	DA	DA
3	N	N	N	DK	DK	DK	PA	N	DK	N	DK	A	A	PA	DA	DA	DA	DA	DA	PA	DA
4	Y	Y	Y	Y	Y	N	Y	DK	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	DA
5	N	N	N	Y	Y	Y	DK	DK	DK	DK	DK	A	A	PA	DA	PA	DA	DA	A	A	A
6	N	N	N	Y	DK	Y	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
7	Y	Y	Y	Y	Y	Y	Y	Y	N	N	Y	DA	PA	A	DA	DA	A	A	A	A	DA
8	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
9	N	N	N	DK	N	Y	N	DK	DK	DK	DK	PA	PA	A	A	A	A	DA	A	DA	DA
10	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	PA	A	A	A	DA
11	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	PA	A	DA	PA	PA	DA
12	Y	Y	N	Y	Y	DK	Y	N	Y	Y	Y	A	A	A	PA	DA	DA	A	A	A	A
13	Y	Y	N	Y	Y	Y	Y	DK	Y	Y	Y	DA	DA	A	DA	PA	PA	A	A	A	DA
14	N	N	N	DK	DK	N	P	P	P	DK	DK	PA	A	PA	PA	A	A	DA	DA	DA	DA
15	N	N	N	DK	DK	N	DK	N	DK	DK	DK	PA	A	PA	PA	A	A	DA	DA	DA	DA
16	N	N	N	DK	N	DK	DK	DK	DK	DK	DK	A	A	PA	PA	PA	PA	DA	DA	DA	DA
17	Y	Y	Y	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A
18	Y	Y	N	Y	N	N	N	N	Y	Y	N	A	A	A	DA	DA	PA	A	DA	A	DA
19	Y	Y	Y	Y	Y	Y	DK	DK	DK	N	Y	DA	A	DA	PA	DA	PA	A	A	A	A
20	Y	Y	N	Y	Y	DK	Y	N	Y	Y	Y	DA	A	A	A	DA	PA	A	PA	A	DA
21	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
22	N	N	N	Y	DK	DK	Y	N	Y	Y	N	A	DA	A	A	DA	DA	A	DA	DA	DA
23	Y	Y	N	Y	DK	DK	DK	DK	DK	DK	DK	PA	A	A	A	PA	PA	PA	A	A	DA
24	Y	Y	Y	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A
25	Y	Y	Y	Y	Y	Y	Y	Y	N	N	Y	DA	DA	DA	DA	DA	DA	A	A	A	DA
26	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	PA	A	A	A	DA
27	Y	Y	N	Y	Y	DK	DK	N	DK	DK	Y	DA	A	DA	PA	DA	DA	A	A	A	DA
28	Y	Y	N	Y	DK	DK	DK	DK	DK	Y	Y	PA	A	PA	DA	DA	DA	PA	PA	DA	DA
29	Y	Y	N	Y	DK	Y	N	N	Y	N	N	A	A	A	A	DA	PA	A	A	A	N
30	Y	Y	N	Y	DK	Y	N	N	Y	N	N	A	A	A	A	DA	PA	A	A	A	N
31	Y	Y	N	Y	N	DK	N	DK	DK	Y	N	A	A	A	A	DA	DA	PA	A	DA	DA
32	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	A	PA	A	DA	DA
33	N	N	N	Y	DK	DK	Y	DK	DK	N	DK	A	PA	DA	A	PA	DA	PA	PA	DA	DA
34	N	N	N	DK	DK	DK	PA	N	DK	N	DK	A	A	PA	DA	DA	DA	DA	DA	PA	DA
35	Y	Y	Y	Y	Y	N	Y	DK	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	DA
36	N	N	N	Y	Y	Y	DK	DK	DK	DK	DK	A	A	PA	DA	PA	DA	DA	A	A	A
37	N	N	N	Y	DK	Y	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
38	Y	Y	Y	Y	Y	Y	Y	Y	N	N	Y	DA	PA	A	DA	DA	A	A	A	A	DA
39	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
40	N	N	N	DK	N	Y	N	DK	DK	DK	DK	PA	PA	A	A	A	A	DA	A	DA	DA
41	Y	Y	N	Y	Y	DK	Y	DK	Y	DK	Y	DA	A	DA	PA	DA	DA	A	A	A	A
42	N	N	N	DK	DK	DK	N	N	DK	DK	DK	A	A	A	PA	DA	PA	PA	PA	A	PA
43	Y	Y	N	Y	Y	DK	Y	DK	DK	DK	Y	DA	A	DA	A	DA	DA	A	A	A	A
44	N	N	N	DK	DK	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
45	N	N	N	DK	DK	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
46	N	N	N	Y	DK	DK	Y	DK	DK	DK	DK	A	A	A	A	A	A	A	A	A	A
47	Y	Y	N	Y	Y	Y	Y	N	Y	DK	Y	DA	A	DA	A	DA	DA	A	A	A	A
48	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	PA	PA	PA	PA	PA
49	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	PA	A	A	A	PA	PA	A	PA	A	PA
50	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
51	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
52	N	N	N	DK	DK	Y	DK	DK	DK	DK	DK	A	A	A	PA	A	PA	PA	PA	PA	PA
53	Y	Y	N	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A
54	Y	Y	N	N	Y	DK	DK	DK	DK	DK	Y	A	A	PA	PA	PA	PA	A	A	A	PA
55	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A
56	Y	Y	N	DK	Y	DK	N	N	Y	DK	Y	A	A	PA	A	DA	DA	A	A	A	PA
57	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A
58	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
59	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A
60	Y	Y	N	DK	Y	Y	Y	DK	DK	DK	Y	A	A	PA	PA	DA	PA	PA	A	A	PA
61	N	N	N	N	N	N	N	N	N	N	N	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
62	N	N	N	Y	DK	Y	Y	Y	DK	Y	Y	A	A	A	A	DA	A	A	A	A	A
63	Y	Y	N	Y	DK	Y	Y	Y	DK	Y	Y	A	A	A	A	DA	A	A	A	A	A
64	N	N	N	Y	DK	Y	Y	Y	DK	Y	Y	A	A	A	A	DA	A	A	A	A	A
65	N	N	N	DK	Y	DK	DK	DK	DK	DK	DK	A	A	A	DA	PA	PA	PA	PA	PA	PA
66	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	PA	PA	PA	PA	PA
67	Y	Y	N	DK	Y	DK	DK	Y	N	DK	Y	PA	PA	A	PA	A	PA	DA	PA	DA	DA
68	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	A
69	Y	Y	N	Y	Y	Y	DK	DK	DK	Y	Y	A	A	A	A	A	A	A	A	A	A
70	N	N	N	DK	Y	Y	N	DK	Y	N	DK	A	DA	PA	A	DA	PA	A	DA	PA	A
71	Y	Y	N	Y	DK	Y	Y	DK	DK	Y	Y	DA	A	DA	PA	DA	PA	A	A	A	A
72	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
73	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	A
74	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	PA
75	Y	Y	N	Y	Y	Y	Y	Y	DK	Y	Y	DA	A	DA	A	DA	DA	A	A	A	A
76	Y	Y	N	Y	Y	DK	DK	DK	Y	DK	Y	PA	DA	A	PA	DA	PA	A	A	A	PA
77	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
78	N	N	N	Y	DK	Y	Y	N	DK	DK	N	A	A	A	A	A	A	A	A	DA	DA
79	N	N	N	N	N	N	N	N	N	N	N	A	A	A	A	A	A	DA	DA	DA	DA
80	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
81	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	PA	A	PA	PA	PA	PA	PA	PA	PA	PA
82	N	N	N	DK	DK	DK	DK	DK	DK	Y	N	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
83	Y	Y	N	Y	Y	DK	DK	Y	DK	Y	DK	A	A	PA	DA	DA	A	A	A	A	DA
84	Y	Y	Y	Y	Y	Y	Y	N	N	DK	Y	DA	A	DA	A	DA	DA	A	A	A	DA
85	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	DA	A	DA	DA	DA	DA	A	A	A	DA
86	Y	Y	N	Y	Y	DK	DK	DK	DK	Y	Y	DA	A	A	A	DA	DA	A	A	A	DA
87	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	DA	DA	DA	A	DA	PA	A	A	A	PA
88	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	A
89	Y	Y	N	Y	DK	DK	DK	DK	DK	DK	DK	DA	A	DA	PA	DA	PA	A	DA	DA	DA
90	N	N	N	Y	Y	DK	N	DK	DK	DK	Y	A	A	A	A	DA	PA	DA	DA	DA	DA
91	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
92	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	DA	A	PA	PA	PA	PA	A	A	PA	PA
93	Y	Y	N	Y	Y	DK	DK	DK	Y	DK	Y	PA	DA	A	PA	DA	PA	A	A	A	PA
94	Y	Y	N	Y	Y	N	N	N	Y	Y	Y	DA	A	A	A	DA	PA	A	A	A	DA
95	Y	Y	N	Y	N	N	DK	DK	DK	DK	N	DA	A	A	A	A	A	A	A	A	A
96	N	N	N	DK	Y	Y	N	DK	Y	N	DK	A	DA	PA	A	DA	PA	A	DA	PA	A
97	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
98	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	A	DA	DA	DA	DA	DA	DA	DA	DA	DA
99	Y	Y	N	Y	Y	DK	Y	N	DK	Y	N	A	A	A	A	DA	PA	A	DA	A	DA
100	N	N	N	N	Y	DK	DK	N	N	DK	DK	PA	PA	A	PA	DA	DA	PA	PA	A	PA
101	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
102	N	N	N	N	N	DK	DK	DK	DK	N	N	A	DA	PA	DA	PA	DA	DA	PA	PA	PA
103	Y	Y	N	Y	Y	Y	Y	N	DK	Y	Y	DA	PA	DA	A	DA	PA	A	A	A	A
104	Y	N	N	Y	DK	DK	N	DK	N	DK	Y	DA	A	A	A	DA	A	A	PA	A	PA
105	Y	Y	Y	Y	Y	N	N	N	N	N	Y	DA	A	DA	A	DA	DA	A	A	A	A
106	N	N	Y	Y	DK	DK	Y	Y	N	N	DK	PA	A	A	DA	DA	PA	PA	A	A	DA
107	Y	Y	Y	Y	DK	DK	DK	DK	N	N	N	N	A	A	PA	PA	DA	DA	A	A	PA
108	Y	Y	N	Y	Y	N	N	DK	DK	DK	Y	DA	A	A	DA	DA	PA	A	A	DA	DA
109	Y	Y	N	Y	Y	N	Y	Y	Y	Y	DK	DA	A	PA	A	A	PA	A	A	A	A
110	N	N	N	DK	DK	DK	N	DK	DK	DK	DK	A	A	A	A	A	A	A	A	A	DA
111	Y	Y	Y	Y	Y	N	DK	DK	N	DK	Y	DA	A	DA	DA	DA	PA	A	A	A	A
112	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	PA	A	A	A	DA
113	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	PA	A	DA	PA	PA	DA
114	Y	Y	N	Y	Y	DK	Y	N	Y	Y	Y	A	A	A	PA	DA	DA	A	A	A	A
115	Y	Y	N	Y	Y	Y	Y	DK	Y	Y	Y	DA	DA	A	DA	PA	PA	A	A	A	DA
116	N	N	N	DK	DK	N	P	P	P	DK	DK	PA	A	PA	PA	A	A	DA	DA	DA	DA
117	N	N	N	DK	DK	N	DK	N	DK	DK	DK	PA	A	PA	PA	A	A	DA	DA	DA	DA
118	N	N	N	DK	N	DK	DK	DK	DK	DK	DK	A	A	PA	PA	PA	PA	DA	DA	DA	DA
119	Y	Y	Y	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A
120	Y	Y	N	Y	N	N	N	N	Y	Y	N	A	A	A	DA	DA	PA	A	DA	A	DA
121	Y	Y	Y	Y	Y	Y	DK	DK	DK	N	Y	DA	A	DA	PA	DA	PA	A	A	A	A
122	Y	Y	N	Y	Y	DK	Y	N	Y	Y	Y	DA	A	A	A	DA	PA	A	PA	A	DA
123	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
124	N	N	N	Y	DK	DK	Y	N	Y	Y	N	A	DA	A	A	DA	DA	A	DA	DA	DA
125	Y	Y	N	Y	DK	DK	DK	DK	DK	DK	DK	PA	A	A	A	PA	PA	PA	A	A	DA
126	Y	Y	Y	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A
127	Y	Y	Y	Y	Y	Y	Y	Y	N	N	Y	DA	DA	DA	DA	DA	DA	A	A	A	DA
128	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	PA	A	A	A	DA
129	Y	Y	N	Y	Y	DK	DK	N	DK	DK	Y	DA	A	DA	PA	DA	DA	A	A	A	DA
130	Y	Y	N	Y	DK	DK	DK	DK	DK	Y	Y	PA	A	PA	DA	DA	DA	PA	PA	DA	DA
131	Y	Y	N	Y	DK	Y	N	N	Y	N	N	A	A	A	A	DA	PA	A	A	A	N
132	Y	Y	N	Y	DK	Y	N	N	Y	N	N	A	A	A	A	DA	PA	A	A	A	N
133	Y	Y	N	Y	N	DK	N	DK	DK	Y	N	A	A	A	A	DA	DA	PA	A	DA	DA
134	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	A	PA	A	DA	DA
135	N	N	N	Y	DK	DK	Y	DK	DK	N	DK	A	PA	DA	A	PA	DA	PA	PA	DA	DA
136	N	N	N	DK	DK	DK	PA	N	DK	N	DK	A	A	PA	DA	DA	DA	DA	DA	PA	DA
137	Y	Y	Y	Y	Y	N	Y	DK	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	DA
138	N	N	N	Y	Y	Y	DK	DK	DK	DK	DK	A	A	PA	DA	PA	DA	DA	A	A	A
139	N	N	N	Y	DK	Y	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
140	Y	Y	Y	Y	Y	Y	Y	Y	N	N	Y	DA	PA	A	DA	DA	A	A	A	A	DA
141	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA
142	N	N	N	DK	N	Y	N	DK	DK	DK	DK	PA	PA	A	A	A	A	DA	A	DA	DA
143	Y	Y	N	Y	Y	DK	Y	DK	Y	DK	Y	DA	A	DA	PA	DA	DA	A	A	A	A
144	N	N	N	DK	DK	DK	N	N	DK	DK	DK	A	A	A	PA	DA	PA	PA	PA	A	PA
145	Y	Y	N	Y	Y	DK	Y	DK	DK	DK	Y	DA	A	DA	A	DA	DA	A	A	A	A
146	N	N	N	DK	DK	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
147	N	N	N	DK	DK	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
148	N	N	N	Y	DK	DK	Y	DK	DK	DK	DK	A	A	A	A	A	A	A	A	A	A
149	Y	Y	N	Y	Y	Y	Y	N	Y	DK	Y	DA	A	DA	A	DA	DA	A	A	A	A
150	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	PA	PA	PA	PA	PA
151	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	PA	A	A	A	PA	PA	A	PA	A	PA
152	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA
153	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA
154	N	N	N	DK	DK	Y	DK	DK	DK	DK	DK	A	A	A	PA	A	PA	PA	PA	PA	PA
155	Y	Y	N	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A

SL. NO.	BCVA(OD)	BCVA (OS)	AS - OD	AS-OS	FUNDUS - OD	FUNDUS - OS
1	6/6 .	6/6.	WNL	WNL	MILD NPDR	MILD NPDR
2	6/6.	6/6	WNL	WNL	WNL	WNL
3	CF-2m	6/60.	SIMC	SIMC	WNL	WNL
4	CF-4m	CF3m	SIMC + PEX	SIMC + PEX	WNL	WNL
5	6/18.	6/60.	SIMC	SIMC	MILD NPDR	MILD NPDR
6	6/6.	6/6.	WNL	WNL	WNL	WNL
7	6/60.	6/60.	SIMC	SIMC	WNL	WNL
8	6/6.	6/6.	WNL	EXT. HARDEOLUM	WNL	WNL
9	6/9.	6/6.	WNL	WNL	MODERATE	MODERATE
10	6/12.	6/24.	PSP	PSP	WNL	WNL
11	Hm+VE	6/18.	SMC	PSP	NO GLOW	WNL
12	6/24.	6/36.	SIMC	SIMC	WNL	WNL
13	6/36.	6/24.	WNL	WNL	MODERATE	MILD NPDR
14	CF-3m	CF-CLOSE TO FACE	SIMC	RAPD	WNL	GLAUCOMATOUS OPTIC ATROPHY
15	6/12.	6/24.	PSP	PSP	WNL	WNL
16	6/36.	6/12.	SIMC	SIMC	WNL	WNL
17	6/6.	6/6.	WNL	WNL	WNL	WNL
18	6/12.	6/18.	WNL	WNL	WNL	WNL
19	6/6.	6/6.	WNL	WNL	WNL	WNL
20	6/9.	6/6.	WNL	WNL	WNL	WNL
21	6/60.	6/36.	SIMC	SIMC	MILD NPDR	MILD NPDR
22	CF-1m	CF-3m	SIMC	SIMC	HAZY	HAZY
23	CF-CLOSSE TO FACE	6/9.	SMC	PSP	NO GLOW	WNL
24	6/6.	6/6.	WNL	WNL	WNL	WNL
25	6/6.	6/6.	WNL	WNL	WNL	WNL
26	6/12.	Hm+VE	PSP	SMC	WNL	NO GLOW
27	6/18.	6/24.	WNL	WNL	MILD NPDR WITH DRUSENS IN MACULA	MILD NPDR WITH DRUSENS IN MACULA
28	6/6.	6/6.	WNL	WNL	WNL	WNL
29	CF-2m	CF-4m	SIMC	SIMC	HAZY	HAZY
30	CF-5m	6/18.	WNL	WNL	MODERATE NPDR+ CSME	MODERATE NPDR
31	6/6.	6/6.	WNL	WNL	WNL	WNL
32	CF-3m	CF-2m	PSP	PSP	ARMD	ARMD
33	6/12.	6/9.	WNL	WNL	MILD NPDR	MILD NPDR
34	6/6.	6/6.	WNL	WNL	WNL	WNL
35	6/36.	6/60.	SIMC	SIMC	MILD NPDR	MODERATE NPDR
36	CF-2m	6/60.	SIMC + PEX	PSP + PEX	HAZY	MODERATE NPDR
37	Hm+VE	PL+VE	SMC	SMC	NO GLOW	NO GLOW
38	6/6.	6/6.	WNL	WNL	WNL	WNL
39	6/18.	6/36.	SIMC	SIMC	MILD NPDR	MODERATE NPDR
40	Hm+VE	6/60.	SMC	PSP + PCO	NO GLOW	MODERATE NPDR
41	CF-5m	6/6.	SIMC	PSP	WNL	WNL
42	CF-3m	Hm+VE	SIMC	SMC	HAZY	HAZY
43	6/9.	6/18.	PSP	PSP	WNL	MILD NPDR
44	6/18.	6/36.	PSP + CHALAZION	PSP	MILD NPDR	MODERATE NPDR + CSME
45	CF-5m	6/36.	SIMC	SIMC	WNL	WNL
46	6/6.	6/6.	WNL	WNL	WNL	WNL
47	6/6.	6/6.	WNL	WNL	WNL	WNL
48	6/12.	6/18.	SIMC	SIMC	WNL	WNL
49	6/60.	6/12.	SIMC	PSP	WNL	WNL
50	6/36.	6/24.	PSP	PSP	WNL	WNL
51	6/36.	6/60.	PSP	PSP	ARMD	ARMD
52	6/12.	6/9.	PSP	PSP	WNL	WNL
53	6/6.	6/6.	WNL	ALLERGIC COWNL	WNL	WNL
54	6/6.	6/6..	WNL	WNL	WNL	WNL
55	6/6.	6/6..	WNL	WNL	WNL	WNL
56	6/36.	6/60.	PSP+PEX	PSP+PEX	MODERATE NPDR+ CSME	MILD NPDR
57	6/6.	6/12.	WNL	SIMC	WNL	WNL
58	6/12.	6/18.	WNL	WNL	WNL	WNL
59	6/9.	6/9.	WNL	WNL	WNL	WNL
60	CF-2m	6.60.	SIMC	SIMC	HAZY	WNL
61	6/9.	6/36.	WNL	WNL	WNL	WNL
62	6/36.	6/36.	PSP	PSP	CDR-0.7	CDR-0.5
63	6/12.	PL+VE	PSP	SMC	NORMAL	NO GLOW
64	6/60.	6/36.	PSP	PSP	ARMD	ARMD
65	CF-2m	6/60.	SIMC + PEX	SIMC + PEX	HAZY	WNL
66	6/9.	CF-2m	PSP+PEX	APHAKIA + CHALA	WNL	WNL
67	PL+VE	CF-3m	PSP	PSP	ARMD	ARMD
68	6/6.	6/6.	WNL	WNL	WNL	WNL
69	6/60.	CF-2m	PSP	SIMC	MODERATE NPDR+ CSME	HAZY
70	CF-2m	PL+VE	SIMC	SMC	HAZY	NO GLOW
71	CF-3m	6/6.	CORNEAL OPACITY	WNL	WNL	WNL
72	CF-2m	6/36.	SIMC	PSP	HAZY	MILD NPDR +CSME
73	6/9.	6/12.	PSP	PSP	NORMAL	NORMAL
74	6/36.	6/60.	SIMC	SIMC	CDR- 0.6	CDR-0.5
75	6/60.	6/9.	WNL	WNL	MODERATE NPDR+ CSME	MILD NPDR
76	6/6.	6/6.	WNL	WNL	WNL	WNL
77	6/36.	6/60.	SIMC	SIMC	WNL	WNL
78	CF-3m	6/9.	SIMC	PSP	HAZY	WNL
79	PL+VE	Hm+VE	SMC + PEX	SMC + PEX	NO GLOW	NO GLOW
80	CF-3m	CF-1m	SIMC	NVG + SIMC	PDR	subhyaloid haemorrhage with old PRP spots
81	6/12.	CF-2m	PSP	PSP + PCO	WNL	HAZY
82	6/18.	6/60.	SIMC	SIMC	WNL	WNL
83	6/6.	6/6.	WNL	WNL	WNL	WNL
84	6/18.	6/9.	CHALAZION	WNL	WNL	WNL
85	6/6.	6/6.	ALLERGIC CO	ALLERGIC CO	WNL	WNL
86	6/60.	6/9.	PSP	PSP	MILD NPDR + CSME	WNL
87	6/6.	6/12.	WNL	PTERYGIUM	WNL	WNL
88	6/6.	6/9.	WNL	WNL	WNL	WNL
89	6/60.	CF-4m	SIMC	SIMC	WNL	WNL
90	6/18.	6/9.	INTERNAL HARDEOLUM	WNL	WNL	WNL
91	6/60.	CF-2m	PSP+PCO	SIMC	WNL	WNL
92	6/9.	6/9.	PSP	PSP	WNL	WNL
93	6/60.	CF-4m	SIMC	SIMC	WNL	WNL
94	6/18.	6/60.	SIMC	PSP + PCO	WNL	WNL
95	6/6.	6/6.	WNL	WNL	WNL	WNL
96	6/9.	6/36.	WNL	WNL	WNL	WNL
97	6/36.	6/18.	SIMC	SIMC	WNL	WNL
98	6/9.	CF-2m	PSP + PEX	SIMC + PEX	WNL	WNL
99	6/18.	6/36.	PSP	PSP	MODERATE NPDR	MODERATE NPDR
100	6/6.	6/6.	WNL	WNL	WNL	WNL
101	Hm+VE	6/36.	SMC	PSP	NO GLOW	ARMD
102	6/12.	6/60.	PSP	PSP + PCO	NORMAL	HAZY
103	6/18.	6/36.	PSP	PSP	ARMD	ARMD
104	6/18.	CF-4m	PSP + CHALAZION	SIMC	MILD NPDR	MILD NPDR
105	6/6.	6/6.	PTERYGIUM	WNL	WNL	WNL
106	6/6.	6/6.	CORNEAL FOREIGWNL BC	WNL	WNL	WNL
107	6/18.	6/12.	WNL	WNL	WNL	WNL
108	CF-3m	6/24.	SIMC	SIMC	HAZY	WNL
109	6/12.	6/18.	WNL	WNL	WNL	WNL
110	Hm+VE	Hm+VE	SMC	SMC	HAZY	HAZY
111	6/6 .	6/6.	PSP	PSP	MILD NPDR	MILD NPDR
112	6/36.	6/6	PSP	PSP	WNL	WNL
113	CF-2m	6/12.	SIMC	SIMC	WNL	WNL
114	CF-4m	CF-1m	SIMC	SIMC	MILD NPDR + CDR - 0.6	MILD NPDR + CDR 0.8
115	6/18.	6/60.	SIMC	SIMC	WNL	WNL
116	6/60.	CF-2m	SIMC	SIMC	WNL	WNL
117	6/60.	CF-3m	SIMC	SIMC	WNL	WNL
118	6/18.	6/36.	PSP	EXT. HARDEOLUM-	MILD NPDR	MILD NPDR
119	6/9.	6/6.	WNL	WNL	WNL	WNL
120	6/12.	6/24.	PSP	PSP	DRUSENS IN MACULA	DRUSENS IN MACULA
121	Hm+VE	6/60.	SMC(TRAUMATIC)	SIMC	NO GLOW	MODERATE
122	6/6.	6/6.	WNL	WNL	WNL	WNL
123	6/36.	6/24.	PSP	PSP	WNL	WNL
124	6/60.	CF-CLOSE TO FACE	WNL	RAPD	CDR-0.6	GLAUCOMATOUS OPTIC ATROPHY
125	CF-2m	CF-5m	PSP + PCO	PSP	SEVERE NPDR	MODERATE NPDR+ CSME
126	6/36.	6/12.	PSP	PSP	ARMD	ARMD
127	6/6.	6/6.	WNL	WNL	WNL	WNL
128	6/12.	6/18.	WNL	WNL	MILD NPDR	MILD NPDR
129	6/6.	6/6.	PSP	PSP	WNL	WNL
130	6/36.	6/60.	PSP	PSP	ARMD	ARMD
131	6/60.	6/36.	SIMC	SIMC	MILD NPDR	WNL
132	CF-1m	CF-3m	SIMC	SIMC	HAZY-SUBHYALOID HAEMORRHAGE	SEVERE NPDR
133	CF-CLOSE TO FACE	6/12.	SMC	PSP	NO GLOW	MILD NPDR
134	6/60.	6/36.	PSP	PSP	ARMD	ARMD
135	6/6.	6/6.	PSP	PSP	WNL	WNL
136	6/12.	Hm+VE	PSP	SMC	MODERATE NPDR	NO GLOW
137	6/18.	6/18.	PSP	PSP	WNL	WNL
138	6/6.	6/6.	WNL	WNL	WNL	WNL
139	CF-2m	CF-4m	SIMC	SIMC	HAZY	HAZY
140	CF-5m	6/18.	PSP	PSP	MODERATE NPDR+ CSME	MODERATE NPDR
141	6/6.	6/6.	PSP	PSP	WNL	WNL
142	CF-3m	CF-2m	PSP	PSP	VERY SEVERE NPDR	SEVERE NPDR+CSME
143	6/6.	6/9.	WNL	WNL	MILD NPDR	MILD NPDR
144	6/6.	6/6.	WNL	WNL	WNL	WNL
145	6/60.	6/36.	PSP	PSP	ARMD	ARMD
146	CF-2m	6/60.	SIMC + PEX	PSP + PEX	HAZY	CDR - 0.7
147	Hm+VE	PL+VE	SMC	SMC	NO GLOW	NO GLOW
148	6/6.	6/6.	WNL	WNL	WNL	WNL
149	6/18.	6/36.	SIMC	SIMC	WNL	WNL
150	Hm+VE	6/60.	SMC	PSP + PCO	NO GLOW	WNL
151	CF-5m	6/6.	SIMC	PSP	WNL	WNL
152	CF-3m	Hm+VE	SIMC	SMC	HAZY	HAZY
153	6/9.	6/18.	PSP	PSP	WNL	MILD NPDR
154	6/18.	6/36.	PSP + CHALAZION	PSP	MILD NPDR	MODERATE NPDR + CSME
155	6/6.	6/6.	WNL	WNL	WNL	WNL

156	Savithramma	60 YEARS	F	NIL	HOUSEWIFE	2	N	2YEARS	Y	OHA	2YEARS	Mo	N	Y	Y	N	N	Y	DK	DK	DK	DK	DK	Y	A	A	PA	PA	PA	PA	A	A	A	PA	PL+VE	6/6.	RAPD	PSP	TRAUMATIC OPTIC NEUROPATHY	WNL
157	Narayanappa	55 YEARS	M	10STD	PEON	2	Y	10YEARS	Y	OHA	10YEARS	H	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A	6/6.	6/6.	WNL	WNL	WNL	WNL
158	Anusalem	45 YEARS	M	8STD	WORKER	2	Y	5YEARS	N	OHA	5YEARS	H	Y	Y	Y	N	DK	Y	DK	N	N	Y	DK	Y	A	A	PA	A	DA	DA	A	A	A	PA	6/12.	6/18.	SIMC	SIMC	WNL	WNL
159	Suma	60 YEARS	F	PUC	HELPER	2	N	2MONTHS	Y	OHA	2MONTHS	S	N	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A	6/60.	6/12.	SIMC	PSP	WNL	WNL
160	Noor Khan	78 YEARS	M	5STD	CATERER	2	N	25YEARS	N	OHA	20YEARS	S	N	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA	6/60.	Hm+VE	PSP	PSP BULLOUS KERA	MODERATE NPDR + CSME	HAZY
161	Rajini Bhai	55 YEARS	F	8STD	TAILOR	2	N	5YEARS	Y	OHA	5YEARS	Mo	N	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A	6/9.	6/6.	WNL	WNL	WNL	WNL
162	Nirmal Singh	50 YEARS	M	10STD	BUSINESS	2	N	3MONTHS	N	NIL	NIL	Mo	N	Y	Y	N	DK	Y	Y	Y	DK	DK	DK	Y	A	A	PA	PA	DA	PA	PA	A	A	PA	6/6.	6/6.	WNL	WNL	WNL	WNL
163	Rathnamma	46 YEARS	F	2STD	FARMER	1	Y	11YEARS	Y	I	11YEARS	S	Y	N	N	N	N	N	N	N	N	N	N	N	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA	6/36.	6/18.	ALLERGIC CO	WNL	MODERATE NPDR + CSME	MILD NPDR
164	Kanakamma	60 YEARS	F	2STD	FARMER	2	Y	7YEARS	Y	OHA	5YEARS	Mo	N	N	N	N	Y	DK	Y	Y	Y	DK	Y	Y	A	A	A	A	DA	A	A	A	A	A	6/60.	CF-4m	SIMC	SIMC	WNL	WNL
165	Rangaswamy	80 YEARS	M	NIL	FARMER	2	N	12YEARS	N	NIL	NIL	S	N	Y	Y	N	Y	DK	Y	Y	Y	DK	Y	Y	A	A	A	A	DA	A	A	A	A	A	CF-1m	6/18.	APHAKIA	PSP	WNL	WNL
166	Fashena	45 YEARS	F	PUC	NURSE	2	Y	5YEARS	Y	OHA	5YEARS	Mo	Y	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	DA	DA	DA	A	DA	PA	A	A	A	PA	6/6.	6/9.	WNL	WNL	WNL	WNL
167	Padmamma	40 YEARS	F	5STD	COOK	2	N	2YEARS	Y	OHA	2YEARS	S	N	N	N	N	DK	Y	DK	DK	DK	DK	DK	DK	A	A	A	DA	PA	PA	PA	PA	PA	PA	6/6.	6/12.	WNL	WNL	WNL	WNL
168	Chinamma	70 YEARS	F	NIL	HOUSEWIFE	2	N	18YEARS	N	NIL	5YEARS	S	N	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	PA	PA	PA	PA	PA	6/12.	6/12.	PSP	PSP	WNL	WNL
169	Narayanappa	70 YEARS	M	NIL	FARMER	1	Y	15YEARS	Y	OHA	15YEARS	Mo	Y	Y	Y	N	DK	Y	DK	DK	Y	N	DK	Y	PA	PA	A	PA	A	PA	DA	PA	DA	DA	6/9.	6/9.	PSP	PSP	WNL	WNL
170	Nagamma	60 YEARS	F	4STD	COOLIE	2	Y	8YEARS	Y	OHA	5YEARS	Mo	N	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	A	CF-2m	6.60.	SIMC	SIMC	HAZY	WNL
171	Thiarayappa	65 YEARS	M	3STD	GARDENER	2	Y	3YEARS	Y	OHA	3YEARS	Mo	Y	Y	Y	N	Y	Y	Y	DK	DK	DK	Y	Y	A	A	A	A	A	A	A	A	A	A	6/9.	6/60.	PSP	SIMC	WNL	HAZY
172	Navyappa	53 YEARS	M	3STD	GARDENER	2	N	5YEARS	Y	OHA	5YEARS	Mo	Y	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	A	6/18.	6/18.	WNL	WNL	CDR-0.5	CDR-0.7
173	Srinivas Rao	60 YEARS	M	9STD	CLERK	1	N	6YEARS	Y	OHA	6YEARS	Mo	Y	Y	Y	N	Y	DK	Y	Y	DK	DK	Y	Y	DA	A	DA	PA	DA	PA	A	A	A	A	6/12.	PL+VE	PSP	SMC	MODERATE NPDR	NO GLOW
174	Suksamma	60 YEARS	F	5STD	VENDOR	2	N	8YEARS	Y	OHA	8YEARS	Mo	N	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA	6/60.	6/36.	PSP	PSP	ARMD	ARMD
175	V Maliyappa	86 YEARS	F	NIL	UNOCCUPIED	2	Y	10YEARS	Y	OHA	10YEARS	S	Y	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	A	CF-2m	6/60.	SIMC + PEX	SIMC + PEX	HAZY	CDR - 0.5
176	Krinsnappa	65 YEARS	M	10STD	CONSTABLE	2	N	3MONTHS	Y	NIL	NIL	H	N	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	PA	CF-2m	6/9.	APHAKIA+PEX	PSP + PEX + CHALA	WNL	WNL
177	Venkatappa	68 YEARS	M	10STD	SECURITY GUARD	2	Y	2YEARS	Y	OHA	2YEARS	Mo	Y	Y	Y	N	Y	Y	Y	Y	Y	DK	Y	Y	DA	A	DA	A	DA	DA	A	A	A	A	PL+VE	CF-3m	CORNEAL OPACITY	PSP + PCO	NO GLOW	HAZY
178	Mary	63 YEARS	F	GRADUATE	NURSE	2	N	10YEARS	Y	OHA	8YEARS	S	N	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	DA	DA	DA	A	DA	PA	A	A	A	PA	6/12.	CF-3m	PSP	SIMC	WNL	HAZY
179	V Narayanappa	45 YEARS	M	10STD	PROVISION STORE	2	Y	6MONTHS	Y	OHA	6MONTHS	S	N	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA	6/6.	6/36.	WNL	PTERYGIUM	WNL	WNL
180	Lakshmamma	68YEARS	F	5STD	COOK	2	Y	3YEARS	N	NIL	NIL	S	N	N	N	N	Y	DK	Y	Y	N	DK	DK	N	A	A	A	A	A	A	A	A	DA	DA	CF-2m	PL+VE	SIMC	SMC	HAZY	NO GLOW
181	Krishnappa	65 YEARS	M	7STD	TAILOR	1	Y	5YEARS	Y	I	5YEARS	Mo	Y	Y	Y	N	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A	CF-3m	6/6.	CORNEAL OPACITY	WNL	WNL	WNL
182	T Narayanswamy	44 YEARS	M	3STD	MASON	2	N	4MONTHS	N	NIL	NIL	Mo	N	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA	6/6.	6/6.	WNL	WNL	WNL	WNL
183	Saidabegum	54 YEARS	F	PUC	BUSINESS	1	Y	3YEARS	Y	I	3YEARS	S	Y	Y	N	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A	PA	6/9.	6/6.	WNL	WNL	WNL	WNL
184	Mahasob Pasha	63 YEARS	M	10STD	CARPENTER	2	Y	10YEARS	Y	OHA	10YEARS	H	Y	N	N	N	DK	DK	DK	DK	DK	DK	Y	N	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA	6/36.	6/60.	SIMC + PEX	SIMC + PEX	CDR- 0.6	CDR-0.8
185	Noorulla	60 YEARS	M	4STD	PAINTER	2	N	8YEARS	Y	OHA	8YEARS	S	Y	Y	Y	N	Y	Y	DK	DK	Y	DK	Y	DK	A	A	PA	DA	DA	A	A	A	A	DA	CF-4m	6/9.	SIMC	PSP	MODERATE NPDR+ CSME	MILD NPDR
186	Venkatamma	60 YEARS	F	NIL	HOUSE MAID	2	N	2YEARS	Y	OHA	2YEARS	S	N	Y	Y	Y	Y	Y	Y	Y	N	N	DK	Y	DA	A	DA	A	DA	DA	A	A	A	DA	6/6.	6/6.	PSP	PSP	WNL	WNL
187	Bhagyamma	42 YEARS	F	10STD	FACTORY	2	N	9MONTHS	Y	OHA	9MONTHS	S	Y	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	DA	A	DA	DA	DA	DA	A	A	A	DA	6/6.	6/6.	WNL	WNL	WNL	WNL
188	Rajamma	70 YEARS	F	NIL	HOUSEWIFE	2	Y	13YEARS	Y	OHA	13YEARS	S	Y	Y	Y	N	Y	Y	DK	DK	DK	DK	Y	Y	DA	A	A	A	DA	DA	A	A	A	DA	CF-3m	6/9.	SIMC	PSP	HAZY	WNL
189	Mamsareni	45 YEARS	F	6STD	FACTORY	2	Y	2YEARS	Y	OHA	2YEARS	Mo	N	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	DA	DA	DA	A	DA	PA	A	A	A	PA	6/6.	6/6.	WNL	WNL	WNL	WNL
190	Selvary	52 YEARS	M	5STD	WELDER	2	Y	5YEARS	N	OHA	3YEARS	S	N	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	A	CF-3m	CF-1m	WNL	NVG	PDR	VH
191	Ashwathappa	75 YEARS	M	4STD	PLUMBER	2	Y	2YEARS	Y	OHA	2YEARS	S	N	Y	Y	N	Y	DK	DK	DK	DK	DK	DK	DK	DA	A	DA	PA	DA	PA	A	DA	DA	DA	6/12.	CF-2m	PSP	PSP + PCO	WNL	HAZY
192	Tulasyappa	46 YEARS	M	PUC	PROVISION STORE	2	Y	6YEARS	Y	OHA	4YEARS	Mo	N	N	N	N	Y	Y	DK	N	DK	DK	DK	Y	A	A	A	A	DA	PA	DA	DA	DA	DA	6/12.	6/6.	WNL	WNL	WNL	WNL
193	Harish	36 YEARS	M	5STD	MECHANIC	2	Y	2YEARS	Y	OHA	2YEARS	Mo	N	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA	6/6.	6/6.	WNL	WNL	WNL	WNL
194	Chellamma	70 YEARS	F	2STD	HOUSEWIFE	2	Y	7YEARS	Y	OHA	4YEARS	Mo	N	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	DA	A	PA	PA	PA	PA	A	A	PA	PA	6/18.	6/9.	CHALAZION	WNL	WNL	WNL
195	Praful	40 YEARS	F	9STD	TAILOR	1	Y	11YEARS	Y	I	11YEARS	S	N	Y	Y	N	Y	Y	DK	DK	DK	Y	DK	Y	PA	DA	A	PA	DA	PA	A	A	A	PA	6/6.	6/6.	ALLERGIC CO	ALLERGIC CO	WNL	WNL
196	Chinna Thesi	63 YEARS	F	PUC	LADY CONSTABLE	2	Y	7YEARS	Y	OHA	7YEARS	Mo	Y	Y	Y	N	Y	Y	N	N	N	Y	Y	Y	DA	A	A	A	DA	PA	A	A	A	DA	6/6.	6/9.	PSP	PSP	WNL	WNL
197	Hanumanappa	62 YEARS	M	9STD	SECURITY GUARD	1	N	15YEARS	N	I	10YEARS	S	Y	Y	Y	N	Y	N	N	DK	DK	DK	DK	N	DA	A	A	A	A	A	A	A	A	A	6/6.	6/12.	WNL	PTERYGIUM	MILD NPDR	MILD NPDR
198	Guruanna Reddy	72 YEARS	M	5STD	TAILOR	2	Y	6YEARS	Y	OHA	6YEARS	S	N	N	N	N	DK	Y	Y	N	DK	Y	N	DK	A	DA	PA	A	DA	PA	A	DA	PA	A	6/6.	6/9.	WNL	WNL	WNL	WNL
199	Chinna Thesi	56 YEARS	M	5STD	PEON	2	Y	7MONTHS	Y	OHA	2MONTHS	S	N	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA	6/12.	6/6.	SIMC	WNL	WNL	WNL
200	Sadashivanand	66 YEARS	M	9STD	DRIVER	1	Y	10YEARS	Y	OHA	10YEARS	S	N	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	A	DA	DA	DA	DA	DA	DA	DA	DA	DA	6/18.	6/9.	INTERNALL HARDEOLUM	PSP	WNL	WNL
201	Jayamma	55 YEARS	F	NIL	COOK	2	Y	3MONTHS	Y	OHA	3MONTHS	S	N	Y	Y	N	Y	Y	DK	Y	N	DK	Y	N	A	A	A	A	DA	PA	A	DA	A	DA	6/60.	CF-2m	PSP+PCO	SIMC	WNL	WNL
202	Rukkamma	53 YEARS	F	NIL	COOK	2	Y	4YEARS	Y	OHA	4YEARS	S	Y	N	N	N	N	Y	DK	DK	N	N	DK	DK	PA	PA	A	PA	DA	DA	PA	PA	A	PA	6/9.	6/9.	WNL	WNL	WNL	WNL
203	Sudarshini	62 YEARS	F	10STD	MAID	1	Y	7MONTHS	N	I	7MONTHS	Mo	Y	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA	6/60.	CF-4m	SIMC	SIMC	WNL	WNL
204	Tulasi Reddy	46 YEARS	M	4STD	ELECTRICIAN	1	N	1MONTHS	Y	NIL	1MONTHS	S	Y	N	N	N	N	N	DK	DK	DK	DK	N	N	A	DA	PA	DA	PA	DA	DA	PA	PA	PA	6/6.	6/6.	WNL	WNL	WNL	WNL
205	Ramappa	60 YEARS	M	2STD	FLORIST	2	N	2YEARS	Y	OHA	2YEARS	S	N	Y	Y	N	Y	Y	Y	Y	N	DK	Y	Y	DA	PA	DA	A	DA	PA	A	A	A	A	6/36.	6/6.	PSP+PCO	PSP	WNL	WNL
206	Shanthakumar	51 YEARS	M	6STD	DRIVER	2	N	3YEARS	Y	OHA	3YEARS	Mo	N	Y	N	N	Y	DK	DK	N	DK	N	DK	Y	DA	A	A	A	DA	A	A	PA	A	PA	6/9.	6/6.	WNL	WNL	WNL	WNL
207	Ratna	33 YEARS	F	PUC	NURSE	2	N	10YEARS	Y	OHA	10YEARS	S	N	Y	Y	Y	Y	Y	N	N	N	N	N	Y	DA	A	DA	A	DA	DA	A	A	A	A	6/6.	6/6.	WNL	WNL	WNL	WNL
208	Kasturi	58 YEARS	F	2STD	FLORIST	2	N	1YEARS	Y	OHA	1YEARS	Mo	N	N	N	Y	Y	DK	DK	Y	Y	N	N	DK	PA	A	A	DA	DA	PA	PA	A	A	DA	6/9.	6/6.	PEX	PEX	WNL	WNL
209	Rukkamma	53 YEARS	F	PUC	PROVISION STORE	2	N	5YEARS	Y	OHA	3YEARS	S	N	Y	Y	Y	Y	DK	DK	DK	DK	N	N	N	N	A	A	PA	PA	DA	DA	A	A	PA	6/18.	6/36.	SIMC	SIMC	MODERATE NPDR	MODERATE NPDR
210	Jayamma	55 YEARS	F	NIL	MAID	2	N	3YEARS	N	NIL	NIL	S	N	Y	Y	N	Y	Y	N	N	DK	DK	DK	Y	DA	A	A	DA	DA	PA	A	A	DA	DA	6/6.	6/6.	WNL	WNL	WNL	WNL
211	Vanajakshi	54 YEARS	F	NIL	HELPER	2	N	6MONTHS	Y	OHA	6MONTHS	Mo	Y	Y	Y	N	Y	Y	N	Y	Y	Y	Y	DK	DA	A	PA	A	A	PA	A	A	A	A	6/6.	6/9.	WNL	PTERYGIUM	WNL	WNL
212	Thilamma	90 YEARS	F	NIL	UNOCCUPIED	2	Y	5YEARS	Y	OHA	5YEARS	Mo	Y	N	N	N	DK	DK	DK	N	DK	DK	DK	DK	A	A	A	A	A	A	A	A	A	DA	6/36.	6/60.	PSP	PSP + PCO	ARMD	HAZY
213	Margret	73 YEARS	F	PUC	NURSE	2	Y	6YEARS	Y	OHA	6YEARS	S	N	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	PA	A	A	A	DA	6/18.	6/36.	PSP	PSP	ARMD	ARMD
214	Sudharshan	63 YEARS	M	9STD	CATERER	1	Y	19YEARS	Y	I	19YEARS	S	N	Y	Y	N	Y	Y	DK	DK	N	DK	DK	Y	DA	A	DA	PA	DA	DA	A	A	A	DA	6/18.	6/9.	PSP + CHALAZION	PSP	WNL	WNL
215	Radha	32 YEARS	F	10STD	FACTORY	2	Y	3YEARS	Y	OHA	3YEARS	S	Y	Y	Y	N	Y	DK	DK	DK	DK	DK	Y	Y	PA	A	PA	DA	DA	DA	PA	PA	DA	DA	6/6.	6/6.	PTERYGIUM	WNL	WNL	WNL
216	B S Adinarayan Shetty	73 YEARS	M	PUC	BUSINESS	2	N	5YEARS	Y	OHA	5YEARS	S	N	Y	Y	N	Y	DK	Y	N	N	Y	N	N	A	A	A	A	DA	PA	A	A	A	N	PL-VE	6/9.	PTHISIS	PSP	.	WNL
217	M Ramappa	77 YEARS	M	10STD	PRINTER	2	N	7YEARS	N	OHA	2YEARS	S	N	Y	Y	N	Y	DK	Y	N	N	Y	N	N	A	A	A	A	DA	PA	A	A	A	N	6/18.	6/12.	PSP	PSP	WNL	WNL
218	Redappa	50 YEARS	M	5STD	MASON	2	N	9YEARS	Y	OHA	7YEARS	S	N	Y	Y	N	Y	N	DK	N	DK	DK	Y	N	A	A	A	A	DA	DA	PA	A	DA	DA	CF-3m	6/24.	WNL	WNL	VERY SEVERE NPDR + CSME	MILD NPDR
219	Sharadamma	55 YEARS	F	1STD	VENDOR	2	N	4YEARS	Y	OHA	4YEARS	S	N	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	A	PA	A	DA	DA	PL+VE	CF-2m	NVI	WNL	PDR	VERY SEVERE NPDR
220	Parvathamma	57 YEARS	F	NIL	COOLIE	1	N	4YEARS	Y	OHA	4YEARS	S	Y	N	N	N	Y	DK	DK	Y	DK	DK	N	DK	A	PA	DA	A	PA	DA	PA	PA	DA	DA	6/9.	6/9.	WNL	WNL	WNL	WNL
221	Lakshmamma	65 YEARS	F	NIL	FARMER	2	Y	7YEARS	Y	I	7YEARS	Mo	Y	N	N	N	DK	DK	DK	PA	N	DK	N	DK	A	A	PA	DA	DA	DA	DA	DA	PA	DA	6/12.	6/9.	PSP	PSP	MILD NPDR	MILD NPDR
222	Nagarappa	65 YEARS	M	9STD	BUTCHER	2	Y	7MONTHS	Y	OHA	7MONTHS	S	Y	Y	Y	Y	Y	Y	N	Y	DK	Y	Y	Y	DA	A	DA	PA	DA	DA	A	A	A	DA	6/6.	6/6.	WNL	WNL	WNL	WNL
223	Gowramma	55 YEARS	F	5STD	COOK	2	Y	9MONTHS	Y	OHA	9MONTHS	S	Y	N	N	N	Y	Y	Y	DK	DK	DK	DK	DK	A	A	PA	DA	PA	DA	DA	A	A	A	6/36.	6/60.	SIMC	SIMC	MILD NPDR	MODERATE NPDR
224	Lakshmidevamma	65 YEARS	F	PUC	HOUSE WIFE	2	Y	5YEARS	Y	OHA	5YEARS	Mo	N	N	N	N	Y	DK	Y	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA	CF-2m	6/60.	SIMC + PEX	PSP + PEX	HAZY	MODERATE NPDR+CDR - 0.6
225	Govindappa	70 YEARS	M	NIL	UNOCCUPIED	2	N	6YEARS	N	OHA	6YEARS	Mo	N	Y	Y	Y	Y	Y	Y	Y	Y	N	N	Y	DA	PA	A	DA	DA	A	A	A	A	DA	Hm+VE	PL+VE	SMC	SMC	NO GLOW	NO GLOW
226	Narayanamma	50 YEARS	F	NIL	HOUSEWIFE	2	Y	7YEARS	N	OHA	2YEARS	S	Y	N	N	N	N	N	N	N	N	N	N	N	DA	DA	DA	DA	DA	DA	DA	DA	DA	DA	6/6.	6/6.	WNL	WNL	NORMAL	NORMAL
227	Muniyamma	60 YEARS	F	5STD	FLORIST	1	Y	11YEARS	N	I	7YEARS	H	Y	N	N	N	DK	N	Y	N	DK	DK	DK	DK	PA	PA	A	A	A	A	DA	A	DA	DA	6/18.	6/36.	SIMC	SIMC	MILD NPDR	MODERATE NPDR
228	Subramani	45 YEARS	M	8STD	GARDENER	2	N	3MONTHS	Y	NIL	NIL	Mo	N	Y	Y	N	Y	Y	DK	Y	DK	Y	DK	Y	DA	A	DA	PA	DA	DA	A	A	A	A	6/6.	6/6.	WNL	WNL	WNL	WNL
229	Dhanalakshmi	40 YEARS	F	PUC	BUSINESS	2	N	9MONTHS	N	OHA	7MONTHS	S	N	N	N	N	DK	DK	DK	N	N	DK	DK	DK	A	A	A	PA	DA	PA	PA	PA	A	PA	6/6.	6/6.	WNL	WNL	WNL	WNL
230	Lakshmi	45 YEARS	F	PUC	FACTORY	2	N	4YEARS	Y	OHA	4YEARS	Mo	N	Y	Y	N	Y	Y	DK	Y	DK	DK	DK	Y	DA	A	DA	A	DA	DA	A	A	A	A	6/9.	6/6.	WNL	WNL	WNL	WNL
231	Venkatesh Murthy	61 YEARS	M	9STD	ELECTRICIAN	1	Y	7YEARS	Y	OHA	7YEARS	S	Y	N	N	N	DK	DK	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA	6/9.	6/18.	PSP	PSP	WNL	MILD NPDR
232	Pushpamma	58 YEARS	F	3STD	MAID	2	N	3YEARS	Y	OHA	3YEARS	Mo	Y	N	N	N	DK	DK	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA	6/12.	6/9.	PSP + CHALAZION	PSP	WNL	WNL
233	Muniyappa	58 YEARS	M	7STD	MECHANIC	2	N	2YEARS	Y	OHA	2YEARS	N	Y	N	N	N	Y	DK	DK	Y	DK	DK	DK	DK	A	A	A	A	A	A	A	A	A	A	CF-5m	6/36.	SIMC	SIMC	WNL	WNL
234	Shantamma	50 YEARS	F	PUC	CONDUCTOR	2	N	3MONTHS	Y	OHA	3MONTHS	S	N	Y	Y	N	Y	Y	Y	Y	N	Y	DK	Y	DA	A	DA	A	DA	DA	A	A	A	A	6/6.	6/6.	WNL	WNL	WNL	WNL
235	A V Ramappa	79 YEARS	M	NIL	UNOCCUPIED	2	N	8YEARS	Y	OHA	6YEARS	S	N	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	A	A	A	A	A	PA	PA	PA	PA	PA	6/6.	6/6.	PSP	PSP	WNL	WNL
236	Venkatalakshmi	80 YEARS	F	NIL	HOUSEWIFE	2	Y	5YEARS	N	OHA	3YEARS	S	Y	Y	Y	N	Y	Y	DK	DK	DK	DK	DK	DK	PA	A	A	A	PA	PA	A	PA	A	PA	6/12.	6/18.	SIMC	SIMC	WNL	WNL
237	Ramappa	40 YEARS	M	10STD	DRIVER	1	Y	2YEARS	N	I	2YEARS	S	Y	N	N	N	DK	DK	DK	DK	DK	DK	DK	DK	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA	6/6.	6/6.	WNL	WNL	WNL	WNL
238	Sukuman	40 YEARS	M	6STD	PLUMBER	2	N	3YEARS	Y	OHA	3YEARS	S	N	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA	6/60.	6/24.	WNL	WNL	MODERATE NPDR + CSME	MODERATE NPDR + CSME
239	Muniyappa	82YEARS	M	4STD	MASON	2	N	1YEARS	Y	OHA	1YEARS	S	Y	N	N	N	DK	DK	Y	DK	DK	DK	DK	DK	A	A	A	PA	A	PA	PA	PA	PA	PA	6/36.	6/60.	PSP	PSP	ARMD	ARMD
240	Ramamma	50 YEARS	F	5STD	FLORIST	2	Y	2YEARS	N	OHA	2YEARS	S	Y	Y	Y	N	Y	Y	Y	DK	DK	DK	DK	Y	DA	A	DA	PA	DA	PA	A	A	A	A	6/6.	6/6.	WNL	WNL	WNL	WNL
241	Shivashankar	52 YEARS	M	PUC	CONSTABLE	2	Y	3YEARS	N	OHA	3YEARS	S	N	Y	Y	N	N	Y	DK	DK	DK	DK	DK	Y	A	A	PA	PA	PA	PA	A	A	A	PA	6/6.	6/6.	WNL	ALLERGIC CO	WNL	WNL
242	Usha	48 YEARS	F	8STD	VENDOR	2	N	5YEARS	N	OHA	5YEARS	Mo	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A	6/6.	6/6..	WNL	WNL	WNL	WNL
243	Sriramappa	60 YEARS	M	NIL	FISHER MAN	2	N	4YEARS	Y	OHA	4YEARS	S	Y	Y	Y	N	DK	Y	DK	N	N	Y	DK	Y	A	A	PA	A	DA	DA	A	A	A	PA	6/6.	6/6..	WNL	WNL	WNL	WNL
244	Srinivasreddy	62 YEARS	M	NIL	BUTCHER	1	Y	8YEARS	N	I	3YEARS	S	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A	CF-3m	6/9.	PSP+PCO+PEX	PSP+PEX	OPTIC NEURITIS	WNL
245	Ashwathappa	72 YEARS	M	7STD	FLORIST	2	N	1YEARS	Y	OHA	6MONTHS	S	N	N	N	N	DK	Y	DK	DK	DK	DK	Y	Y	A	A	DA	A	DA	DA	DA	A	A	DA	6/6.	6/12.	WNL	SIMC	WNL	WNL
246	Subramani	65 YEARS	M	10STD	AUTO DRIVER	2	N	9MONTHS	Y	OHA	6MONTHS	S	N	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	A	6/12.	6/18.	WNL	WNL	WNL	WNL
247	Parvathamma	55 YEARS	F	NIL	MAID	2	N	3YEARS	Y	OHA	3YEARS	S	Y	Y	Y	N	DK	Y	Y	Y	DK	DK	DK	Y	A	A	PA	PA	DA	PA	PA	A	A	PA	6/9.	6/9.	WNL	WNL	WNL	WNL
248	Santhamma	70 YEARS	F	NIL	COOK	2	Y	7YEARS	Y	OHA	5YEARS	S	Y	N	N	N	N	N	N	N	N	N	N	N	PA	PA	PA	PA	PA	PA	PA	PA	PA	PA	CF-2m	6.60.	SIMC	SIMC	HAZY	WNL
249	Chaitra	38 YEARS	F	PUC	NURSE	2	Y	2YEARS	Y	OHA	2YEARS	S	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	DA	PA	DA	PA	DA	PA	A	A	A	PA	6/6.	6/6.	WNL	WNL	WNL	WNL
250	Reddappa	60 YEARS	M	PUC	CONDUCTOR	2	Y	8YEARS	Y	OHA	8YEARS	S	Y	Y	Y	N	Y	DK	Y	Y	Y	DK	Y	Y	A	A	A	A	DA	A	A	A	A	A	6/36.	6/36.	PSP	PSP	CDR-0.7	CDR-0.5