“CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE II DIABETES MELLITUS PATIENTS”

By

DR. ANUNITHA RAYAPURAJU, M.B.B.S

Dissertation submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, CENTRE, TAMAKA, KOLAR

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

UNDER THE GUIDANCE OF
DR. SANGEETHA T
M.B.B.S, M.S, F.P.R.S

UNDER THE CO-GUIDANCE OF
DR. YASHWANTH A L
MD, DM-CARDIO

DEPARTMENT OF OPHTHALMOLOGY

SRI DEVARAJ URS MEDICAL COLLEGE

TAMAKA, KOLAR

2025

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **―CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE II DIABETES MELLITUS PATIENTS‖** is a bonafide and genuine research work carried out by me under the guidance of **DR. SANGEETHA T** , M.B.B.S., M.S., F.P.R.S., Professor and Head of the Department, Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamaka, Kolar and in co-guidance of **DR. YASHWANTH A L**, MD, DM-CARDIO, Consultant Interventional Cardiologist, Narayana Health Heart Centre, RLJH in partial for the award of M.S degree in Ophthalmology to be held in 2025. This dissertation has not been submitted in part or full to any other University or towards any other degree before this below mentioned date.

Date: **Signature of the Candidate**

Place: Kolar **DR. ANUNITHA RAYPURAJU**

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation **"CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE II DIABETES MELLITUS PATIENTS"** is a Bonafide research work done by **DR. ANUNITHA RAYAPURAJU** in partial fulfilment of the requirement for the degree of MASTER OF SURGERY IN OPHTHALMOLOGY as per regulations of SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, KOLAR. I have great pleasure in forwarding this to the University.

Date:

Place: Kolar

DR. SANGEETHA T
M.B.B.S., M.S., F.P.R.S.
Professor and Head of the Department,
Department of Ophthalmology,
Sri Devaraj Urs Medical College
Tamaka, Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE

CERTIFICATE BY THE CO-GUIDE

This is to certify that the dissertation **"CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE II DIABETES MELLITUS PATIENTS"** is a Bonafide research work done by **DR. ANUNITHA RAYAPURAJU** in partial fulfilment of the requirement for the degree of MASTER OF SURGERY IN OPHTHALMOLOGY as per regulations of SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, KOLAR. I have great pleasure in forwarding this to the University.

DATE:

PLACE: KOLAR

DR. YASHWANTH A L
M.B.B.S., M.D., D.M.
INTERVENTION CARDIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE

ENDORSEMENT BY THE HOD, PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **―CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE II DIABETES MELLITUS PATIENTS‖** is a Bonafide research work done by **DR.ANUNITHA RAYAPURAJU** under the guidance of DR. SANGEETHA T , M.B.B.S., M.S., F.P.R.S., Professor and Head of the Department, Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamaka, Kolar and in co-guidance of Dr. YASHWANTH. A, MD, DM-CARDIO, Consultant Interventional Cardiologist, Narayana Health Heart Centre, RLJH.

DR. SANGEETHA.T
Professor and HOD
Department of Ophthalmology,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date
Place: Kolar

DR. PRABHAKAR K
Principal
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date
Place: Kolar

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that Sri Devaraj Urs Academy of Higher Education and Research Centre, Kolar shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic / research purpose.

Date: **Signature of the Candidate**

Place: Kolar **DR. ANUNITHA RAYAPURAJU**

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH

SRI DEVARAJ URS MEDICAL COLLEGE

Tamaka, Kolar

INSTITUTIONAL ETHICS COMMITTEE

No. DMC/KLR/IEC/85/ 2023-24 **Date: 10/04/2023**

PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **" Correlation Of Diabetic Retinopathy With Myocardial Remodeling And Quality Of Life In Type Ii Diabetes Mellitus Patients"** being investigated by **Dr.Anunitha Rayapuraju, Dr. Sangeetha. T, & Dr. Yashwanth. L**[1] in the Departments of Ophthalmology & Narayana Health Heart Centre, R. L. Jalappa Hospital[1] at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

Sujatha M.P
Member Secretary
Member Secretary
Institutional Ethics Committee
Sri Devaraj Urs Medical College
Tamaka, Kolar.

Chairman
CHAIRMAN
Institutional Ethics Committee
Sri Devaraj Urs Medical College
Tamaka, Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH
TAMAKA, KOLAR, KARNATAKA, INDIA 563103

CERTIFICATE OF PLAGIARISM CHECK

Title of the Thesis/Dissertation	**CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE 2 DIABETES MELLITUS PATIENTS**
Name of the Student	DR ANUNITHA RAYAPURAJU
Registration Number	22OP1083
Name of the Supervisor / Guide	DR SANGEETHA T
Department	OPHTHALMOLOGY
Acceptable Maximum Limit (%) of Similarity (PG Dissertation /Ph.D. Thesis)	10 %
Similarity	10%
Software used	TURNITIN
Paper ID	2675535437
ORCID ID	0009-0002-1573-0631
Submission Date	15/05/2025

Signature of Student

Signature of Guide/Supervisor

Professor & HOD
Department of Ophthalmolgy
Sri Devaraj Urs Medical College
Tamaka, Kolar - 563 101

HOD Signature

Professor & HOD
Department of Ophthalmolgy
Sri Devaraj Urs Medical College
Tamaka, Kolar - 563 101

University Librarian

University Library
Learning Resource Centre
SDUAHER, Tamaka
KOLAR-563103

PG Coordinator

PG Coordinator
Sri Devaraj Urs Medical College
Tamaka, Kolar-563103

turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	DR ANUNITHA RAYAPURAJU
Assignment title:	PG Dissertation - 2025
Submission title:	CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL...
File name:	QUALITY_OF_LIFE_IN_TYPE_II_DIABETES_MELLITUS_PATIENTS_...
File size:	328.6K
Page count:	44
Word count:	17,949
Character count:	107,381
Submission date:	15-May-2025 04:27PM (UTC+0530)
Submission ID:	2675535437

Document Viewer

Turnitin Originality Report

Processed on: 15-May-2025 16:28 IST
ID: 2675535437
Word Count: 17949
Submitted: 3

CORRELATION OF DIABETIC RETINOPATHY WITH MYOC... By DR ANUNITHA RAYAPURAJU

Similarity Index	Similarity by Source	
10%	Internet Sources:	9%
	Publications:	10%
	Student Papers:	2%

include quoted | include bibliography | excluding matches < 14 words | mode: quickview (classic) report | print | refresh | download

1% match ("Biomarkers in Diabetes", Springer Science and Business Media LLC, 2023)
"Biomarkers in Diabetes", Springer Science and Business Media LLC, 2023

<1% match (Internet from 20-Mar-2025)
https://www.frontiersin.org/research-topics/29401/epub

<1% match (Internet from 19-Mar-2025)
https://www.frontiersin.org/research-topics/15049/epub

<1% match (Internet from 04-Apr-2025)
https://www.frontiersin.org/journals/pharmacology/articles/10.3389/fphar.2025.1548491/full

<1% match (Internet from 29-Mar-2025)
https://www.frontiersin.org/research-topics/54761/epub

<1% match ("Abstracts of the IDF Congress in Paris 2003", Diabetologia, 2003)
"Abstracts of the IDF Congress in Paris 2003", Diabetologia, 2003

<1% match (Internet from 27-Mar-2025)
https://mdpi-res.com/d_attachment/ijms/ijms-25-07569/article_deploy/ijms-25-07569.pdf?version=1720610329

<1% match (Internet from 13-Jan-2024)
https://mdpi-res.com/bookfiles/book/8303/Clinical_Research_on_Type_2_Diabetes_and_Its_Complications.pdf?v=1704820833

<1% match (Internet from 20-Nov-2024)
https://mdpi-res.com/bookfiles/book/9435/Vascular_Oxidative_and_Inflammatory_Dysregulation_in_Neurodegenerative_Diseases_Current_Status_and_Futu
v=1732068510

<1% match ()
Lucia Saucedo, Isabel B. Pfister, Christin Schild, Justus G. Garweg. "Association of inflammation-related markers and diabetic retinopathy severity in the aqueous humor, but not serum of type 2 diabetic patients", PLOS ONE

<1% match ()
Wenhua Yu, Bo Yang, Siting Xu, Yun Gao, Yan Huang, Zhongqun Wang. "Diabetic Retinopathy and Cardiovascular Disease: A Literature Review", Diabetes, Metabolic Syndrome and Obesity

<1% match (Internet from 05-May-2025)
https://pmc.ncbi.nlm.nih.gov/articles/PMC11762433/

<1% match (Internet from 15-Dec-2024)
https://pmc.ncbi.nlm.nih.gov/articles/PMC10811430/

<1% match (Internet from 23-Feb-2025)
https://pmc.ncbi.nlm.nih.gov/articles/PMC11450131/

<1% match (Internet from 17-Feb-2021)
https://dagensdiabetes.se/index.php/component/content/archive/2015/6

<1% match (Internet from 10-Mar-2020)
https://worldwidescience.org/topicpages/d/diabetic+retinopathy.html

<1% match (Internet from 05-Mar-2025)
https://ijmedph.org/Issue/1048

<1% match (Internet from 05-May-2025)
https://ijprt.org/index.php/pub/article/download/381/292/748

<1% match (Internet from 26-Jun-2024)
http://ijlbpr.com

<1% match (Internet from 04-Feb-2023)
https://rssdi.in/newwebsite/pdfdata/Volume-40-Issue3-july-september-2020.pdf

<1% match (Internet from 28-Sep-2022)
https://biblio.ugent.be/publication/4304894

ACKNOWLEDGEMENT

First and foremost, I would like to thank the almighty for giving me the strength and ability to carry out this study. I take this opportunity to express my gratitude to my teacher and guide **DR. SANGEETHA T , M.B.B.S., M.S., F.P.R.S.,** Professor and Head of the Department, Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamaka, Kolar and co-guide of **DR. YASHWANTH. A, MD, DM-CARDIO**, Consultant Interventional Cardiologist, Narayana Health Heart Centre, RLJH, for their invaluable guidance, constant support and encouragement during the course of study and completion of dissertation.

I would like to express my wholehearted thanks and gratitude to my Professors**, DR. MANJULA T.R, DR. USHA. B R, Dr. RASHMI G** and Associate Professors **DR. CHAITRA M C**, **DR. INCHARA N** Assistant Professors **DR. APOORVA N** and **DR. NAVEENA A**, **DR. NARENDRAN BS**, **DR. ATHIRA K** and Senior Residents **DR. SHWETHA** Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamaka, Kolar for their constant help and suggestions and encouragement rendered during this study.

I thank all my teachers throughout my life for having made me what I am today. My gratitude and thanks to **DR. PRABHAKAR K**, Principal, Sri Devaraj Urs Medical College, Tamaka, Kolar, for letting me use the college and hospital facilities and resources.

I would like to specially thank **DR. HITHESH, DR**. **HANEELA, DR.ALISHA, DR.SANJANA, DR.RAVEENA , DR.PRAMUKH, DR. ATHMIKA, DR. HIMATEJA & DR. BHAVISHYA** for all their help during this study and making my journey through it smooth. The list will be incomplete without all my seniors and juniors and all my friends for their help and support. I am deeply grateful for the blessings and love of my late mother, **MRS. RAYAPURAJU MADHAVI LATHA**. Her memory and guidance continue to inspire me every day. I would like to thank my parents, **MR. RAYAPURAJU RAVI KUMAR RAO, RAYAPURAJU SAVITHA** and my brother **MR RAYAPURAJU ANUNAI** whose countless sacrifices and blessings have made me who I am today, thank you for always being with me and giving me strength at every step of my life. My sincere gratitude to my Grandparents **MS KALAKUNTLA KARUNA DEVI, RAYAPURAJU SAVITRI DEVI, RAYAPURAJU VENKAT RAO,** my rest extended family **MR MADHU MOHAN RAO R, SHAILAJA R, HANUMANTH RAO R, YASHASRI R, AKHILA R, ANANYA R, AMITHA R, ASHRITHA R, MR VENU K, MRS KAVITHA K, MRS MADHURI K, MR RAGHURAM K, MRS SHAILAJA B** and my dear friends **DR. SUMANA T, DR PRAVALLIKA N, MR DHEIRAJ M, MR. SAGAR SDV, DR MANEESH, MS AISHWARYA S, MRS JAHNAVI B, DR. KASHAIFA MAJEED, DR**. **SHOBHET SAXENA, DR RAHUL THAPA** for their encouragement, support, patience and understanding.

Last but not the least, I thank all my patients involved in this study, without whose cooperation, this dissertation would have never materialized.

I sincerely thank my institute Sri Devaraj Urs Medical College, Tamaka, Kolar for giving me a wonderful foundation and forum of knowledge in the field of Ophthalmology, which will stand with me for the rest of my life.

DATE: **SIGNATURE OF CANDITATE**

PLACE: KOLAR. **DR ANUNITHA RAYAPURAJU**

LIST OF ABBREVATIONS

Abbreviation	Full Form
BP	Blood Pressure
DR	Diabetic Retinopathy
DM	Diabetes Mellitus
ECG	Electrocardiogram
FBS	Fasting Blood Sugar
HbA1c	Hemoglobin A1c
LA	Left Atrial
LVDD	Left Ventricular End-Diastolic Diameter
LVEF	Left Ventricular Ejection Fraction
LVDS	Left Ventricular Systolic Diameter
NPDR	Non-Proliferative Diabetic Retinopathy
OHA	Oral Hypoglycemic Agents
PDR	Proliferative Diabetic Retinopathy
PPBS	Post-Prandial Blood Sugar
QoL	Quality of Life
Rx	Prescription/Treatment
SD	Standard Deviation
S. Creatinine	Serum Creatinine
T2DM	Type II Diabetes Mellitus
UCG	Ultracardiography

LIST OF TABLES

Sr. No.	Name of Tables	Page no.
01	Distribution of NPDR Severity by Age Group	54
02	Distribution of NPDR Severity by Gender	55
03	Comparison of Blood Pressure, Glycemic Control, and Renal Function Parameters Across NPDR Severity Groups	56
04	Comparison of Echocardiographic Parameters Across NPDR Severity Groups	58
05	Demographic Characteristics	59
06	Clinical and Biochemical Parameters	61
07	Cardiac Remodeling Parameters	63
08	ECG Changes and Quality of Life	64
09	Treatment Modalities	66
10	NPDR Subgroup Analysis (Mild, Moderate, Severe)	67
11	Association of Diabetes Duration (DM <10 vs. DM >10 Years)	70

LIST OF FIGURES

Sr. No.	Name of Figures	Page no.
1.	Bar graph representation of Distribution of NPDR severity by age group	54
2.	Pie chart representing Distribution of NPDR Severity by Gender	55
3.	Graph showing the comparison of Blood Pressure, Glycemic Control, and Renal Function Parameters Across NPDR Severity Groups	57
4.	Bar graph representing Comparison of Echocardiographic Parameters Across NPDR Severity Groups	58
5.	Bar graph representing demographic parameters- Age distribution	60
6.	Bar graph representing demographic parameters- Gender distribution	60
7.	Graph showing the Clinical and Biochemical Parameters	62
8.	Graphical representation of cardiac remodeling parameters	63
9.	Bar graph representation of ECG abnormalities among patients with and without DR	64
10.	Graphical representation of quality of life among among patients with and without DR	65
11.	Bar graph showing distribution of treatment modalities among patients with and without DR	66
12.	Bar graph of Mean age distribution among patients with Mild, Moderate, and Severe Non-Proliferative Diabetic Retinopathy (NPDR).	68
13.	Comparison of Gender Distribution, ECG Abnormalities, and Quality of Life among Patients with Mild, Moderate, and Severe Non-Proliferative Diabetic Retinopathy (NPDR).	68

14.	Comparison of Systolic Blood Pressure, HbA1c Levels, and Left Ventricular Ejection Fraction (LVEF) among Patients with Mild, Moderate, and Severe Non-Proliferative Diabetic Retinopathy (NPDR).	69
15.	Distribution of Retinopathy Severity Based on Duration of Diabetes Mellitus (DM).	71
16.	Cardiac Remodeling Parameters in Patients with Diabetes Mellitus (DM) Based on Duration	71
17.	Serum Creatinine Levels in Diabetic Patients Based on Duration of Diabetes	72
18.	Insulin Usage in Diabetic Patients by Duration of Diabetes	72

LIST OF IMAGES

Sr. No.	Name of Images	Page no.
1.	Fundus photograph of a study patient with Moderate Non-Proliferative	06
2.	Fundus photograph of a study patient with High Risk Proliferative	06
3.	Showing the common risk factors and pathophysiological mechanisms of DR and CVD, the new progress of diagnostic techniques for DR, and the biomarkers for early screening of DR.	08
4.	Comparative image of a patient without DR changes and a patient with DR changes	17
5.	Image showing fundus photographs of different grades of DR	19
6.	The role of oxidative stress on DR And Cardio Vascular Disease	29
7.	Pathophysiology mechanism that leads to dialated cardiomyopathy	31
8.	An example of echocardiographic analysis combining all three views is generated with reduced global longitudinal strain of −12.5% and reduced LVEF (47.6%).	33
9.	Fundus photograph of a study patient with normal fundus study	121
10.	ECG of the same study patient showing normal ECG	121
11.	Echocardiography of the same study patient with Normal LVDD and EF.	121

12.	Fundus photograph of a study patient with Mild NPDR	122
13.	ECG of the same study patient showing Low T wave abnormality	122
14.	Echocardiography of the same study patient with reduced LVDD and 34 %EF.	122
15.	Fundus photograph of a study patient with Moderate NPDR	123
16.	ECG of the same study patient showing left bundle branch block	123
17.	Echocardiography of the same study patient with LVH and 27% EF	123
18.	Fundus photograph of a study patient with Severe NPDR	124
19.	ECG of the same study patient showing left ventricular hypertrophy- abnormal ECG	124
20.	Echocardiography of the same study patient with Left Ventricular Hypertrophy and 20% EF	124
21.	Fundus photograph of a study patient with Proliferative Diabetic Retinopathy	125
22.	ECG of the same study patient showing left ventricular hypertrophy- abnormal ECG	125
23.	Echocardiography of the same study patient with Left Ventricular Hypertrophy and 20% EF	125
24.	Slit lamp examination	126
25.	90D examination	126
26.	IDO examination	127

27.	ECHOCARDIOGRAPHY PERFORMED ON A PATIENT	127
28.	TOPCON FUNDUS CAMERA	128

ABSTRACT

Type II diabetes mellitus (T2DM) is a global health concern associated with severe microvascular and macrovascular complications, including diabetic retinopathy (DR) and myocardial remodeling. These conditions significantly impact patients' quality of life (QoL) due to visual impairment, cardiovascular dysfunction, and psychological distress. Emerging evidence suggests shared pathophysiological pathways between DR and cardiac remodeling, yet their direct relationship and combined effect on QoL remain underexplored. This study investigates the correlation between DR severity, cardiac structural changes, and QoL in T2DM patients to inform integrated care strategies.

Materials and Methods:

A cross-sectional observational study was conducted at R. L. Jalappa Hospital & Research Centre, Kolar, involving 97 T2DM patients over 18 months. Participants, diagnosed with T2DM for over five years and without prior cardiac issues, underwent clinical assessments including visual acuity testing (Snellen chart), slit lamp biomicroscopy, posterior segment evaluation (indirect ophthalmoscopy and +90D biomicroscopy), and echocardiography. DR was classified using the Early Treatment Diabetic Retinopathy Study (ETDRS) criteria. QoL was assessed via self-reported questionnaires. Data were analyzed using SPSS software, employing descriptive statistics and logistic regression to explore associations, with a significance threshold of $p<0.05$.

Results:

DR severity correlated with progressive myocardial remodeling, evidenced by rise in left ventricular end-diastolic and systolic diameters and reduced left ventricular ejection fraction (LVEF). ECG abnormalities were significantly higher in proliferative DR (90%) compared to normal fundus (30%), indicating elevated cardiovascular risk. Patients with severe DR showed higher systolic blood pressure, poorer glycemic control (elevated HbA1c), and increased nephropathy markers (serum creatinine). A shift from oral hypoglycemic agents to insulin was observed with advancing DR. QoL declined markedly with DR progression, driven by visual loss, cardiac symptoms, and psychological burden. Longer diabetes duration (>10 years) was associated with more severe DR, cardiac remodeling, and renal impairment.

Conclusion:

The study establishes a significant link between DR progression, myocardial remodeling, and reduced QoL in T2DM patients. Severe DR serves as a marker of cardiovascular risk, necessitating integrated ophthalmic and cardiac monitoring. Early detection, aggressive glycemic management, and multidisciplinary care involving endocrinologists, cardiologists, and ophthalmologists are crucial to mitigate complications and enhance patient outcomes. Comprehensive support addressing physical and psychological needs is essential to improve QoL.

Keywords:

Type II Diabetes Mellitus, Diabetic Retinopathy, Myocardial Remodeling, Quality of Life, Cardiovascular Risk, Multidisciplinary Care

TABLE OF CONTENTS

SL.NO	PARTICULARS	PAGE NO:
1	INTRODUCTION	01
2	AIMS AND OBJECTIVES	10
3	REVIEW OF LITERATURE	12
4	MATERIALS AND METHODS	48
5	RESULTS	53
6	DISCUSSION	73
7	CONCLUSION	80
8	SUMMARY	82
9	BIBLIOGRAPHY	84
10	ANNEXURES	96
11	MASTERCHART	129

INTRODUCTION

CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE II DIABETES MELLITUS PATIENTS

INTRODUCTION

Diabetes mellitus is a form of chronic metabolic disorder characterized by hyperglycemia due to the defect in insulin secretion, or action, or both. Over 90% of all cases of diabetes globally are type II diabetes mellitus (T2DM), the most prevalent form of the disease. Those afflicted with this condition are usually thought of as having obesity, physically inactive, or genetically predisposed. Insulin resistance and pancreatic beta cell dysfunction emerge as this disease advances. After a while, the multitude of microvascular and macrovascular complications due to chronic hyperglycemia woefully deteriorate the patient's health and quality of life.[1] Among all these complications, diabetic retinopathy (DR) and cardiac remodeling are most striking owing to their extreme complications and rising incidence among patients suffering with type 2 diabetes.

Diabetic retinopathy is easy to prevent, yet it remains one of the most well-known vision hazards today. It occurs due to chronic hyperglycemia which increases the permeability of blood vessels in the retina, disrupts microvascular units, inflates the vasculature, and damages endothelial cells. Diabetic retinopathy (DR) is classified as proliferative after non-proliferative diabetic retinopathy (PDR) where microaneurysms and haemorrhages take place while neovascularization occurs at the later stage leading to vision loss from retinal detachment or vitreous hemorrhage.

Additionally, Diabetic macular oedema (DME) is a major complication that causes loss of central vision. Microvascular injury is one of the consequences of DR pathophysiology, which features oxidative stress, inflammation, and RAAS activation. Many studies have indicated a strong relationship between DR and cardiovascular disorders, suggesting an underlying common pathogenic link which connects the complications of diabetes.[2]

Myocardial remodelling describes heart structural and functional changes due to long-term everyday abnormalities like Ischaemia, diabetes, and hypertension. Myocardial fibrosis, left ventricle hypertrophy, and decreased diastolic function in type 2 diabetes are consequences of prolonged hyperglycemia, insulin resistance, and systemic inflammation. Diabetic cardiomyopathy describes the heart's failure associated with diabetes while excluding hypertension or coronary artery disease which is a recognized clinical entity. Heart failure either with preserved or reduced ejection fraction develops from early diastolic dysfunction and subsequently from systolic failure. In DR patients, microvascular damage may suggest more advanced processes and possible cardiac remodelling and heart failure. The presence of DR in people with type 2 diabetes can be an indicator of underlying cardiovascular problems.[3]

Clinical research is paying increasing attention to the association of diabetic retinopathy with cardiac remodelling. Several population-based studies indicate that patients with diabetic retinopathy (DR) have a greater propensity towards heart failure and cardiovascular mortality. Common pathophysiological consequences due to persistent hyperglycemia include oxidative stress, inflammation, endothelial injury, and the body’s accumulation of advanced glycation end-products (AGE). In addition, fibrosis and vascular dysfunction along with adverse cardiac remodelling result from the activation of the renin angiotensin aldosterone system (RAAS) which is important for retinal and myocardial remodelling. These findings warrant enhanced cardiovascular evaluation in T2DM patients with DR.

Along with physiological changes, diabetic retinopathy and cardiac remodeling greatly impact quality of life (QoL) in people suffering from type 2 diabetes. The visual limitation caused by DR severely hampers the performance of daily tasks, mobility, and self-sufficiency, thereby resulting in psychological strain and reduced social participation. Patients suffering from heart failure and myocardial dysfunction are also impacted, as these conditions commonly lead to fatigue, shortness of breath, and low stamina for physical activities. Measurements of health-related quality of life (HRQoL) indicate that these T2DM patients with DR and heart remodeling bear significantly greater burdens of deficient physical, emotional, and mental health. Such patients are more susceptible to anxiety and depression, which underscores the importance of comprehensive treatment strategies that address the clinical and psychosocial facets of diabetes care.

Diabetic retinopathy, cardiac remodeling, and reduced QoL collectively pose a significant burden in type 2 diabetes patients, so recognizing these interrelations is important for proactive risk evaluation and intervention. Developing specific biomarkers and imaging techniques aimed at identifying early-stage myocardial changes in patients with DR may enhance cardiovascular management. Integrated care involving cardiology, endocrinology, and ophthalmology…

Rationale of the Study

The most common form of diabetes mellitus is type II diabetes (T2DM), which is a major health concern globally. T2DM is associated with a broad range of microvascular complications such as diabetic retinopathy (DR) and macrovascular complications like cardiac remodelling and heart failure, which immensely increase the burden of diabetes and adversely affect overall life satisfaction due to elevated morbidity and mortality.[1] is an emerging area of research as it is still understudied but believed that microvascular and macrovascular complications share similar pathophysiological characteristics. How these factors interrelate could potentially shift the paradigm of T2DM patient management, from reactive to proactive through early risk evaluation and assessment, and slowing disease progression. These relationships are crucial to understanding how patients with type 2 diabetes, cardiac remodelling, and DR—as well as their shared impact on QoL—interact.

Diabetic Retinopathy and Systemic Vascular Dysfunction

Diabetic retinopathy remains one of the primary causes for loss of vision globally and is considered a microvascular complication of diabetes. Prolonged hyperglycemia is thought to induce DR due to its impact on the renin-angiotensin-aldosterone system (RAAS), endothelial dysfunction, oxidative stress, and chronic low grade inflammation.These mechanisms cause pathological angiogenesis, elevated vascular permeability, and retinal microvascular injury, all of which contribute to progressive vision impairment. As DR progresses through many stages from non-proliferative diabetic retinopathy (NPDR) to proliferative diabetic retinopathy (PDR), it can also lead to diabetic macular oedema (DME), which exacerbates vision loss. [3]

Although DR primarily affects the retina, its significance extends beyond ocular pathology. Numerous studies have demonstrated that diabetic microvascular dysfunction is not confined to the eyes but reflects generalized endothelial impairment, which predisposes patients to other systemic complications, including cardiovascular disease (CVD). The existence of DR has been found to be a predictor of negative cardiovascular outcomes, such as heart failure, myocardial infarction, and cardiac remodelling. The retina is thought of as a "window" to the systemic vasculature. The molecular relationship between DR and myocardial remodelling in individuals with type 2 diabetes is still not well understood, despite this increasing awareness. Examining this connection may assist improve risk assessment techniques in clinical practice and offer important insights into the common pathophysiological pathways driving diabetes complications.

IMAGE 1: Fundus photograph of a study patient with Moderate Non-Proliferative

IMAGE 2 : Fundus photograph of a study patient with High Risk Proliferative

Myocardial Remodeling in Type II Diabetes Mellitus

One of the main causes of morbidity and death in people with type 2 diabetes is cardiovascular problems. Lack of hypertension and coronary artery disease marks diabetic cardiomyopathy as a clinical condition defining changes in the physiological and anatomical structure of the heart. Type II diabetes subjects are primarily affected by inflammation, oxidative stress, insulin resistance, persistent hyperglycemia, and dyslipidemia as contributors of cardiac remodeling. This leads to myocardial fibrosis, left ventricular hypertrophy, and impaired diastolic function. Eventually, these changes culminate into heart failure and increase systolic dysfunction which significantly heightens adverse cardiovascular events and reduces life span.

Some primary mechanisms driving diabetic myocardial remodeling include Endothelial dysfunction, which predominantly cause increased vascular stiffness, microvascular rarefaction, along with a drop in nitric oxide bioavailability. Interestingly, these same mechanisms are also key for DR, suggesting that impaired retinal microvasculature might indicate concurrent structural changes in the heart. Recently, more studies have associated DR with subclinical left ventricular diastolic dysfunction (LVDD), myocardial fibrosis, and altered left ventricular geometry, which demonstrates the growing understanding of these results.

The Interconnection Between Diabetic Retinopathy and Myocardial Remodeling

According to a number of studies, DR may be a reliable indicator of cardiovascular illness, such as myocardial infarction and heart failure. DR and cardiac remodelling share pathophysiological pathways, such as oxidative stress caused by hyperglycemia, chronic inflammation, endothelial dysfunction, AGE activation, and renin-angiotensin-aldosterone system (RAAS) upregulation. These variables contribute to both retinal vascular damage and heart structural alterations, bolstering the concept that DR might be an early indication of cardiac dysfunction in T2DM patients.

Despite these associations, there is a lack of comprehensive studies directly investigating the correlation between DR severity and myocardial remodeling using advanced imaging modalities such as echocardiography, cardiac MRI, or speckle-tracking echocardiography. Knowing this association may help identify high-risk individuals early and enable prompt therapies to stop the development of diabetes-related cardiovascular and ocular problems.

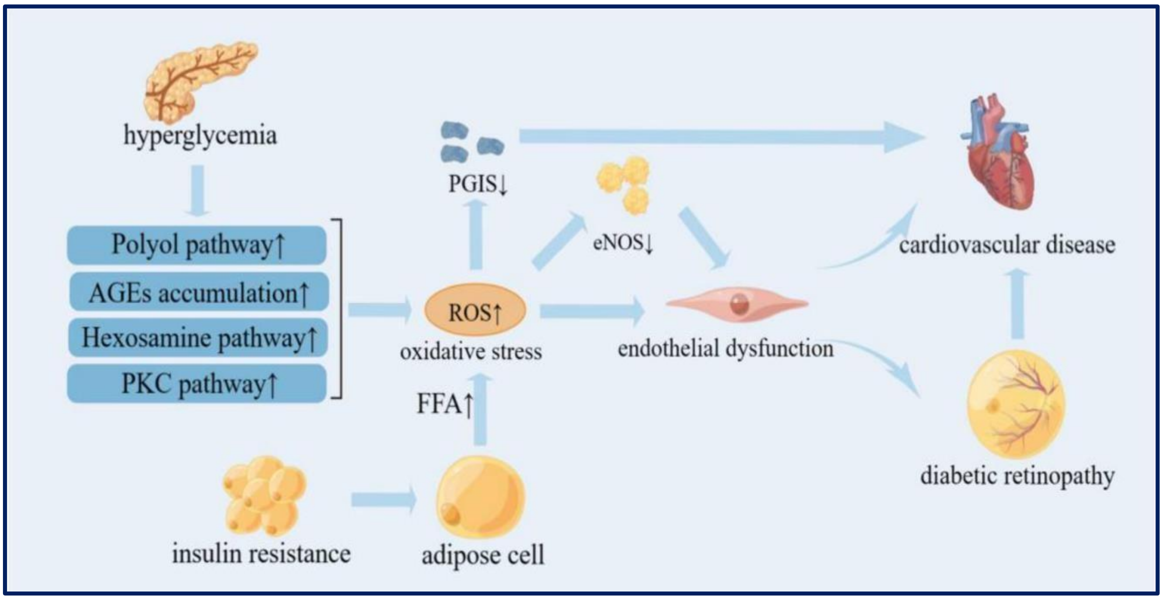

IMAGE 3: Showing the common risk factors and pathophysiological mechanisms of DR and CVD, the new progress of diagnostic techniques for DR, and the biomarkers for early screening of DR. [84]

Impact on Quality of Life in T2DM Patients

Beyond its physiological effects, DR, cardiac remodelling, and diabetes-related comorbidities interact to greatly impact T2DM patients' quality of life (QoL). Visual impairment due to DR can lead to substantial limitations in daily activities, mobility, independence, and employment, thereby increasing the risk of social isolation, depression, and anxiety. Similarly, myocardial remodeling and associated cardiac dysfunction contribute to exercise intolerance, fatigue, dyspnea, and reduced functional capacity, further diminishing overall well-being.

Physical, mental, and social well-being ratings in T2DM patients with DR and cardiovascular problems are consistently lower than those in patients without these issues, according to health-related quality of life (HRQoL) measures. Furthermore, having both cardiac dysfunction and visual impairment can have a psychological toll that worsens stress, impairs glycemic control, and makes it harder to follow diabetes treatment plans. In order to overcome these obstacles, a multidisciplinary, patient-centered strategy is needed, one that emphasizes psychological support, rehabilitation, and lifestyle changes in addition to disease management in order to enhance the general quality of life for those who are impacted.

Need for This Study

Given that diabetes and its complications are becoming more prevalent globally, there is an urgent need to improve risk prediction, early diagnosis, and comprehensive management strategies.

Despite having significant clinical practice implications, the relationship between DR, myocardial remodeling, and QoL in T2DM patients is still understudied. This study is designed to address this gap by systematically investigating:

1. The relationship between DR severity and myocardial remodeling using echocardiographic and clinical assessments.
2. How these issues affect quality of life and shed light on the wider effects of end-organ damage linked to diabetes
3. The potential role of DR as a predictive marker for cardiovascular risk stratification, aiding in the early detection of diabetic cardiomyopathy.

By highlighting these connections, the study's findings may help develop integrated screening and care plans for individuals with type 2 diabetes and promote cooperation among endocrinologists, cardiologists, and ophthalmologists.

Furthermore, the study could provide evidence for the implementation of personalized treatment approaches that address both the medical and psychosocial challenges faced by patients with diabetes complications.

AIMS AND OBJECTIVES:

AIMS AND OBJECTIVES:

AIM OF THE STUDY:

To correlate diabetic retinopathy severity with myocardial remodeling and quality of life in type 2 diabetes mellitus patients.

OBJECTVES OF THE STUDY:

1. To analyse the association of different grades of diabetic retinopathy with cardiac remodelling in terms of 2D ECHO and ECG in type 2 Diabetes mellitus.

2. To assess the association of grades of retinopathy with other diabetic target organ damage (Diabetic nephropathy) and quality of life by Visual Functioning Questionnaire – 25 (VFQ-25).

REVIEW OF LITERATURE

REVIEW OF LITERATURE

I. Diabetes Mellitus

Persistent hyperglycemia is a hallmark of diabetes mellitus (DM), a chronic metabolic disease caused by abnormalities in either insulin action or production, or both. The majority of DM patients are Type II Diabetes Mellitus (T2DM), which is characterised by insulin resistance and a developing insulin secretory malfunction.[4] Type 2 diabetes is becoming more prevalent worldwide, and because it is a chronic condition with many associated complications, it has significant socioeconomic effects. Microvascular (diabetic retinopathy, nephropathy, and neuropathy) or macrovascular (coronary artery disease, peripheral arterial disease, and cerebrovascular disease) events are commonly used to describe these problems, which significantly impair quality of life and increase morbidity and mortality among those affected. Due to the complex interactions between genetic, environmental, and behavioral factors in its pathogenesis, type 2 diabetes can present in a variety of ways and progress over time. [5]

A. Type II diabetes mellitus definition and epidemiology:

The characteristic feature of Type II Diabetes Mellitus (T2DM), a common metabolic disease, is chronic hyperglycemia, which is brought on by insulin resistance and an insufficient compensatory insulin secretory response. A combination of medication and lifestyle management is required to control blood glucose levels because this form of diabetes is progressive in nature. In type 2 diabetes, there is usually an insulin resistance, which is the inability of the body cells to respond appropriately to insulin.[6] In contrast, Type I diabetes mellitus is identified by deficiency of insulin due to the loss of pancreatic beta cells, with little or no beta-cell functioning. The pancreatic beta cells may also progressively lose their capacity to generate enough insulin as the illness worsens.[7]

Epidemiology indicates that type 2 diabetes is a severe global public health concern. While more common in those over 45, increasing sedentary behavior and obesity rates are leading to younger and younger diagnoses, including children and adolescents.

As stated by the International Diabetes Federation (IDF), approximately 463 million individuals aged between 20 and 79 years old had diabetes in 2019. This figure is projected to increase to 700 million by 2045.A combination of several factors like age, lifestyle, socioeconomic standing, ethnicity, and family history may contribute to the risk of developing Type 2 diabetes. As of 2020, the CDC reports that more than 34 million Americans are afflicted with this condition, constituting about 90-95% of the adult population living with the disease.[8]

The number of individuals with type 2 diabetes worldwide differs by region and ethnicity. Higher prevalence communities are situated within the Western Pacific, Middle Eastern, and North African regions; this was attributed to lifestyle choices and underlying genetics endemic to these regions. Increased cases of type 2 diabetes have also been linked to urban and industrial development, suggesting that a combination of genetic factors and environmental exposure drives this phenomenon.

Type 2 diabetes is highly associated with pathological changes in almost all of the body's organ systems. Complications such as neuropathy, nephropathy, and retinopathy can progress to limb amputation, renal failure, and vision loss respectively. Other macrovascular interests like peripheral artery disease, coronary artery disease, and cerebrovascular disease increase the risk of mortality. These complications require a comprehensive regimen of glycaemic control, lifestyle adjustment, and regular monitoring of blood sugar levels. [9]

B. Diabetes pathophysiology and systemic complications:

The pathophysiology of Type II Diabetes Mellitus (T2DM) involves a complex interplay of genetic, environmental, and metabolic factors leading to insulin hypofunction and beta-cell failure. Insulin resistance targets adipose tissue, muscle, and the liver first. An underactive insulin response to glucose results in uncontrolled hepatic glucose output and also inhibits glucose uptake by muscle and adipose. Subsequent loss of beta-cells leads to relative insulin deficiency as the autoimmune destruction of insulin-producing beta-cells increases in response to unmet demand. This complicated pathophysiology contributes significantly to the development of hyperglycemia, the hallmark of diabetes, through a variety of biochemical pathways. This, in turn, contributes significantly to the onset of other systemic issues.[10]

Hyperglycemia primarily damages tissue through increased flow of the polyol pathway, increased formation of advanced glycation end product (AGE), activation of protein kinase C (PKC) isoforms, and hyperactivity of the hexosamine pathway. Together, these pathways play a significant role in the microvascular and macrovascular issues that characterise diabetes, which are exacerbated by persistent hyperglycemia.[11]

Microvascular Complications: The three primary ones are diabetic retinopathy, neuropathy, and nephropathy. Damage to the retina's small blood vessels produces diabetic retinopathy, one of the most common causes of adult blindness. Endothelial cell failure and vascular basement membrane thickening brought on by prolonged hyperglycemia lead to hypoxia, vascular leakage, and eventually neovascularisation. Nephropathy, another serious side effect that can lead to end-stage renal failure and proteinuria, is caused by similar vascular damage to the kidneys. High blood sugar destroys nerve fibres throughout the body, causing pain, loss of feeling, and organ malfunction. This condition is known as neuropathy, which is characterised by the loss of nerve function.[12]

Macrovascular Complications: These are brought on by the effects of persistently high glucose levels on larger blood arteries, which worsen atherosclerosis. People with type 2 diabetes have an increased risk of developing coronary artery disease, peripheral arterial disease, and cerebrovascular disease. The way hyperglycemia interacts with other risk factors including dyslipidaemia, hypertension, and inflammatory markers—all of which accelerate the formation of atheroma—links diabetes and these disorders pathophysiologically.

The quality of life for people with type 2 diabetes is significantly impacted by the systemic effects of these problems. A significant contributor to disability and increased mortality rates is chronic hyperglycemia and its aftereffects. Additionally, they typically require significant medical care and cause considerable morbidity. These problems impair a patient's mobility, vision, and general health, which significantly reduces quality of life in addition to reducing life expectancy. [13]

II. Overview of Diabetic Retinopathy

Diabetic retinopathy (DR), one of the most prevalent microvascular consequences of diabetes, is marked by damage to the retinal blood vessels and can, in severe cases, lead to blindness. The length of diabetes and the degree of glycaemic control are directly related to its onset and progression; a longer duration and inadequate glycaemic control greatly raise the risk. Microaneurysms and retinal capillary leakage are the first stages of DR pathogenesis. More severe forms of the disease include vitreous haemorrhage and the growth of new blood vessels on the retina. [2]

Clinically, there are two forms of DR: non-proliferative diabetic retinopathy (NPDR), which is the early stage, and proliferative diabetic retinopathy (PDR), which is more advanced and involves neovascularisation. Among the management strategies for DR are lipid-lowering drugs, hypertension control, and glycaemic control optimisation. Certain treatments, such as intravitreal injections of anti-VEGF medications, laser photocoagulation, and vitreoretinal surgery, are employed in more severe instances to prevent vision loss. [14]

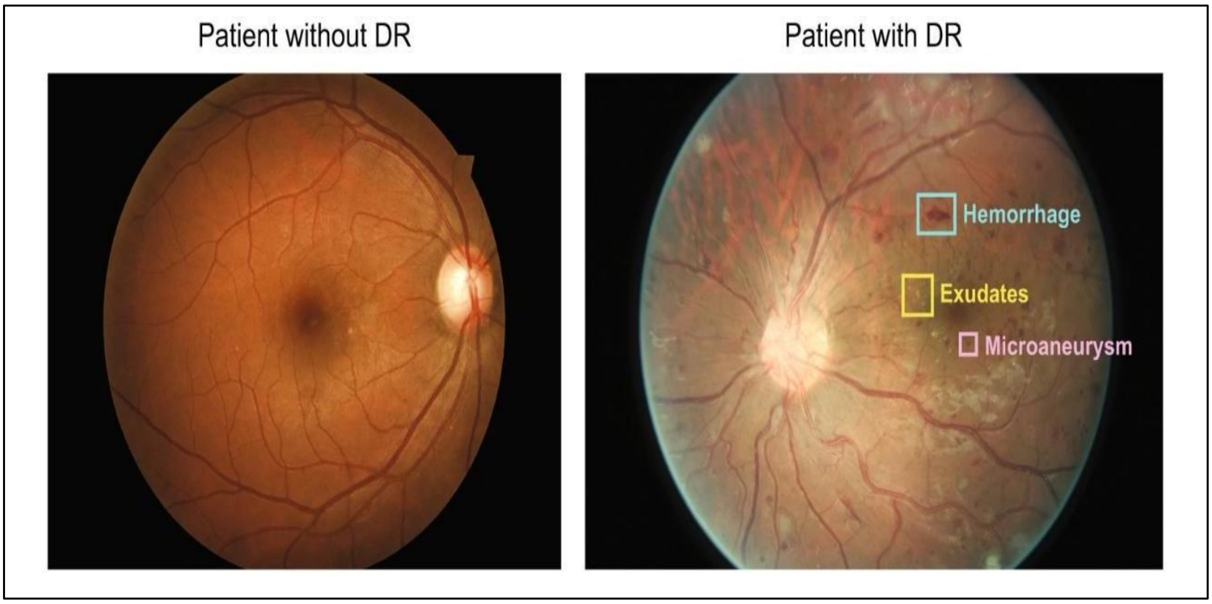

IMAGE 4 : Comparative image of a patient without DR changes and a patient with DR changes [85]

A. Definition and classification of diabetic retinopathy

At first, diabetic retinopathy may only result in minor visual issues or no symptoms at all. But blindness may eventually result from it. Diabetes may strike anyone with type 1 or type 2 diabetes. If you have diabetes for a long time and your blood sugar is not well managed, you are more prone to develop this eye condition.

Diabetic Retinopathy Classification: Proliferative diabetic retinopathy (PDR) and non-proliferative diabetic retinopathy (NPDR) are the two primary types of diabetic retinopathy, which are distinguished by the severity and pathological features observed in the retina. [15]

Non-Proliferative Diabetic Retinopathy (NPDR): Microaneurysms, retinal oedema, and dot haemorrhages are characteristics of background retinopathy (NPDR), the early stage of diabetic eye disease. In NPDR, the retina's blood vessel walls deteriorate, and minute bulges known as microaneurysms emerge from the vessel walls of the smaller capillaries, sometimes causing blood and fluid to seep into the retina. NPDR is categorised as mild, moderate, or severe based on the existence and degree of these retinal abnormalities.

Proliferative Diabetic Retinopathy (PDR): PDR is the most severe form of the illness. At this stage, oxygen cannot reach the retina due to circulation problems. The retina and the vitreous gel that fills the eye may develop new, fragile blood vessels as a result. Blood leaking from these arteries can cause blindness or severe visual loss. PDR is indicated by the formation of new blood vessels on the retina and posterior surface of the vitreous. Because of their propensity for bleeding, these new blood vessels can cause substantial visual impairment by clouding the vitreous and occasionally causing a retinal detachment by traction. [16]

Diagnosis and Staging: Diagnosis of diabetic retinopathy is performed primarily through fundoscopic examination by an ophthalmologist using dilation to better view the retina. Additional diagnostic tools include fluorescein angiography and optical coherence tomography (OCT) to assess the extent of retinopathy and to identify the specific type of retinal changes present. Classification and staging of diabetic retinopathy are essential for determining the appropriate treatment strategy and for evaluating the risk of progression to more advanced stages that could result in vision loss.[17]

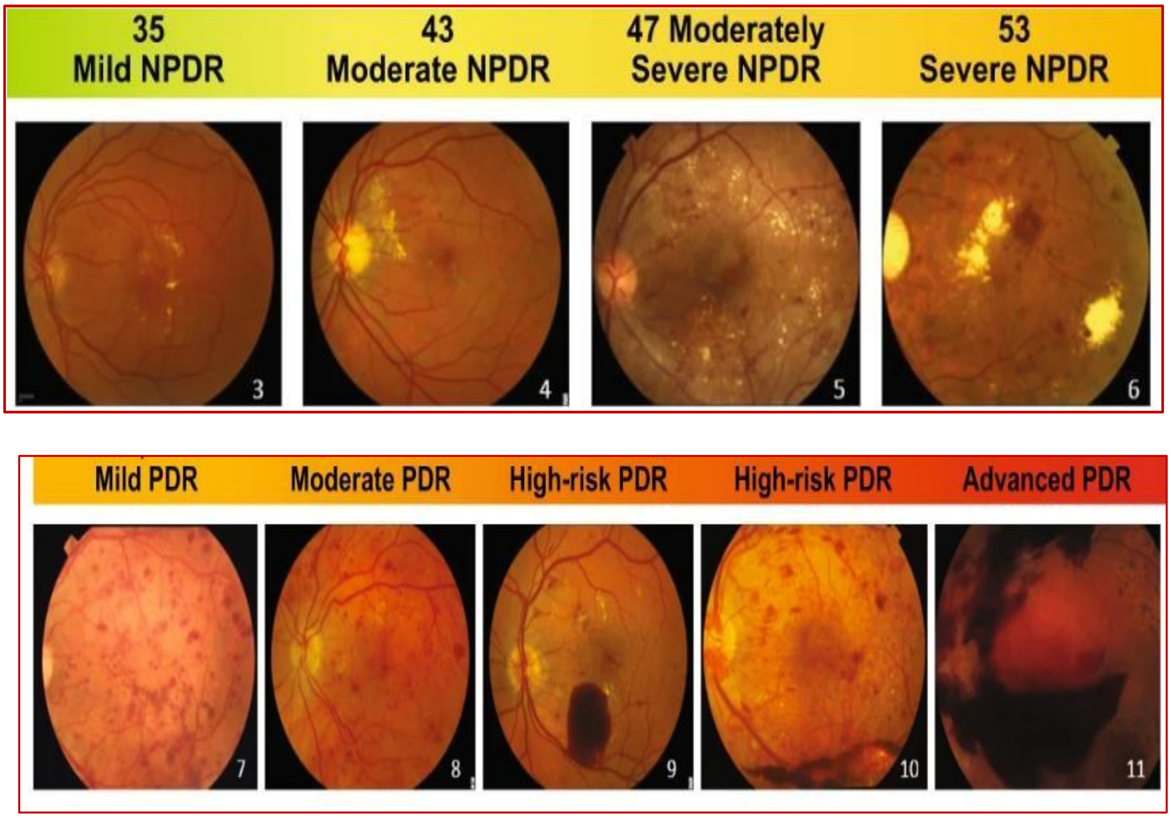

IMAGE 5: Image showing fundus photographs of different grades of DR [85]

Management and Treatment: Diabetes must be treated with diet, exercise, and blood sugar-lowering medications in order to prevent or delay the development of diabetic retinopathy. For those who develop either NPDR or PDR, treatments can range from medical monitoring without intervention in mild cases to more aggressive treatments in more advanced cases. Laser surgery, injection of corticosteroids or anti-VEGF agents directly into the eye, and vitrectomy are common treatments designed to reduce the risk of blindness.

Implications for Patients: Patients are more likely to experience severe vision loss during the crucial transition from NPDR to PDR. Regular eye exams are vital for timely detecting diabetic retinopathy in patients with diabetes. Patients with this condition can greatly improve their quality of life by preventing visual impairment and optimizing results through early detection and treatment. [18]

B. Pathophysiology and Course of Diabetic Retinopathy:

Diabetic retinopathy (DR) exhibits vascular and neuronal degeneration due to the combined effect of diabetes, hypertension, inflammation, and genetic predisposition. Sustained hyperglycemia is responsible for causing and amplifying many pathophysiological changes that contribute to diabetic retinopathy (DR). Diabetic retinopathy involves a myriad of pathological changes which include oxidative stress, increased vascular endothelial growth factor (VEGF), the presence of advanced glycation end products (AGEs), and activation of protein kinase C (PKC) pathways.[2]

1. Early Pathogenic Changes: At the microscopic level, AGEs' formation is triggered by the non-enzymatic glycation of proteins and lipids due to chronic hyperglycemia. These AGEs modify the structural and functional attributes of various tissues including the retina and the arterial walls. They encourage inflammation and the coagulation process resulting in thickening of the capillary basement membrane, which is one of the earliest histological changes in DR. Furthermore, by increasing vascular permeability and encouraging the release of pro-inflammatory cytokines, AGEs interact with their receptor RAGE on the surface of leukocytes and endothelial cells, initiating a cascade of intracellular signalling events that exacerbate retinal tissue damage.

2. Vascular Endothelial Growth Factor (VEGF) and Neovascularization: VEGF plays a pivotal role in the progression of DR, particularly in its transition from the non-proliferative to the proliferative stage. Under normal conditions, VEGF is essential for angiogenesis and the maintenance of vascular integrity. However, in the context of DR, hypoxic conditions in the retina due to capillary nonperfusion upregulate VEGF expression. Proliferative diabetic retinopathy (PDR) is characterised by the growth of new blood vessels that are fragile and leaky, as well as vitreous haemorrhage and tractional retinal detachment, two significant causes of severe vision loss.[18]

3. Oxidative Stress and Inflammatory Pathways: Yet another important factor in the pathophysiology of DR is oxidative stress. In addition to increasing the generation of free radicals, elevated glucose levels also weaken the antioxidant defence systems in retinal cells. This imbalance exacerbates endothelial dysfunction and increases vascular permeability by causing oxidative damage to cellular lipids, proteins, and nucleic acids. Moreover, lipoprotein accumulation in the retinal arteries due to diabetes-related dyslipidaemia might cause local inflammation. The retinas of diabetic patients have elevated amounts of inflammatory mediators, including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α). The blood-retinal barrier is weakened by these mediators, resulting in macular oedema and the development of fibrovascular structures. [19]

4. Protein Kinase C (PKC) Activation: The PKC pathway, particularly its beta isoform, is activated by diacylglycerol which is synthesized more in hyperglycemic conditions. Activation of PKC-beta has been implicated in several key processes in the pathogenesis of DR, including increased vascular permeability, blood flow changes, angiogenesis, and upregulation of VEGF. PKC-beta pharmacological inhibition has demonstrated promise in mitigating these side effects and delaying the course of DR.

The Severe Stage of Diabetic Retinopathy (DR): The shift from Non-Proliferative Diabetic Retinopathy (NPDR) to Proliferative Diabetic Retinopathy (PDR) signals a significant reduction change increase in disease state. Retinal ischemia worsens over time, leading to the hypersecretion of pro-angiogenic factors such as VEGF, which drives the neovascularization seen in PDR. Newly formed, poorly constructed vessels of PDR increase the risk of vitreous hemorrhage, fibrovascular proliferation, and tractional retinal detachment – a leading cause of blindness in patients with diabetic retinopathy (DR). NPDR can be marked by deterioration in retinal blood flow, formal retinal ischemia, and a rise in oedematous tissue.[20]

C. Risk factors and epidemiology

The epidemiology and risk factors associated with diabetic retinopathy (DR) provide an important framework for understanding the impact of DR on patients with Type II Diabetes Mellitus (T2DM). Since diabetic retinopathy continues to be the leading cause of blindness and visual impairment in working-age individuals globally, it is crucial for both public health and individual patient treatment. It is crucial to identify and understand the risk factors for DR in order to prevent its onset and development, enhance quality of life, and reduce the burden of cardiac remodelling linked to macrovascular and microvascular diabetes effects. [14]

Epidemiology of Diabetic Retinopathy: Approximately one-third of diabetics worldwide, according to recent estimates, have DR symptoms, and of those, about one-third have vision-threatening stages of retinopathy, such as proliferative DR or diabetic macular oedema.

. The prevalence of DR varies widely depending on the population studied, the duration of diabetes, the criteria used to define diabetes, and the methods used to assess retinopathy. According to a comprehensive evaluation of 35 research, the prevalence of DR among people with diabetes was 34.6% worldwide, indicating a substantial health burden. Furthermore, demographic trends such as aging populations and increasing prevalence of diabetes suggest that DR will continue to be a major public health challenge.[16]

Risk Factors for Diabetic Retinopathy:

1. **Duration of Diabetes**: The duration of diabetes is the most significant risk factor for the development of DR. The longer a person has diabetes, the higher their chance of acquiring DR. Studies show that nearly all individuals with Type 1 diabetes and more than 60% of

those with Type 2 diabetes will have some kind of DR 20 years after the onset of their diabetes.

2. **Glycemic Control**: Poor glycaemic control is one of the greatest markers of DR. Higher levels of haemoglobin A1c (HbA1c), a gauge of long-term glucose control, are associated with an increased risk of DR. According to the Diabetes management and Complications Trial (DCCT) and the UK Prospective Diabetes Study (UKPDS), improving glycaemic management can significantly reduce the incidence and progression of diabetes mellitus.

3. **Hypertension:** High blood pressure is another significant risk factor for the development and progression of DR. The transition from NPDR to PDR is accelerated by hypertension, which worsens the retina's blood vessel damage. People with diabetes who have their blood pressure properly managed can lower their chance of getting DR. [21]

4. **Lipid Levels**: Dyslipidaemia, or elevated blood lipid levels, has been linked to a higher incidence of macular oedema and hard exudates in people with DR. Serum lipid levels can be controlled to either lower the risk of DR or delay its development.

5. **Nephropathy**: Diabetic nephropathy appears to be associated with DR; the presence of kidney disease in patients with diabetes increases the risk of developing advanced DR. The shared pathophysiological pathways between nephropathy and retinopathy underscore the systemic nature of diabetes complications.

6. **Genetic Factors**: The vulnerability to DR is influenced by genetic predisposition and family history, albeit the precise genetic components are not entirely understood. A person's risk of having DR may be influenced by a number of genetic loci and polymorphisms that have been discovered.[22]

III. Myocardial Remodeling in Diabetes

A significant cardiac change linked to diabetic cardiomyopathy, a separate condition marked by structural and functional alterations in the myocardium without the presence of hypertension or coronary artery disease, is myocardial remodelling in diabetes. Interstitial fibrosis, myocardial steatosis, and changes in the myocardial microvasculature are among the pathophysiological causes of myocardial remodelling in diabetes.[23] Chronic hyperglycemia is the main cause of these alterations because it increases the deposition of collagen and other extracellular matrix proteins, which increases cardiac stiffness and decreases compliance. Additionally, the buildup of advanced glycation end products (AGEs) and oxidative stress resulting from hyperglycemia damages cardiomyocytes and endothelial cells, further exacerbating cardiac dysfunction. Since these modifications are often associated with diastolic dysfunction and eventually heart failure with preserved ejection fraction (HFpEF), a prevalent and debilitating condition among diabetics, they are medically significant.[24]

A. Definition and mechanisms of myocardial remodeling

In the context of diabetes, these changes are predominantly driven by metabolic disturbances characteristic of the disease, including hyperglycemia, insulin resistance, and the resultant systemic inflammation and oxidative stress. The remodeling process in diabetes is complex, involving alterations in both the myocardial cells (cardiomyocytes) and the extracellular matrix, which together contribute to the functional impairment of the heart.

1. Cellular and Molecular Mechanisms: The primary cellular events in diabetic myocardial remodeling include hypertrophy of the cardiomyocytes, apoptosis, and necrosis. These changes are exacerbated by the diabetic milieu, particularly through the effects of persistent hyperglycemia. High glucose levels lead to an excess of reactive oxygen species (ROS) through mitochondrial dysfunction, resulting in oxidative stress that damages cells' proteins, lipids, and DNA. The mitogen-activated protein kinase (MAPK) pathways are among the signalling pathways that are triggered by oxidative stress and promote cellular growth and death. [25]

Insulin resistance, another hallmark of type II diabetes, contributes to myocardial remodeling by altering lipid metabolism in the heart. Insulin-resistant states increase the delivery of free fatty acids to the cardiomyocytes, enhancing lipid accumulation (lipotoxicity) within these cells, which further deteriorates their function and survival. The excessive fatty acid influx also leads to increased fatty acid oxidation, which can overwhelm the mitochondrial capacity, leading to more ROS production and further oxidative damage.

Advanced glycation end products (AGEs), which are produced when sugars interact non-enzymatically with proteins, lipids, and nucleic acids, accumulate in several organs when chronic hyperglycemia develops. By cross-linking with matrix proteins and changing the myocardium's structural integrity, AGEs in the heart contribute to myocardial stiffness. Additionally, AGEs interact with certain receptors (RAGE) on the surface of cardiac cells, triggering inflammatory processes that support cellular malfunction and fibrosis.[26]

2. Extracellular Matrix Changes: The extracellular matrix (ECM) is essential for preserving the structure and functionality of the heart. The ECM changes significantly in diabetic cardiac remodelling, mostly due to an increase in fibrosis. Excessive collagen and other matrix protein deposition causes fibrosis, which alters the myocardium's architectural structure and results in stiffness and poor ventricular filling—two characteristics that are characteristic of diabetic cardiomyopathy. Transforming growth factor-beta (TGF-β), a cytokine that is increased in the diabetic heart and promotes fibroblast proliferation and extracellular matrix protein deposition, mediates the fibrosis process.[27]

3. Microvascular Dysfunction: Diabetes also affects the coronary microcirculation, leading to microvascular dysfunction, which is characterized by impaired vasodilation, increased microvascular resistance, and reduced coronary flow reserve. This dysfunction exacerbates myocardial ischemia and contributes to the progression of remodeling by limiting nutrient and oxygen supply to the myocardium and by facilitating the accumulation of metabolic by-products that further injure the cardiac tissue.

4. Diastolic Dysfunction: The heart's mechanical characteristics change as the remodelling process goes on, which might result in diastolic dysfunction, which impairs the heart's capacity to fill and relax during diastole. In diabetic individuals, this syndrome frequently occurs before systolic dysfunction and is a common early indicator of heart failure. [25]

B. The impact of diabetes on cardiac structure and function

One significant way that diabetes manifests itself is through its effects on heart structure and function, which can result in particular alterations known as diabetic cardiomyopathy. This syndrome encompasses specific structural and functional myocardiac changes with no other antecedent cardiovascular risk factors like coronary artery disease or hypertension. The pathophysiology of diabetic cardiomyopathy involves myocardial fibrosis, microvascular dysfunction, alteration of myocardial metabolism, and progressive deterioration in cardiac function with an emphasis on diastolic failure. Knowledge of these changes is essential in managing the cardiovascular concerns of patients with diabetes. [28]

1. Myocardial Fibrosis: Among the chronic complications of diabetes, myocardial fibrosis is one of the most serious in relation to the structure of the heart. Diabetic cardiomyopathy causes fibrosis due to imbalance of extracellular matrix proteins production and degradation. High glucose conditions lead to protease-resistant non-enzymatic glycation which forms advanced glycation end products, collars, and cross-links with collagen increasing cardiac tissue stiffness. Besides increasing rigidity of the myocardium, this extensive fibrosis disrupts the architectural design of the heart, systolic and diastolic dysfunction.

2. Microvascular Dysfunction: Diabetes markedly affects the coronary microvasculature, leading to endothelial dysfunction and reduced capillary density, known as microvascular dysfunction. This condition reduces the efficiency of coronary blood flow and impairs myocardial perfusion. Endothelial cells in the microvasculature are damaged by oxidative stress induced by hyperglycemia, which decreases the production of nitric oxide, a critical regulator of vasodilation. Consequently, the diminished vasodilatory response in diabetic patients exacerbates myocardial ischemia, especially during increased cardiac demand, and contributes to the progressive decline in cardiac function.[29]

3. Altered Myocardial Metabolism: In the diabetic heart, there is a metabolic shift from the normal use of glucose towards an increased reliance on fatty acid oxidation for energy. This shift is primarily driven by insulin resistance, which impairs glucose uptake and utilization in cardiomyocytes. The excessive oxidation of fatty acids leads to the accumulation of toxic lipid intermediates (lipotoxicity), which can induce apoptosis and further contribute to myocardial damage. Moreover, the increased fatty acid metabolism enhances oxygen consumption and decreases cardiac efficiency, manifesting as reduced myocardial contractility and systolic dysfunction over time. [30]

4. Diastolic Dysfunction: Diastolic dysfunction, which is typified by the heart's decreased capacity to relax and fill during diastole, is the hallmark of diabetic cardiomyopathy in its early stages. This dysfunction results from the combined effects of myocardial fibrosis, microvascular disease, and altered cellular metabolism. As the disease progresses, the compliance of the ventricular wall decreases due to stiffening from fibrosis and AGEs.

Furthermore, diastolic filling pressures are made worse by reduced cardiomyocyte relaxation, which results in heart failure with preserved ejection fraction (HFpEF) symptoms.

5. Systolic Dysfunction: While diastolic dysfunction is the initial feature of diabetic cardiomyopathy, prolonged disease progression can lead to systolic dysfunction. Systolic dysfunction in diabetes is less common than diastolic dysfunction and typically occurs as a later manifestation. It is primarily due to a decrease in myocardial contractility, which can result from a direct impairment of the contractile machinery of cardiomyocytes, cumulative cellular damage from oxidative stress, and the adverse effects of chronic hyperglycemia on calcium handling within the cells.[31]

C. Link between diabetic complications and cardiac changes

The connection between diabetic complications and cardiac changes is profoundly significant, as diabetes exerts systemic effects that influence a wide range of organs, including the heart. The link is particularly evident in how microvascular and macrovascular complications arising from diabetes predispose patients to various forms of cardiac pathology, most notably diabetic cardiomyopathy and coronary artery disease. This interconnectedness highlights the complexity of diabetes as a systemic disease rather than just a disorder of glucose metabolism, implicating a broad network of pathophysiological pathways that converge on the cardiovascular system.

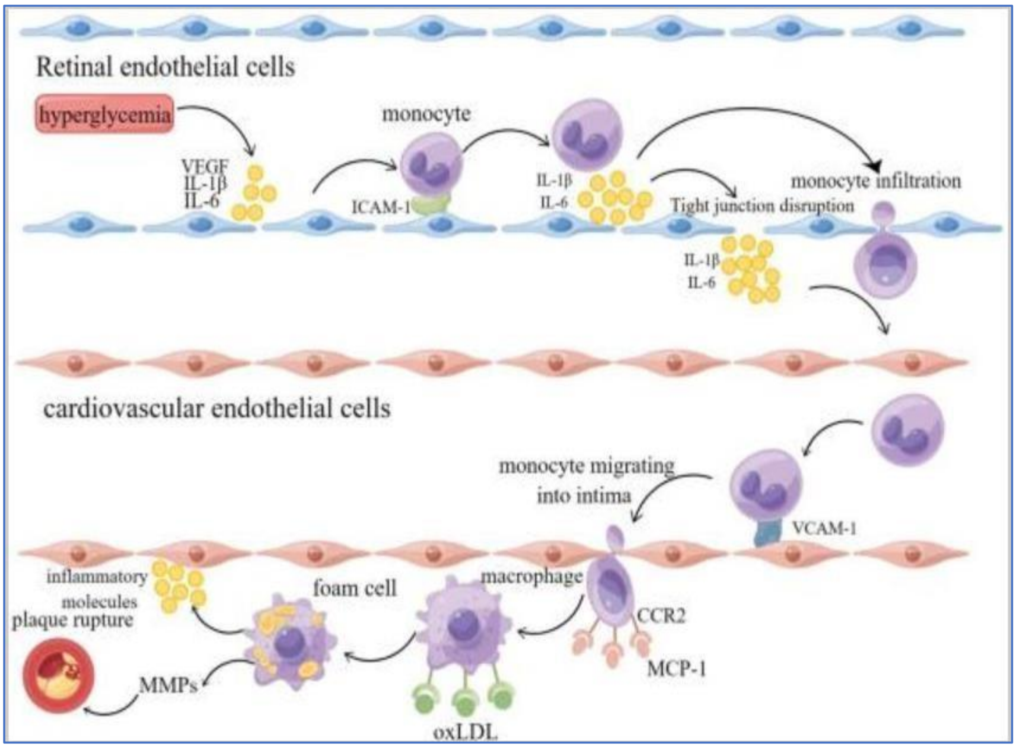

IMAGE 6: The role of oxidative stress on DR And Cardio Vascular Disease [84]

1. Microvascular Complications and Cardiac Changes: diabetes heart changes share pathogenic pathways with diabetes microvascular problems, including retinopathy, neuropathy, and nephropathy. The main cause of these issues, chronic hyperglycemia, sets off a series of metabolic reactions that thicken the vascular basement membrane and increase its permeability. Even in the absence of atherosclerotic coronary artery disease, myocardial ischaemia and fibrosis are caused by comparable microvascular alterations in the heart. Myocardial blood flow is further reduced by endothelial dysfunction, which is typified by poor nitric oxide bioavailability. This exacerbates ischaemia and promotes the development of myocardial remodelling. [32]

2. Macrovascular Complications and Cardiac Changes: The macrovascular complications of diabetes, including atherosclerosis, are precipitated by the interaction of hyperglycemia, dyslipidemia, and hypertension, which are prevalent in diabetic individuals. These circumstances create an environment that makes it easier for atheromatous plaques to grow in the coronary arteries. This may lead to coronary artery disease (CAD), which may subsequently result in myocardial infarction and heart failure. Diabetes makes the atherogenic process worse by promoting inflammatory responses, speeding up the migration and proliferation of vascular smooth muscle cells, and increasing the oxidation of low-density lipoproteins (LDL) inside the arterial wall. Cardiac function is significantly impacted by the ensuing artery stiffness and lumen constriction, which restrict blood flow to the myocardium.[33]

3. Hyperglycemia-Induced Myocardial Damage: Extended exposure to elevated glucose levels results in the production of advanced glycation end products (AGEs) at the cellular level. The structure and function of the cardiac muscle and small arteries are altered by these AGEs.

AGEs cause the myocardium to develop interstitial and perivascular fibrosis, which decreases its flexibility and hinders the heart's ability to fill and contract. Diabetic cardiomyopathy, which is characterised by diastolic dysfunction and, ultimately, systolic failure, is mostly caused by these alterations. [34]

4. Insulin Resistance and Cardiac Function: Another characteristic of type 2 diabetes is insulin resistance, which changes the heart's use of substrates, making cardiac dysfunction worse. Normally, the heart gets its energy from a combination of glucose and fatty acids. However, there is a greater dependence on fatty acids in the context of insulin resistance, which results in a buildup of harmful lipid intermediates and is energetically less efficient. In addition to having a direct impact on cardiac contractility, this metabolic change causes oxidative stress and mitochondrial dysfunction, both of which accelerate the onset of heart failure.

5. Autonomic Neuropathy and Cardiovascular Risk: Diabetic autonomic neuropathy is a common side effect of diabetes that impairs the autonomic control of heart rate and blood pressure. This can lead to arrhythmias, silent myocardial ischaemia, and abrupt cardiac death. Autonomic dysfunction-related lack of normal heart rate variability in diabetics is a direct predictor of mortality and is associated with a higher risk of cardiovascular disease. [35]

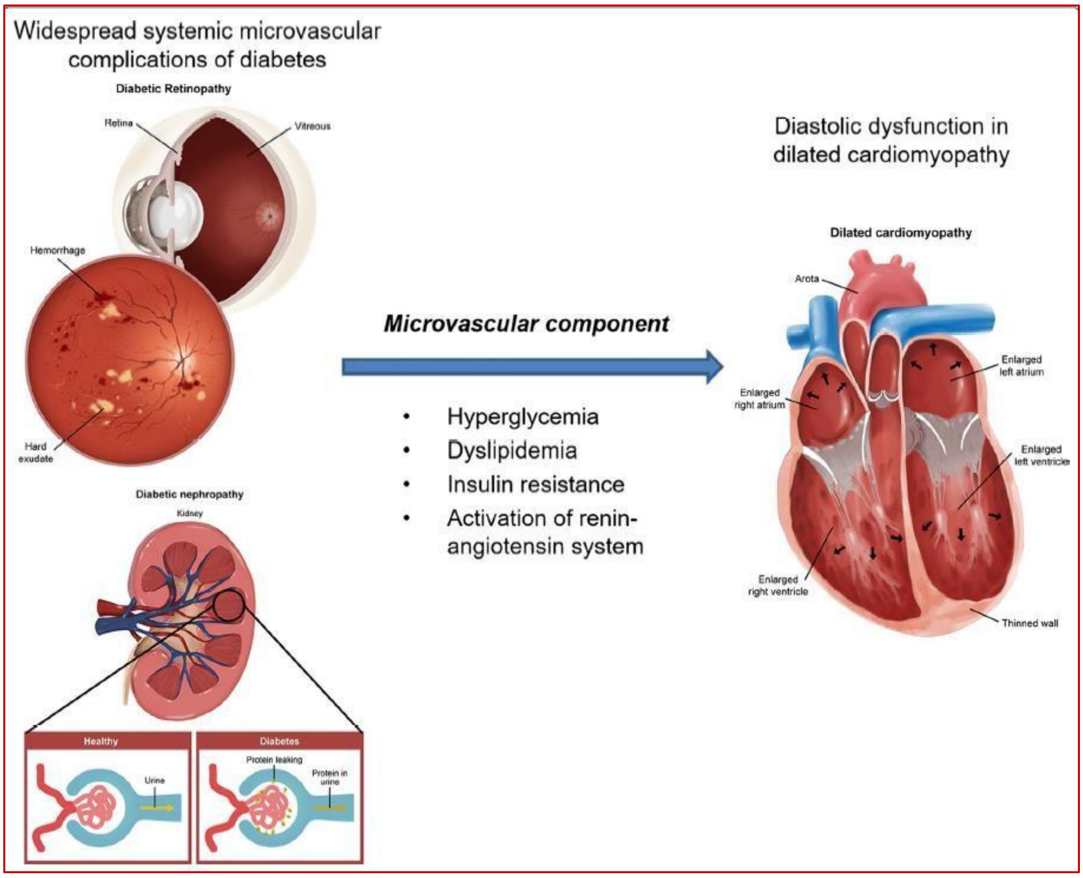

IMAGE 7: Pathophysiology mechanism that leads to dialated cardiomyopathy [49]

D. Assessment techniques for myocardial remodeling

Assessment techniques for myocardial remodeling in the context of diabetic cardiomyopathy are critical for early diagnosis, monitoring progression, and guiding therapeutic interventions. These techniques range from basic clinical assessment to advanced imaging modalities and biochemical markers, providing a comprehensive overview of the structural and functional changes occurring in the heart due to diabetes.

1. Echocardiography: The gold standard for evaluating myocardial remodelling is still echocardiography. It is a widely accessible, reasonably priced, and non-invasive technology that enables in-depth assessment of heart anatomy and function. Systolic and diastolic function, chamber diameters, wall thickness, and left ventricular mass are among the parameters that are often measured. Speckle tracking echocardiography, a newer technique, provides advanced insights by measuring myocardial strain, offering an early indication of subclinical myocardial dysfunction before changes in ejection fraction or diastolic parameters become apparent. The detection of reduced longitudinal strain, in particular, has been associated with diabetic cardiomyopathy and is predictive of future cardiovascular events.[36]

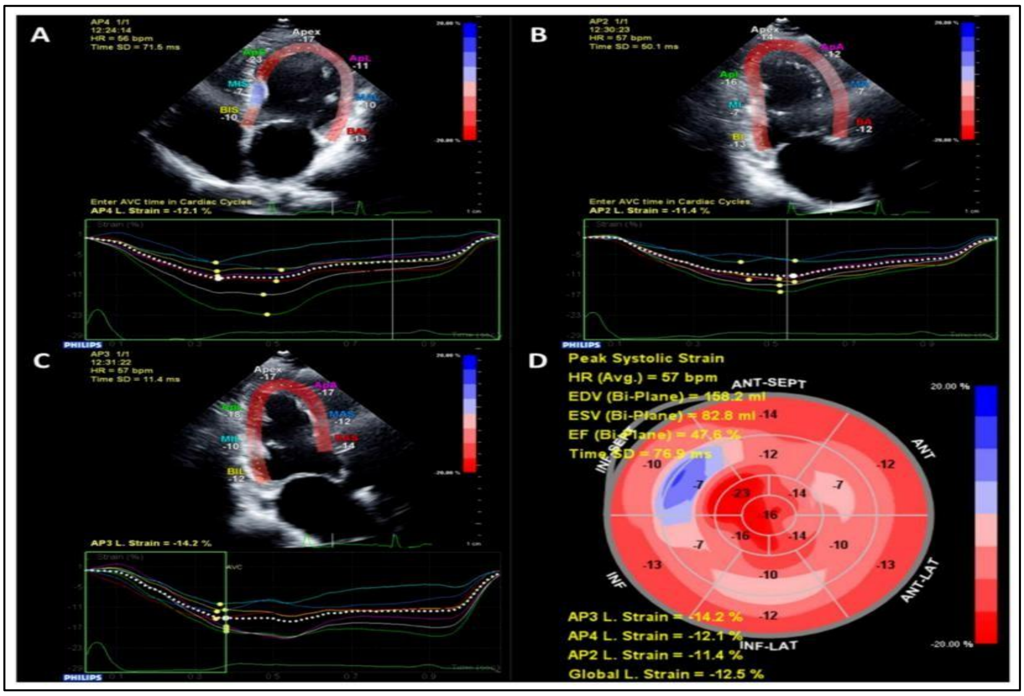

IMAGE 8: An example of echocardiographic analysis combining all three views is generated with reduced global longitudinal strain of −12.5% and reduced LVEF (47.6%). [67]

2. Cardiac Magnetic Resonance Imaging (CMR): CMR is the gold standard for quantifying myocardial mass, volume, and ejection fraction, providing high-resolution images without the exposure to ionizing radiation. It is particularly useful for characterizing myocardial fibrosis using late gadolinium enhancement (LGE) techniques, which can detect focal areas of fibrosis typically seen after myocardial infarction. Additionally, CMR with T1 mapping allows for the assessment of diffuse myocardial fibrosis, a hallmark of diabetic cardiomyopathy that may not be evident on LGE. T1 mapping provides a quantitative measure of myocardial extracellular volume, indicative of fibrosis and remodeling.

3. Cardiac Computed Tomography (CT): While less commonly used for direct assessment of myocardial remodeling, cardiac CT can provide valuable information about coronary artery calcification (CAC), which is frequently seen in diabetic patients. The extent of CAC correlates with coronary atherosclerosis and can indirectly suggest myocardial ischemia and remodeling. Additionally, cardiac CT can accurately measure myocardial mass, ventricular function, and volumes, enhancing the results of other imaging modalities. [37]

4. Nuclear Imaging: The main objective of methods such as Positron Emission Tomography (PET) and Single Photon Emission Computed Tomography (SPECT) is to evaluate the heart's vitality and perfusion. These techniques can detect reduced myocardial perfusion, which is caused by microvascular dysfunction in diabetes, even when there is no overt signs of coronary artery disease. PET can specifically assess cardiac metabolism and offer details on the altered metabolic states characteristic of diabetic cardiomyopathy, such as increased fatty acid metabolism and decreased glucose utilization.

5. Serum Biomarkers: Several serum biomarkers are emerging as useful tools for assessing myocardial stress and fibrosis, which are integral to remodeling. Biomarkers such as B-type natriuretic peptide (BNP) or N-terminal pro b-type natriuretic peptide (NT-proBNP) are elevated in heart failure and correlate with both systolic and diastolic dysfunction. Other markers such as galectin-3, which is associated with fibrosis and inflammation, and cardiac troponins, which indicate myocardial injury, are also relevant in monitoring diabetes-related cardiac changes.[38]

6. Electrocardiography (ECG): Although ECG is a basic tool, it provides essential information on cardiac rhythm and conduction abnormalities, which are common in diabetic cardiomyopathy. Changes in ECG patterns, such as QT interval prolongation, can indicate alterations in myocardial repolarization secondary to myocardial hypertrophy or fibrosis.

7. Stress Testing: When used with imaging techniques like nuclear imaging or echocardiography, exercise or medication stress testing can evaluate the heart's functional ability and identify ischaemia. Stress testing is essential for assessing coronary artery disease risk in diabetes individuals since it can lead to myocardial remodelling. [39]

IV. Quality of Life in Diabetes Patients

The physical and psychological strains of controlling diabetes have a substantial influence on patients' quality of life (QoL). Diabetes and its associated complications can lead to functional limitations, emotional stress, and financial pressures, all of which deteriorate the overall quality of life. [40] Chronic hyperglycemia and its consequences, like myocardial remodeling and diabetic retinopathy, exacerbate everyday living and social interactions by increasing morbidity and disability. Research shows that the prevalence of diabetes-related issues, particularly in terms of psychological and physical functioning, is clearly correlated with lower QoL ratings. Improving life quality requires comprehensive care that takes mental health and patient education into account, along with effective management techniques that include good glycemic control. [41]

A. Factors influencing diabetic patients' quality of life:

Numerous factors, including clinical symptoms and outcomes, social support systems, and psychological health, have a substantial impact on the quality of life (QoL) of diabetic patients. Diabetes is a chronic, complex illness that often necessitates lifestyle modifications and ongoing medical care, both of which can significantly affect a patient's overall quality of life.

1. Physical Complications: Physical difficulties are one of the main things reducing diabetics' quality of life. These sequelae, which include cardiovascular diseases like myocardial remodeling, diabetic neuropathy, nephropathy, and retinopathy, can cause significant physical impairment. For example, diabetic retinopathy may cause visual impairments that affect a patient's capacity to carry out daily tasks, while diabetic neuropathy may cause chronic pain or numbness that impairs mobility and daily activities. Physical disability is also exacerbated by diabetic foot ulcers and the risk of amputation. Myocardial remodeling highlights cardiovascular complications that result in decreased cardiac efficiency, which can cause fatigue and a decreased ability to tolerate physical activity, thus lowering quality of life. [42]

2. Psychological Factors: Managing diabetes requires ongoing awareness, which may lead to psychological distress. Compared to the general population, diabetic patients are more likely to experience anxiety and depression. The anxiety of hypoglycemia episodes or the strain of managing chronic hyperglycemia may exacerbate these psychological issues. A patient's motivation and capacity to adequately manage their diabetes are also impacted by mental health conditions, which can further deteriorate their quality of life by forming a cyclical pattern.[40]

3. Social and Economic Factors: Diabetes patients' quality of life is greatly impacted by social variables, such as the support of friends and family. Social support can increase adherence to treatment programs and reduce the psychological burden that comes with treating a chronic illness. On the other hand, a lower quality of life and worse health outcomes have been associated with social isolation. Economic considerations are also very important; the cost of continuing medical care, prescription drugs, monitoring devices, and the possibility of losing one's job because of a disability can be very stressful and restrict access to quality care, which lowers a patient's quality of life.

4. Management of Diabetes: One important aspect influencing quality of life is the degree of diabetes management. Effective care that maintains blood glucose levels within a target range can improve quality of life by preventing or postponing the onset of issues and minimizing symptoms. It can be exhausting and negatively impact quality of life to follow the routine required to achieve this control, which includes frequent blood glucose checks, medication compliance, and dietary and activity modifications. Additionally, a patient's assessment of their quality of life may be even more impacted by their anxiety about long-term issues and medication side effects.[43]

5. Lifestyle Limitations: Although changing one's lifestyle is essential for managing diabetes, it may also have negative effects on one's quality of life. Food enjoyment and social interactions can be impacted by dietary restrictions. Even though it helps manage diabetes, complications like neuropathy or cardiovascular issues may limit physical activity. The patient's general level of life satisfaction may be impacted by these limitations, which may cause them to feel frustrated and deprived.

6. Education and Self-Efficacy: Quality of life can be greatly impacted by patient education regarding diabetes management and related complications. If patients understand the illness process and know how to manage various aspects of it, they may feel more empowered, have higher self-efficacy, and potentially lead better lives. Patients who believe they can manage their diabetes have a higher quality of life than those who lack. [44]

B. Measurement of quality of life: Instruments and scales

Assessing the quality of life (QoL) of individuals with diabetes is essential to comprehending the disease's wider effects and managing it. A wide range of perspectives on health and well-being, including social, psychological, and physical aspects, are included in quality of life. Numerous instruments and scales have been developed and validated to assess these aspects, particularly in the diabetic population, reflecting the unique challenges and burdens that persons with diabetes face.

1. Generic Quality of Life Instruments: In order to provide a comprehensive view of health state, generic instruments are made to evaluate quality of life across all illnesses and situations.

One of the most often used general tools is the Short Form Health Survey (SF-36). Vitality (energy/fatigue), social functioning, role limitations due to emotional problems, physical functioning, role limitations due to physical health, bodily pain, general health, and mental health (psychological distress and psychological well-being) are the eight domains covered by its 36 items. This instrument is very useful for both clinical practice and research since it allows comparisons between the health status of people with diabetes and other chronic diseases as well as the general population. [45]

One well-liked generic tool is the EuroQol five-dimension questionnaire (EQ-5D). It assesses health outcomes in five domains: self-care, mobility, pain/discomfort, anxiety/depression, and regular activities. Additionally, users can score their overall health on a scale of 0 to 100 using its visual analogue scale (VAS). Due to its ease of use, this measure has been frequently used in economic assessment studies to assess the health-related quality of life of individuals with diabetes.

2. Diabetes-Related Quality of Life Measures: Diabetes-specific resources focus on issues that are particularly significant to individuals with the condition, including the impact of symptoms, diabetes management, and potential psychological repercussions.

The Diabetes Quality of Life (DQOL) measure was developed especially for individuals with diabetes to evaluate the effects of diabetes on a person's life. It is divided into four categories: life satisfaction, diabetes impact, diabetes-related concerns, and treatment satisfaction. This scale has been essential in highlighting the particular challenges faced by individuals with diabetes and the effectiveness of therapies intended to improve their quality of life.[46]

Diabetes-specific emotional distress is measured by the Problem Areas In Diabetes (PAID) scale. It includes 20 items that measure concerns about care access, emotional burden, support, and disease management. This scale is frequently used in clinical trials to assess psychological interventions because it is sensitive to changes in emotional distress associated with diabetes.

3. Tools Particular to Pediatric Diabetes: Children and adolescents with diabetes and their families are the focus of quality of life assessments in pediatric populations.

The Paediatric Quality of Life Inventory (PedsQL) Diabetes Module is intended for children and adolescents. Among the crucial areas it evaluates are diabetes symptoms, treatment barriers, treatment adherence, anxiety, and communication. This is crucial because managing diabetes in younger people entails variables that are distinct from managing diabetes in adults, such as family relationships and school contexts. [47]

4. Utility-Based Instruments: These instruments are designed to measure the preference or utility of different health states and are particularly useful for health economic evaluations.

The Health Utilities Index (HUI) evaluates a number of health attributes, including vision, hearing, speech, mobility, dexterity, emotion, cognition, and pain, and gives each attribute a handicap degree. It facilitates cost-utility assessments in healthcare decision-making, particularly diabetes treatment programs, by producing utility ratings that represent the quality of life associated with various health conditions. [48]

V. Interrelationship between Diabetic Retinopathy and Myocardial Remodeling

The systemic character of diabetes sequelae is shown by the interaction between diabetic retinopathy (DR) and cardiac remodelling in individuals with Type II Diabetes Mellitus (T2DM). The underlying cause of both conditions is diabetes's chronic hyperglycemic environment, which results in changes to the micro and macrovascular systems. In DR, prolonged exposure to high glucose levels harms the retinal blood vessels, causing hypoxia and retinal ischaemia. In a similar vein, metabolic diseases and microvascular dysfunction affect myocardial remodeling by reducing myocardial blood flow and resulting in structural changes such as hypertrophy and fibrosis.

New research suggests that the presence of DR may predict myocardial remodelling and other cardiovascular disorders. Research indicates that the degree of heart failure and the severity of DR are correlated, pointing to a common pathophysiological mechanism that includes oxidative stress, inflammation, and endothelial dysfunction. This link highlights the importance of careful cardiovascular monitoring in patients with DR as part of an all-encompassing approach to managing diabetes and its associated consequences.[49]

A. Evidence of the link between cardiac changes and diabetic retinopathy

Diabetes mellitus has a systemic effect on vascular health, as evidenced by the association between diabetic retinopathy (DR) and cardiac alterations, specifically myocardial remodeling. Both disorders are signs of the macrovascular and microvascular problems linked to diabetes's chronic hyperglycemia. Numerous studies have connected the prevalence and severity of diabetic retinopathy to a range of cardiac problems, indicating that diabetes and these conditions share pathogenic mechanisms. [50]

1. Microvascular Dysfunction as a Common Mechanism: Chronic hyperglycemia causes microvascular damage that affects both diabetic retinopathy and cardiac remodelling. Increased arterial permeability, endothelial dysfunction, and thickening of the basement membrane are the hallmarks of this injury. These alterations cause retinal ischaemia in the retina, which is followed by proliferative diabetic retinopathy. Similarly, in the heart, microvascular dysfunction can impair myocardial blood flow, contributing to myocardial ischemia, a critical factor in myocardial remodeling. Studies using fluorescein angiography have shown that retinal vascular leakage in diabetic retinopathy correlates with impaired myocardial perfusion in patients with diabetes, indicating a systemic microvascular dysfunction. [36]

2. Endothelial Dysfunction and Inflammatory Pathways: Endothelial dysfunction is well documented to play a role in the development of heart problems and diabetic retinopathy. Endothelial cells are harmed by reactive oxygen species (ROS), which are produced as a result of oxidative stress caused by hyperglycemia. Both the pathophysiology of DR and myocardial remodeling are significantly impacted by this damage. Furthermore, diabetes is linked to an inflammatory state characterized by high levels of inflammatory cytokines like tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). These cytokines are known to play a part in the fibrotic processes involved in heart remodeling and have been linked to the development of diabetic retinopathy.

3. Shared Risk Factors: The epidemiological links between diabetic retinopathy and myocardial remodeling are further supported by common risk factors like hypertension, dyslipidemia, and the duration of diabetes. Studies have demonstrated that these cardiovascular risk factors can predict the extent of myocardial remodeling as well as diabetic retinopathy. For example, hypertension accelerates endothelial damage and vascular remodeling by placing additional strain on the vascular system, which worsens the course of both disorders. [51]

4. Diabetic Retinopathy's Predictive Value for Heart Failure: Numerous long-term studies have demonstrated that diabetic retinopathy is predictive of myocardial dysfunction and subsequent cardiac events. This predictive association underscores the potential for diabetic retinopathy to serve as a clinical indicator of systemic vascular disease, including cardiac abnormalities. For example, patients with proliferative diabetic retinopathy have a higher chance of developing heart failure, suggesting that severe retinal disease could be a sign of more severe systemic microvascular damage, such as myocardial damage. [52]

5. Clinical Research and Imaging Data: Modern imaging techniques have reinforced the connection between diabetic retinopathy and cardiac remodeling. Echocardiography and cardiac magnetic resonance imaging (MRI) have been used to assess the shape and function of the myocardium in patients with diabetic retinopathy. These studies indicate that myocardial fibrosis and left ventricular hypertrophy are more common in patients with advanced stages of diabetic retinopathy. [53]

B. Mechanistic understanding of diabetic retinopathy's systemic effects on the heart

Both myocardial remodeling and diabetic retinopathy (DR) are serious side effects of diabetes that point to underlying systemic effects that go beyond their respective pathophysiological settings. Important information about the wider systemic effects of diabetes on cardiovascular health can be gained from the interactions between these conditions. Numerous mechanistic pathways, including oxidative stress, inflammatory processes, chronic hyperglycemia, and shared vascular risk factors, mediate this association.

These processes not only make myocardial and retinal damage worse, but they also demonstrate how interrelated diabetic complications are.

1. Chronic Hyperglycemia and Metabolic Dysregulation: Chronic hyperglycemia is the main cause of both diabetic retinopathy and cardiac remodeling. Through ischemic-driven angiogenesis, prolonged exposure to high glucose levels damages the capillaries in the retina, leading to microaneurysms, hemorrhages, and eventually proliferative diabetic retinopathy. In a similar vein, hyperglycemia leads to the accumulation of advanced glycation end-products (AGEs) in the myocardial tissue of the heart. The cross-linking of collagen and elastin fibers brought on by these AGEs stiffens the heart and promotes fibrosis. This fibrotic process impairs myocardial compliance and function, resulting in diastolic dysfunction and, eventually, systolic failure.[54]

2. Oxidative Stress and Inflammatory Pathways: Diabetic retinopathy and cardiac remodeling are associated with elevated levels of oxidative stress and inflammation. An imbalance between the generation of reactive oxygen species (ROS) and the antioxidant defense system's capacity causes oxidative stress in diabetic retinopathy. Leakage, neovascularization, and vision loss result from the oxidative stress that damages retinal cells and capillaries. Similar to this, oxidative stress in the heart causes harm to the microvascular endothelium and cardiomyocytes, which leads to fibrosis and cell death. The inflammation associated with diabetes, characterised by elevated levels of cytokines such as TNF-α, IL-6, and IL-1β, exacerbates tissue damage in the heart and retina by promoting fibrosis and endothelial dysfunction. [33]

3. Endothelial Dysfunction and Microvascular Damage: Endothelial dysfunction, which affects several organ systems, including the heart and retina, is a common consequence of diabetes. In diabetic retinopathy, endothelial damage results in the breakdown of the blood-retinal barrier, increased vascular permeability, and macular oedema. Endothelial dysfunction in the heart can cause myocardial ischaemia and remodelling, as well as lowering nitric oxide availability, impeding vasodilation, and increasing vascular resistance. Thus, diabetes-related microvascular damage offers a mechanistic connection between retinal and cardiac symptoms, indicating that treatments targeted at enhancing endothelial function may help both diabetic retinopathy and heart health. [12]

4. Neurohormonal Activation: Diabetes is connected to alterations in neurohormonal activity, notably the sympathetic nervous system and the renin-angiotensin-aldosterone system (RAAS). These alterations directly support diabetic retinopathy and cardiac remodelling in addition to causing hypertension and vascular damage. For example, angiotensin II, a key effector of RAAS, promotes inflammation, endothelial dysfunction, and vascular permeability in the retina, and stimulates myocardial fibrosis and hypertrophy. Thus, RAAS inhibitors, which are commonly used in the management of diabetic patients, may provide therapeutic benefits by attenuating the progression of both DR and cardiac remodeling.

5. Shared Genetic and Environmental Factors: The susceptibility to both diabetic retinopathy and myocardial remodeling may be influenced by genetic predispositions that affect the systemic response to diabetes. Furthermore, both illnesses are made worse by environmental variables including nutrition, smoking, and physical inactivity, underscoring the need of lifestyle management in reducing the systemic effects of diabetes.[55]

C. Clinical studies and outcomes

Aguilar D et al. (2008) employed stereoscopic fundus photography to assess the degree of diabetic retinopathy in 531 Mexican-American individuals with type 2 diabetes. The severity of retinopathy was linked to higher HbA1c, albuminuria, duration of diabetes, cardiovascular disease, and hypertension; severe retinopathy was also associated with a larger left ventricular mass and left atrial size, a worse LV ejection percentage, and fractional shortening. [56]

The 2015 research by Herman WH et al. We calculated the advantages of type 2 diabetes screening and early treatment using the Michigan Model and information from the ADDITION-Europe project. When compared to delayed diagnosis, the results demonstrated significant risk reductions in cardiovascular outcomes with screening and early treatment. The advantages of early diagnosis and timely multifaceted therapy are greater than those of treatment intensity, underscoring the significance of screening for fast intervention. [57]

Zhen Z et al. in 2015 study of 134 patients with T2DM and no coronary artery disease, retinal photography and echocardiography were used to assess diabetic retinopathy and cardiac function. Those with diabetic retinopathy showed impaired diastolic and systolic function at rest and during exercise, as indicated by higher E/E′ ratios, impaired GLS, low DFRI, and stress GLS. These findings highlight the impact of diabetic retinopathy on myocardial function.[58]

Ernande L et al. in 2014 study of 172 type 2 diabetes patients, those with εL alteration had higher LV end-systolic volumes and relative wall thickness (RWT) at baseline. Over 3 years, LV volumes decreased in patients with normal εL but not in those with εL alteration. εL alteration was linked to higher RWT, larger LV volumes, and less LV volume reduction, suggesting early adverse LV remodeling.[59]

In 2021, Ben ÂJ et al. assessed the utility values of 206 people with type 2 diabetes and diabetic retinopathy using the Brazilian EQ-5D tariffs. Bilateral blindness and sight-threatening retinopathy were associated with significantly lower utility evaluations than those without retinopathy. Although more study is required, these findings show how diabetic retinopathy affects health-related quality of life and might help guide economic assessments. [60]

The impact of diabetic retinopathy (DR) on both overall health-related quality of life (HRQoL) and vision-related quality of life (VRQoL) was examined by Zayed MG et al. in 2024.

According to meta-analyses of 93 research, VRQoL decreased as DR severity increased, with vision-threatening DR showing the most loss. VRQoL may be enhanced by interventions that stop the course of DR. HRQoL had a substantial correlation with vision-threatening DR and a minor correlation with non-vision-threatening DR.[61]

Poonoosamy J et al. in 2023 study review examines the impact of rapid treatment of chronic hyperglycemia on diabetes complications. While controlling hyperglycemia is crucial, rapid correction can worsen microangiopathy and neuropathy. The effects on macroangiopathy are mixed, with increased cardiovascular mortality linked to intensive treatment. New therapies, like gliflozins and hybrid insulin delivery systems, offer personalized approaches for managing diabetes and chronic hyperglycemia. [62]

Horton WB et al. in 2021 study review explores microvascular dysfunction in diabetes and cardiometabolic disease, highlighting its role as an early marker of complications. Microvascular injury begins years before observable damage in organs like the retina, kidneys, and nerves. Early-stage dysfunction is reversible, but if untreated, it can lead to irreversible damage, including heart failure, sarcopenia, and cognitive decline. Novel preventive interventions show promise. [63]

Luneva EB et al. in 2022 study aimed to identify predictors of cardiac fibrosis in type 2 diabetes (T2DM) patients. It found that serum galectin-3 levels were increased in T2DM with left ventricular hypertrophy. A correlation between PICP levels and HbA1c was observed, while TIMP-1 levels were independently associated with cardiac fibrosis. Additionally, increased arterial stiffness and changes in vascular parameters were linked to fibrosis in T2DM. [64]

In a 2023 pilot study, Saucedo L et al. examined the levels of inflammation-related proteins ZAG, Reg-3a, elafin, and RBP-4 in the blood and aqueous humour of diabetic patients with different stages of diabetic retinopathy (DR). ZAG, RBP-4, Reg-3a, and elafin levels are greater in advanced phases (NPDR/PDR) compared to controls. These findings suggest that increased pro-inflammatory markers may contribute to the pathogenesis of DR, even while anti-inflammatory proteins may function as a compensatory strategy.[65]

MATERIALS AND METHODS

MATERIAL AND METHODOLOGY

Source of Data: The data for this cross-sectional observational study came from 97 people with type 2 diabetes mellitus who were recruited from the R. L. Jalappa Hospital & Research Centre in Kolar with ethical approval from the Institutional Ethics Committee of Sri Devaraj Urs Medical College. All participants gave written informed permission before to participating in the study.

Study Design: This cross-sectional observational study sought to determine how people with type II diabetes mellitus, diabetic retinopathy, and cardiac remodelling related to their quality of life.

Study Location: The study was conducted at the R. L. Jalappa Hospital & Research Centre, Kolar, in the departments of Ophthalmology and Cardiology, the latter in collaboration with Narayana Health Heart Centre.

Study Duration: The study spanned a period of 18 months. Recruitment of participants and data collection began after the necessary ethical clearances were obtained and continued until the required sample size was reached.

Sample Size: According to preset inclusion and exclusion criteria, 97 patients with type II diabetes mellitus were included in the study.

Inclusion Criteria:

1. Individuals who have had a type 2 diabetes diagnosis for longer than five years.

2. Individuals without a history of heart problems.

Exclusion Criteria: Patients were excluded from the study if they had retinopathy due to:

1. Hypertension.
2. Anemia.
3. Leukemia.
4. Hypercoagulable states.
5. Radiation exposure.

Procedure and Methodology:

1. Visual Acuity Assessment: The Snellen chart, a common instrument in ophthalmology, was used to measure visual acuity, or each participant's clarity of vision. Each eye was evaluated independently while the other eye was occluded, and patients were positioned 6 feet away from the chart as a standard. The patient's visual acuity, expressed as a fraction (e.g., 6/60, 6/36, etc.), was assessed by accurately reading the shortest line of text. In order to identify any visual impairment that can be caused by diabetic retinopathy, this test is essential.

2. Slit Lamp Biomicroscopy: Slit lamp biomicroscopy was done to carry out a thorough examination of the cornea, iris, and lens of the anterior segment of the eye. Slit lamp facilitated the diagnosis of any diabetic alterations in the iris and lens, such as cataract or neovascularization, which are important findings in diabetic patients. The slit lamp biomicroscope, facilitates accurate evaluation and recordation of anomalies by providing an amplified view of the anatomy of the eye.

3. Posterior Segment Evaluation: Posterior segment evaluation was done using indirect ophthalmoscopy and +90D biomicroscopy. These examinations were performed by two experienced ophthalmologists who were unaware of the diabetes and other potential organ damage of the patients. The blinding allowed us to ensure that retinal health assessments were objective and dependent solely on the ocular signs observed. Indirect ophthalmoscopy enabled a wide view of the retina, which was crucial in the detection of microaneurysms, hemorrhages, and neovascularization, where all these signs were indicative of diabetic retinopathy. Detection and grading of diabetic retinopathy based on the degree and severity of retinal abnormality were facilitated by the +90D biomicroscopy.

Diabetic Retinopathy Classification: The Early Treatment Diabetic Retinopathy Study (ETDRS) grading system was used in grading diabetic retinopathy. The system grades retinopathy into several stages based on the presence and severity of retinal lesions, including microaneurysms, retinal hemorrhages, venous beading, and intraretinal microvascular abnormalities. The study categorization is important to standardize evaluation of the severity of diabetic retinopathy and for deciding the treatment.

Sample Processing

As this cross-sectional observational study focused on clinical assessments and the examination of existing medical records, there was no need to process biological samples. This approach enabled comprehensive data collection regarding the health status of each participant without complicating study procedures or placing a burden on patients.

Data Collection

Standardised questionnaires that were designed to obtain a wide variety of information were employed in gathering data in an orderly fashion. Demographics (age, gender, etc.), duration of diabetes, methods of managing diabetes (diet, exercise, drugs), and specific findings related to diabetic retinopathy and cardiovascular status were all included in the questionnaires. In addition to learning more about diabetes and its complications' effect on their daily activities, patients also undertook a self-reported quality of life measure.

Statistical Analysis

SPSS software, was used to analyse the data. The data was summarised using descriptive statistics, which included proportions, averages, and standard deviations. The associations between diabetic retinopathy, cardiac remodelling, and quality of life were then analyzed using logistic regression models. To ensure that the relationships revealed were strong and trustworthy, these models controlled for possible confounders including age, the length of diabetes, and other risk factors. The results were considered significant if the p-value was less than 0.05. The findings, which were presented as odds ratios with 95% CIs, provide a clear picture of the direction and intensity of the connections under investigation.

RESULTS

RESULTS

Table 1: Distribution of NPDR Severity by Age Group

Age Group	Mild n (%)	Moderate n (%)	Severe n (%)	Total (%)	P value
<40	1 (2.6%)	0 (0.0%)	0 (0.0%)	1 (2.6%)	0.05
41–50	2 (5.3%)	3 (7.9%)	1 (2.6%)	6 (15.8%)	
51–60	4 (10.5%)	2 (5.3%)	3 (7.9%)	9 (23.7%)	
61–70	4 (10.5%)	3 (7.9%)	5 (13.2%)	12 (31.6%)	
>70	2 (5.3%)	3 (7.9%)	5 (13.2%)	10 (26.3%)	
Total	**13 (34.2%)**	**11 (28.9%)**	**14 (36.8%)**	**38 (100%)**	

Table 1 shows the distribution of NPDR severity by age group, revealing a higher prevalence and severity in older age groups, particularly those aged 61–70 years, who exhibit the maximum percentages of moderate and severe NPDR. Notably, the youngest age group (<40 years) shows very low incidence, with only one case of mild NPDR reported, highlighting a significant age-related progression of NPDR. The p-value of 0.05 for the youngest group suggests a statistically significant difference from other groups, indicating the impact of age on NPDR progression.

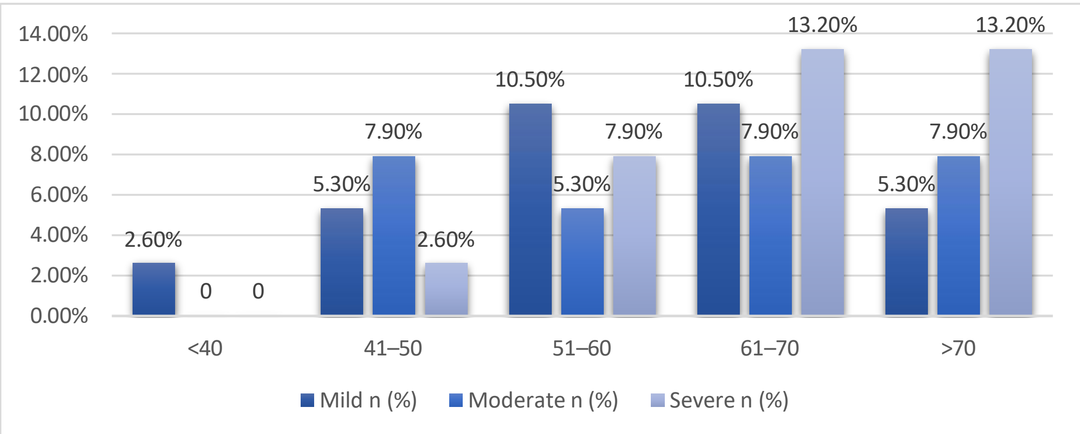

Figure 1: Bar graph of Distribution of NPDR severity by age group

Table 2: Distribution of NPDR Severity by Gender

Gender	Mild n (%)	Moderate n (%)	Severe n (%)	Total (%)	P value
Male	6 (15.8%)	9 (23.7%)	7 (18.4%)	22 (57.9%)	0.01
Female	9 (23.7%)	3 (7.9%)	4 (10.5%)	16 (42.1%)	
Total	**15 (39.5%)**	**12 (31.6%)**	**11 (28.9%)**	**38 (100%)**	

Table 2 focuses on the gender distribution of NPDR severity, where males display a higher overall percentage of NPDR, particularly in moderate and severe categories, compared to females. This is supported by a statistically significant p-value of 0.01, suggesting that gender differences may influence the severity of NPDR.

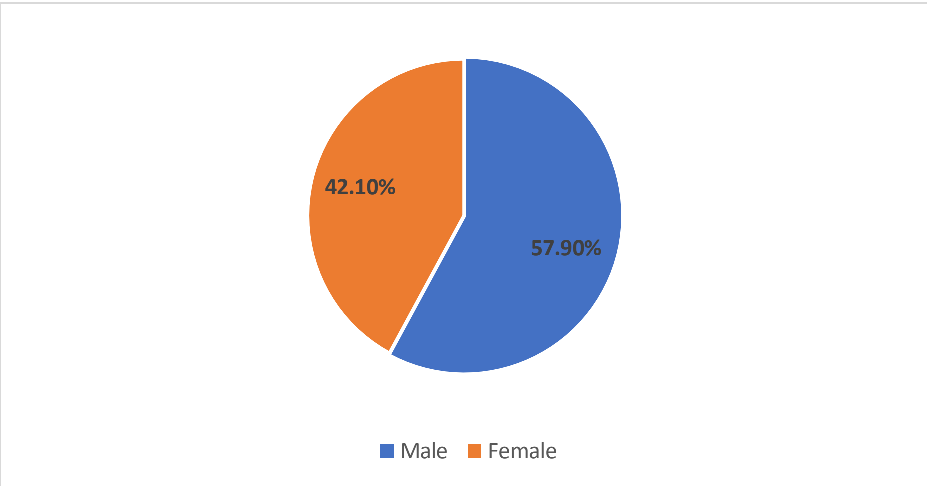

Figure 2: Pie chart representing Distribution of NPDR Severity by Gender

Table 3: Comparison of Blood Pressure, Glycemic Control, and Renal Function Parameters Across NPDR Severity Groups

Parameter	Mild NPDR	Moderate NPDR	Severe NPDR	P value
Systolic BP (mmHg)	126.3 ± 15.1	127.2 ± 14.9	129.2 ± 15.3	0.01
Diastolic BP (mmHg)	79.4 ± 9.4	80.6 ± 9.3	80.2 ± 9.3	0.05
FBS (mg/dL)	148.9 ± 43.1	148.1 ± 42.4	147.2 ± 41.5	0.01
PPBS (mg/dL)	211.2 ± 66.9	210.8 ± 67.2	210.1 ± 67.1	0.001
HbA1c (%)	9.8 ± 2.4	9.1 ± 2.2	9.4 ± 2.5	0.05
Blood Urea (mg/dL)	32.2 ± 15.4	32.4 ± 15.2	31.9 ± 14.9	0.01
Serum Creatinine (mg/dL)	1.9 ± 1.1	1.9 ± 1.1	2.1 ± 1.2	0.05

Table 3 States the association between NPDR severity and different systemic health parameters including blood pressure, glycemic control and renal function. The pattern of increasing systolic blood pressure and decreasing fasting blood sugar (FBS) levels with advancing NPDR severity suggests that tighter glycemic control may be associated with more advanced stages of retinopathy. Furthermore renal function parameters such as blood urea and serum creatinine levels show minor variations across severity groups but are statistically significant, indicating a potential link between renal impairment and NPDR progression.

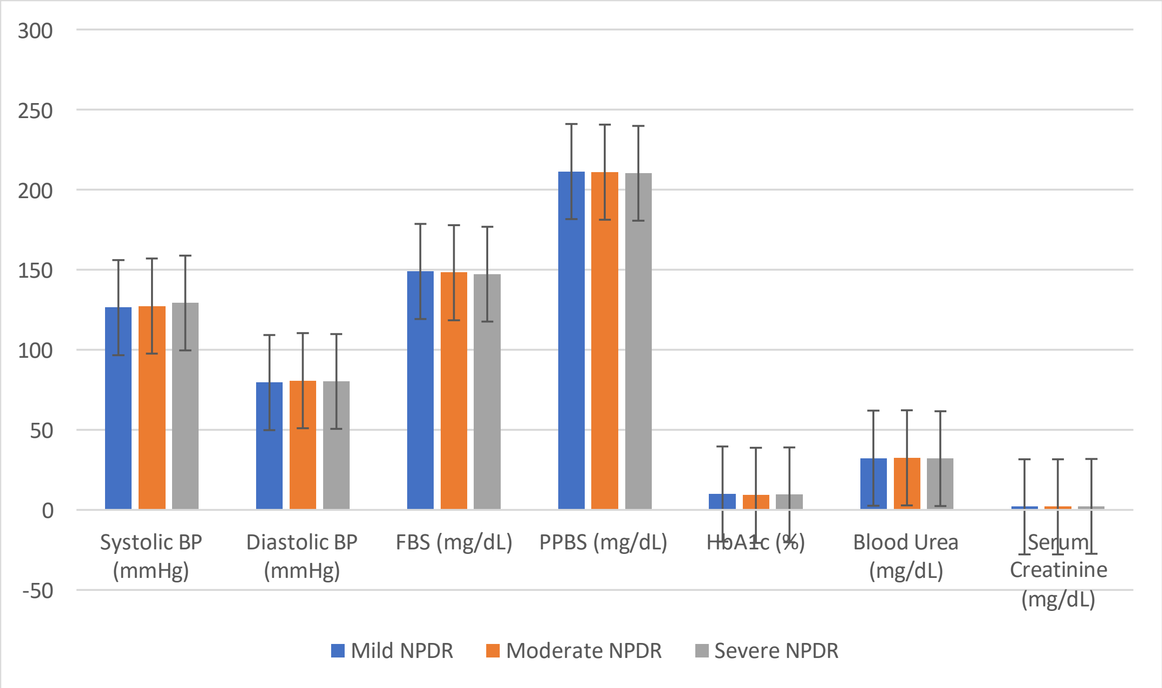

Figure 3: Graph showing the comparison of Blood Pressure, Glycemic Control, and Renal Function Parameters Across NPDR Severity Groups

Table 4: Comparison of Echocardiographic Parameters Across NPDR Severity Groups

Parameter	Mild NPDR	Moderate NPDR	Severe NPDR	P value
LVDD (mm)	44.4 ± 5.4	42.6 ± 5.1	44.6 ± 5.2	0.01
LVDS (mm)	30.1 ± 4.1	30.2 ± 4.6	29.3 ± 4.3	0.01
LVEF (%)	47.8 ± 7.5	49.2 ± 7.3	48.1 ± 7.1	0.05
LA Diameter (mm)	37.4 ± 4.5	38.1 ± 4.2	37.8 ± 4.3	0.01

Table 4 compares echocardiographic parameters across NPDR severity groups, revealing subtle but significant variations in left ventricular dimensions and function. Significant variations in left atrial (LA) diameter and left ventricular ejection percent (LVEF) indicating cardiac alterations that are correlated with the degree of retinopathy, although left ventricular end-diastolic diameter (LVDD) and systolic diameter (LVDS) are comparatively constant between groups.

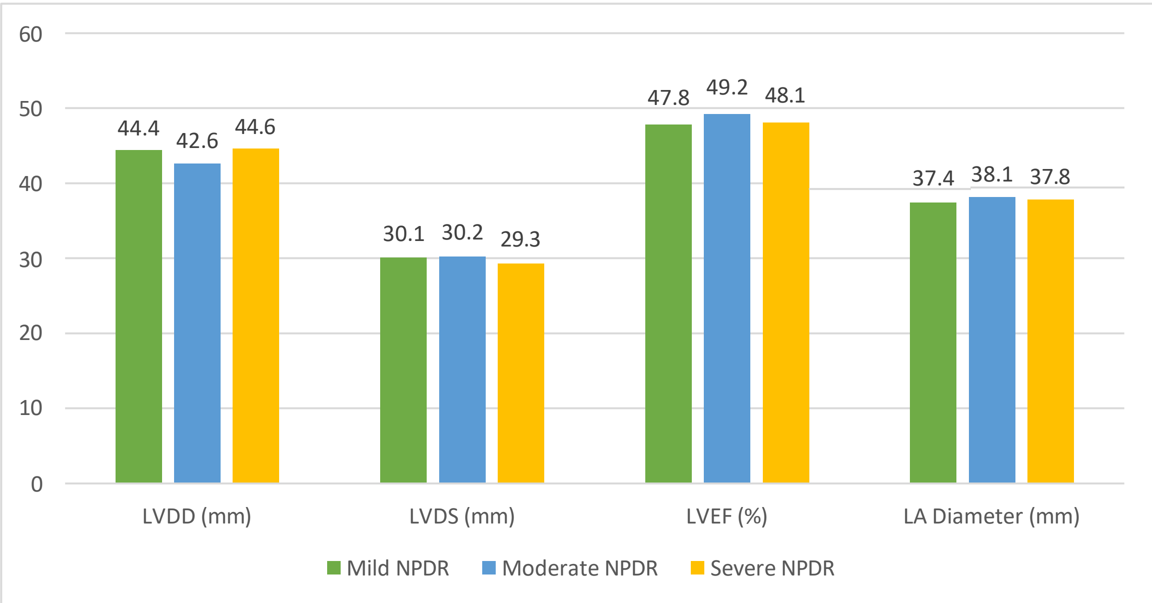

Figure 4: Bar graph representing Comparison of Echocardiographic Parameters Across NPDR Severity Groups

Table 5: Demographic Characteristics

Variable	Category	Total (N=98)	Normal Fundus (n=40)	NPDR (n=38)	PDR (n=20)	p-value
Age Group	<40	3 (3.1%)	2 (66.7%)	1 (33.3%)	0 (0%)	0.12
	41-50	16 (16.3%)	8 (50.0%)	6 (37.5%)	2 (12.5%)	
	51-60	22 (22.4%)	10 (45.5%)	9 (40.9%)	3 (13.6%)	
	61-70	35 (35.7%)	15 (42.9%)	12 (34.3%)	8 (22.9%)	
	>70	22 (22.4%)	5 (22.7%)	10 (45.5%)	7 (31.8%)	
Gender	Male	58 (59.2%)	25 (43.1%)	22 (37.9%)	11 (19.0%)	0.34
	Female	40 (40.8%)	15 (37.5%)	16 (40.0%)	9 (22.5%)	

Table 5: This table outlines the age and gender distribution among the study participants, who total 97. The age groups are segregated into five categories, showing the distribution across the normal fundus, NPDR, and PDR conditions. Interestingly, the youngest age group (<40 years) is predominantly in the normal fundus category, while the older age groups (>70 years) show higher incidences of PDR. The gender distribution shows that males constituted a higher percentage across all conditions compared to females.

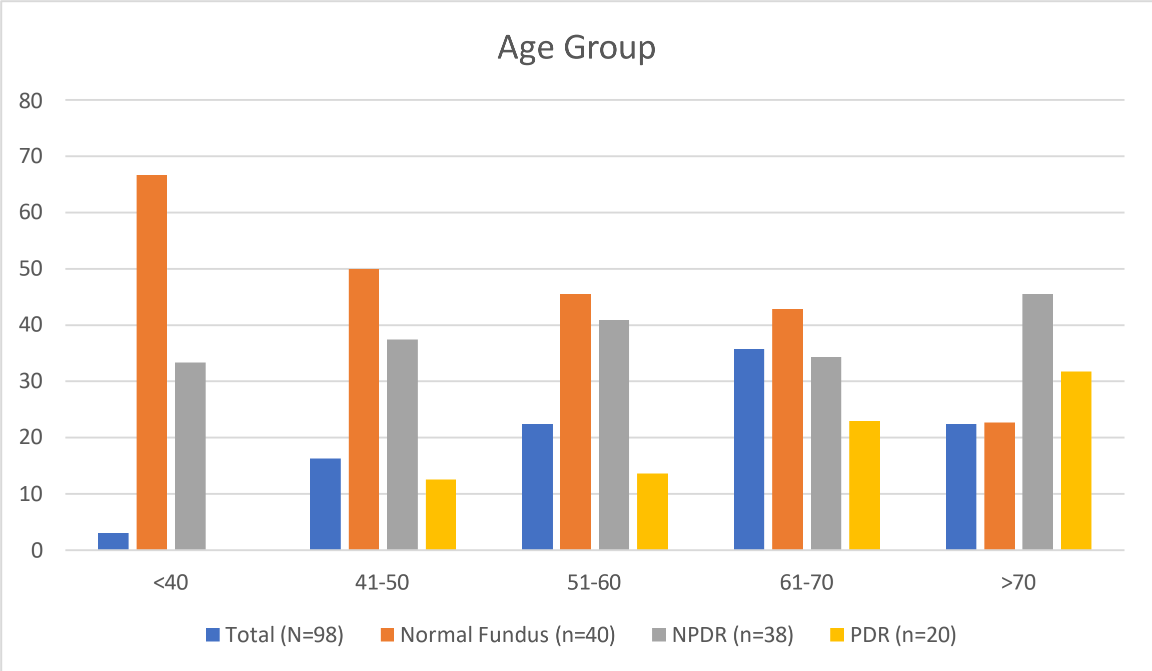

Figure 5: Bar graph representing demographic parameters- Age distribution

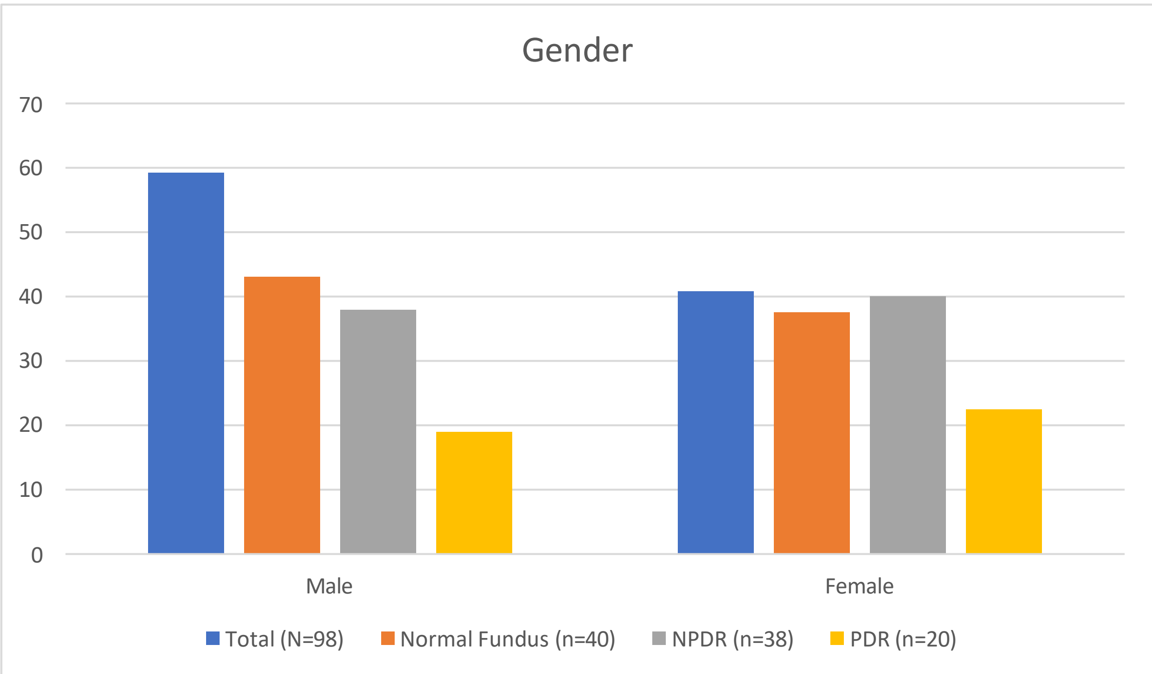

Figure 6: Bar graph representing demographic parameters- Gender distribution

Table 6: Clinical and Biochemical Parameters

Parameter	Normal Fundus (Mean ± SD)	NPDR (Mean ± SD)	PDR (Mean ± SD)	p-value
Systolic BP (mmHg)	124.3 ± 12.8	128.6 ± 15.2	135.4 ± 18.6	0.03*
Diastolic BP (mmHg)	78.2 ± 8.5	80.1 ± 9.3	82.7 ± 10.1	0.12
FBS (mg/dL)	132.5 ± 34.1	148.9 ± 42.6	167.2 ± 55.3	0.01*
PPBS (mg/dL)	185.6 ± 58.2	210.3 ± 67.4	245.8 ± 72.9	<0.01*
HbA1c (%)	8.1 ± 1.6	9.3 ± 2.1	10.8 ± 2.5	<0.001*
B. Urea (mg/dL)	25.4 ± 11.3	32.7 ± 15.8	38.5 ± 18.2	0.02*
S. Creatinine (mg/dL)	1.2 ± 0.6	1.8 ± 1.1	2.4 ± 1.5	<0.01*

Table 6: There are significant variations in the clinical and biochemical indicators of diabetic retinopathy throughout its different stages. Compared to individuals with NPDR and normal fundus, those with PDR exhibited greater levels of systolic blood pressure, fasting blood sugar (FBS), post-prandial blood sugar (PPBS), HbA1c, blood urea, and serum creatinine. Significant p-values showed that metabolic control and renal function deteriorated as retinopathy severity increased.

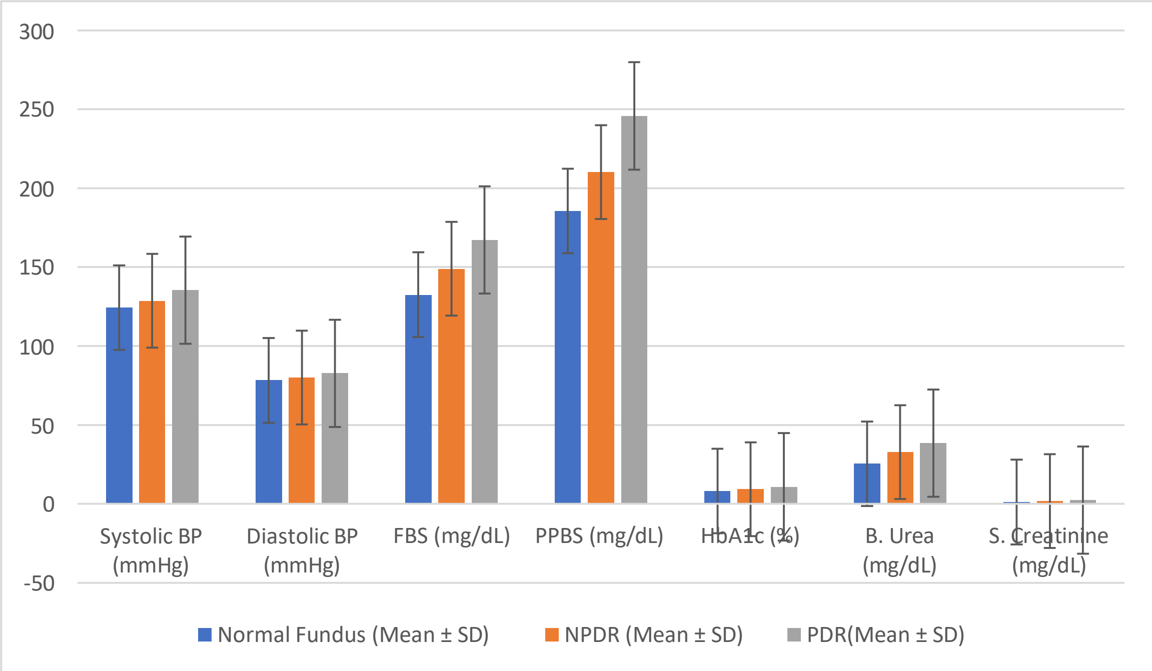

Figure 7: Graph showing the Clinical and Biochemical Parameters

Table 7: Cardiac Remodeling Parameters

Parameter	Normal Fundus (Mean ± SD)	NPDR (Mean ± SD)	PDR (Mean ± SD)	p-value
LVDD (mm)	42.5 ± 4.2	44.8 ± 5.1	47.3 ± 6.5	0.01*
LVDS (mm)	28.3 ± 3.8	30.6 ± 4.5	33.2 ± 5.7	0.02*
LVEF (%)	55.2 ± 6.1	49.8 ± 7.3	43.5 ± 8.9	<0.001*
LA Diameter (mm)	34.6 ± 3.9	37.2 ± 4.5	40.1 ± 5.2	<0.01*

Table 7: Changes in cardiac shape and function are shown in this table, which shows that as diabetic retinopathy progresses, cardiac remodelling gets worse. A progressive increase or decrease in metrics including left ventricular diastolic diameter (LVDD), left ventricular systolic diameter (LVDS), left ventricular ejection fraction (LVEF), and left atrial (LA) diameter is associated with the severity of the retinopathy. The most significant changes are seen in PDR patients.

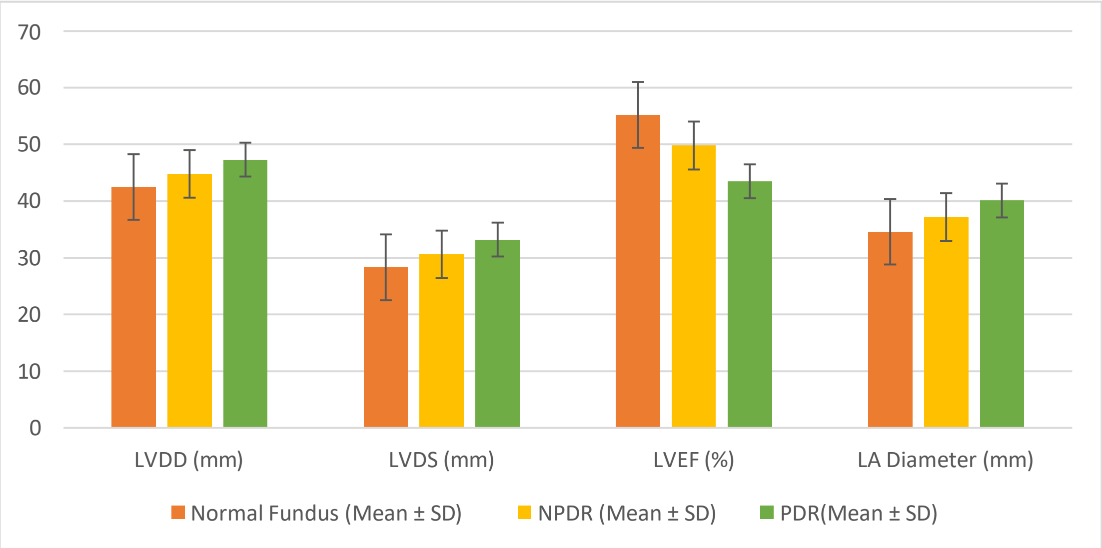

Figure 8: Graphical representation of cardiac remodeling parameters

Table 8: ECG Changes and Quality of Life

Variable	Normal Fundus (n=40)	NPDR (n=38)	PDR (n=20)	p-value
ECG Abnormalities	12 (30.0%)	25 (62.5%)	18 (90.0%)	<0.001*
Quality of Life				
- Good	28 (70.0%)	20 (50.0%)	8 (40.0%)	0.04*
- Fair	8 (20.0%)	12 (30.0%)	9 (45.0%)	
- Excellent	4 (10.0%)	8 (20.0%)	3 (15.0%)	

Table 8: Electrocardiogram (ECG) abnormalities are more prevalent in patients with more severe diabetic retinopathy, with 90% of PDR patients showing ECG abnormalities. As retinopathy worsens, patients' self-reported quality of life likewise declines; individuals with PDR are more likely to have a "fair" or "poor" quality of life than those with less severe or no retinopathy.

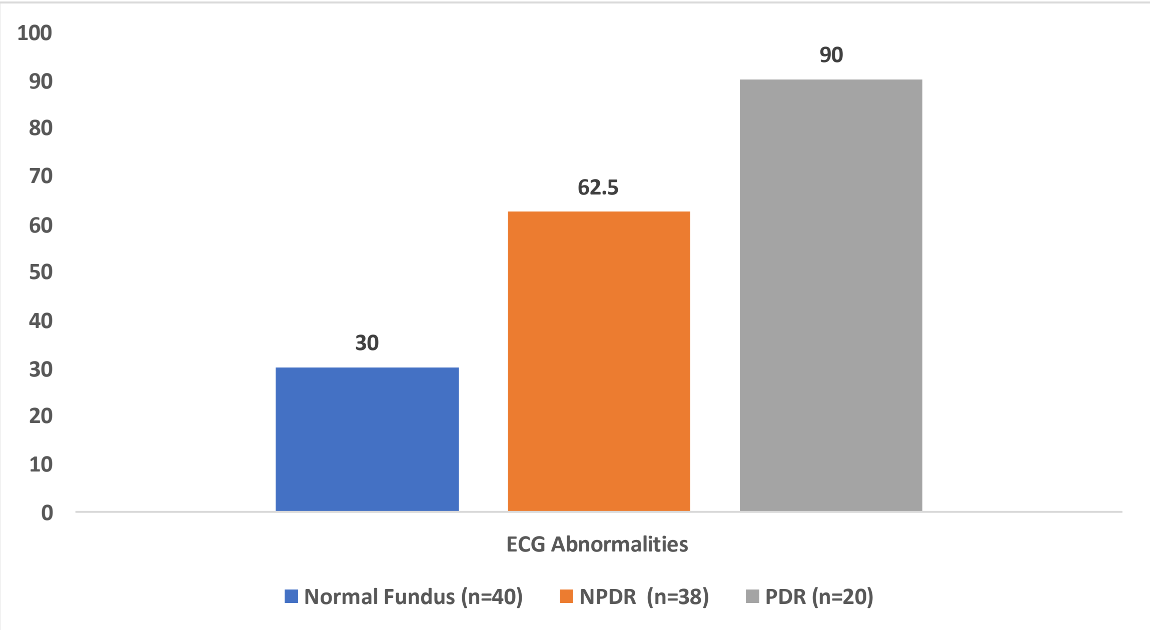

Figure 9: Bar graph representation of ECG abnormalities among patients with and without DR

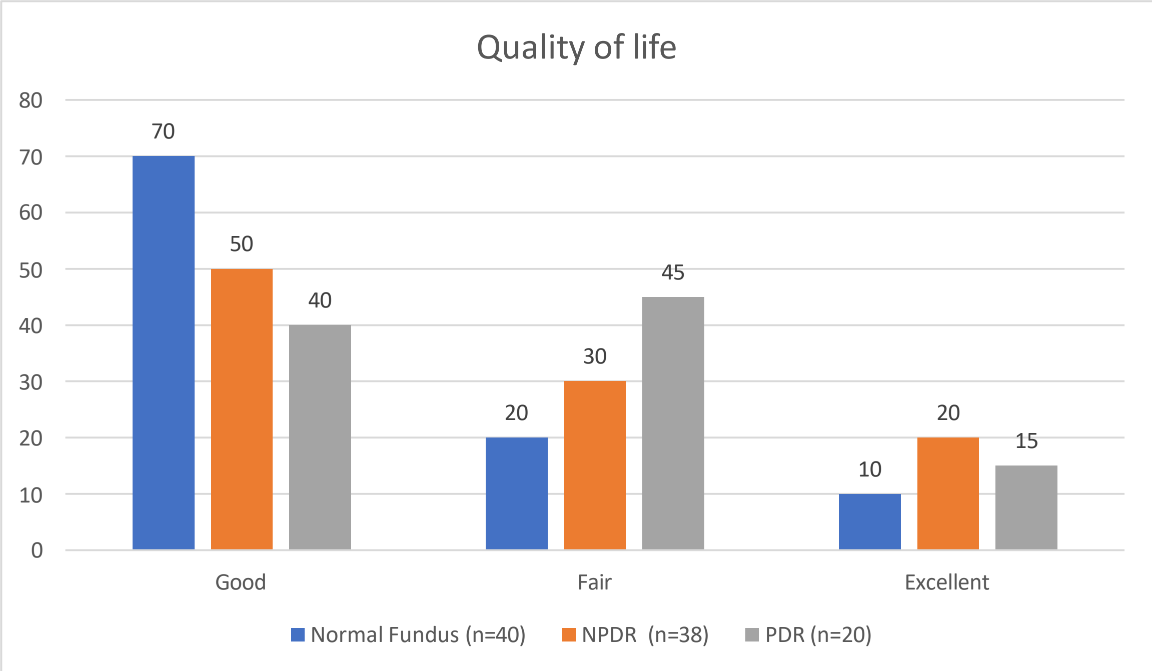

Figure 10: Graphical representation of quality of life among among patients with and without DR

Table 9: Treatment Modalities

Treatment	Normal Fundus (n=40)	NPDR (n=38)	PDR (n=20)	p-value
No Rx	10 (25.0%)	5 (12.5%)	2 (10.0%)	0.22
OHA	18 (45.0%)	22 (55.0%)	8 (40.0%)	0.15
Insulin	8 (20.0%)	25 (62.5%)	15 (75.0%)	<0.001*
Combined	4 (10.0%)	10 (25.0%)	5 (25.0%)	0.18

Table 9: The approach to managing diabetes varies with the severity of retinopathy. While most patients with a normal fundus are either on no medication or oral hypoglycemic agents (OHA), the use of insulin significantly increases with NPDR and PDR. This shift underscores the need for more aggressive treatment as diabetic retinopathy progresses.

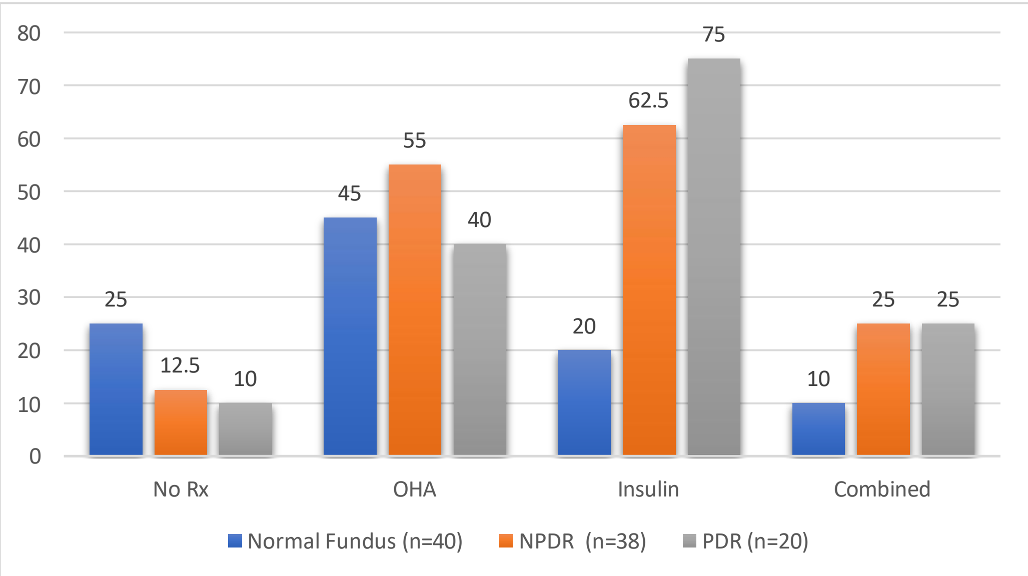

Figure 11: Bar graph showing distribution of treatment modalities among patients with and without DR

Table 10: NPDR Subgroup Analysis (Mild, Moderate, Severe)

Variable	Mild NPDR	Moderate NPDR	Severe NPDR	p-value
Age (Mean ± SD)	58.2 ± 9.4	62.5 ± 8.7	65.3 ± 10.1	0.02*
Gender (% Male)	55.6%	60.9%	48.0%	0.41
Systolic BP (mmHg)	126.4 ± 14.3	132.8 ± 16.1	138.5 ± 17.9	0.01*
HbA1c (%)	8.9 ± 1.8	9.7 ± 2.2	11.2 ± 2.6	<0.001*
LVEF (%)	50.1 ± 6.5	46.3 ± 7.1	41.8 ± 8.4	<0.001*
ECG Abnormalities (%)	45.0%	68.2%	85.7%	<0.001*
Quality of Life (Good)	60.0%	45.5%	28.6%	0.03*

Table 10: categorizes patients into subgroups based on the severity of NPDR—mild, moderate, and severe. A clear trend is observed where age increases with the severity of NPDR, with mean ages of 58.2, 62.5, and 65.3 years respectively, showing a statistically significant difference (p-value = 0.02). The percentage of males also varies across these groups, although this does not reach statistical significance (p-value = 0.41). Systolic blood pressure and HbA1c levels show a progressive increase as NPDR severity worsens, with systolic pressures of 126.4 mmHg in mild cases to 138.5 mmHg in severe cases (p-value = 0.01), and HbA1c levels ranging from 8.9% in mild NPDR to 11.2% in severe NPDR, with a highly significant p-value of less than 0.001. Similarly, left ventricular ejection fraction (LVEF) and the percentage of ECG abnormalities show a significant worsening trend in more severe cases (p-values <0.001 for both), underscoring the cardiovascular risks associated with higher NPDR severity. Furthermore, reported quality of life deteriorates significantly with increasing NPDR severity, with 60.0% of mild cases reporting good quality of life compared to only 28.6% in severe cases (p-value = 0.03).

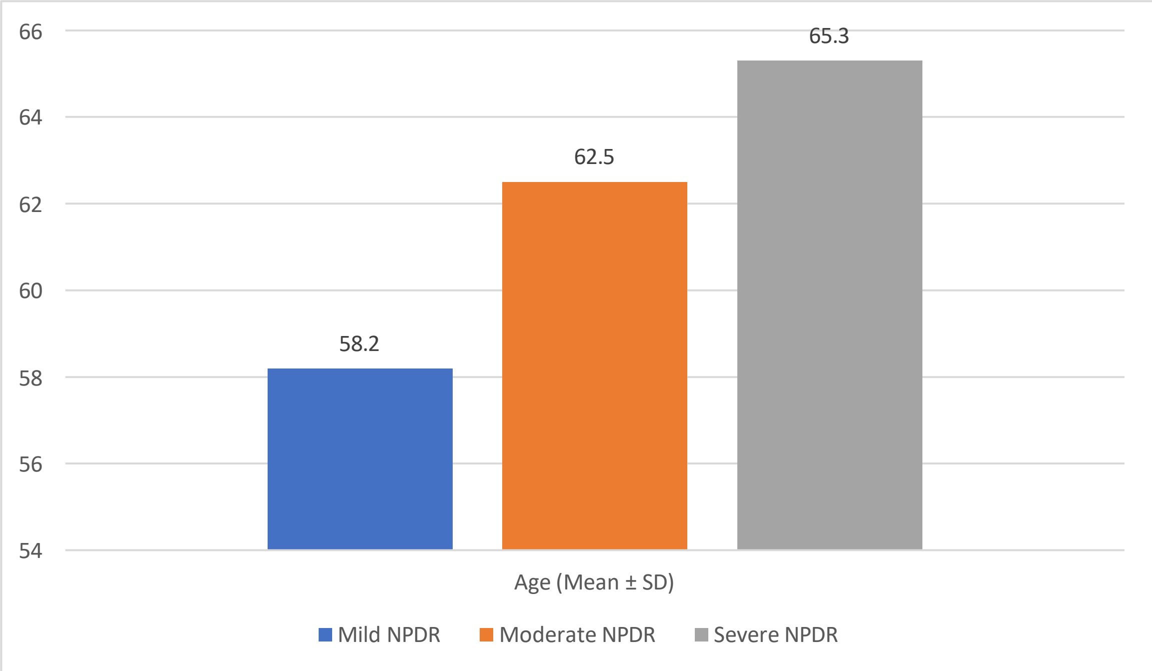

Figure 12: Bar graph of Mean age distribution among patients with Mild, Moderate, and Severe Non-Proliferative Diabetic Retinopathy (NPDR).

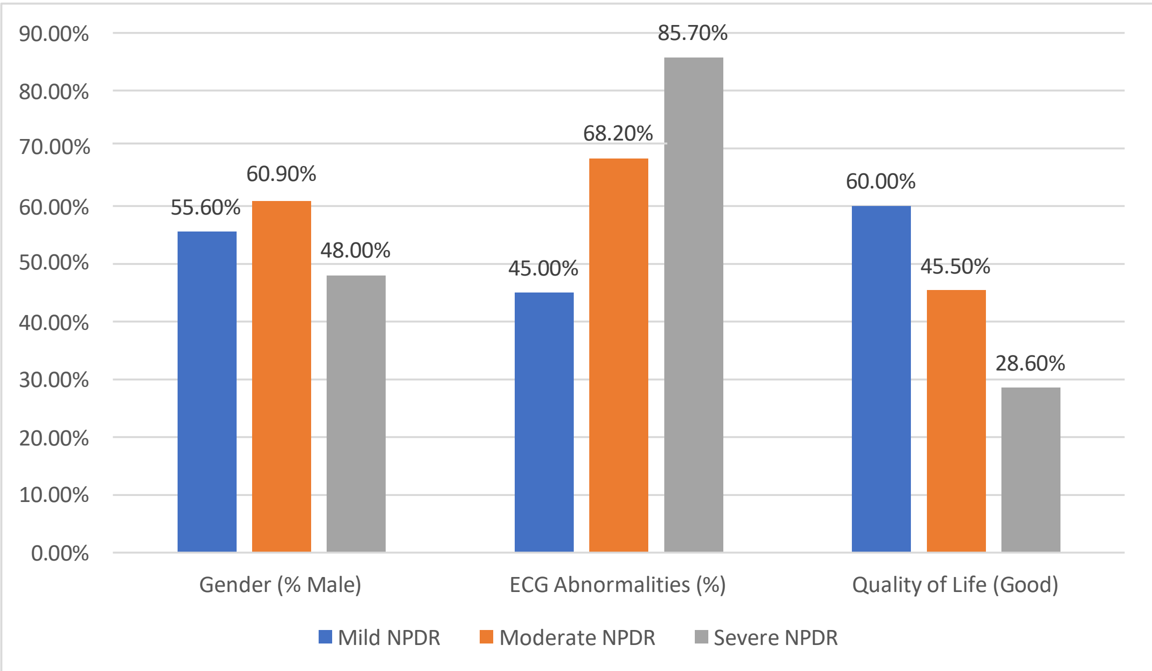

Figure 13: Comparison of Gender Distribution, ECG Abnormalities, and Quality of Life among Patients with Mild, Moderate, and Severe Non-Proliferative Diabetic Retinopathy (NPDR).

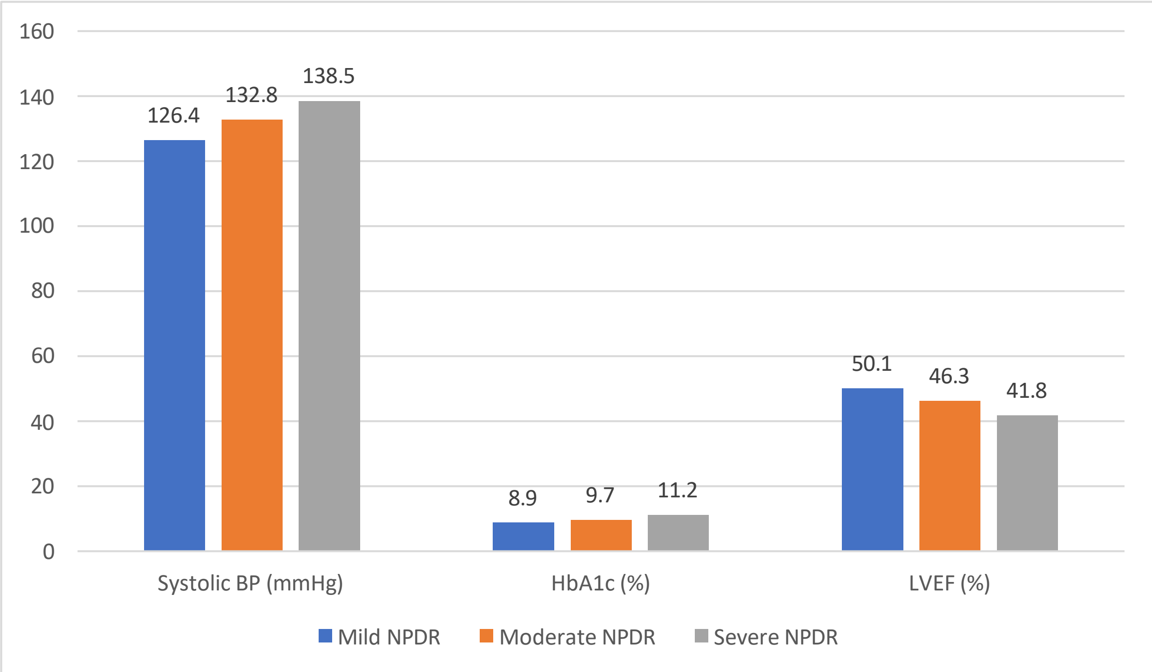

Figure 14: Comparison of Systolic Blood Pressure, HbA1c Levels, and Left Ventricular Ejection Fraction (LVEF) among Patients with Mild, Moderate, and Severe Non-Proliferative Diabetic Retinopathy (NPDR).

Table 11: Association of Diabetes Duration (DM <10 vs. DM >10 Years)

Parameter	DM <10 Years	DM >10 Years	p-value
Retinopathy Severity			<0.001*
- Normal Fundus	65.2%	12.5%	
- NPDR	28.3%	56.3%	
- PDR	6.5%	31.2%	
Cardiac Remodeling			
- LVDD (mm)	42.1 ± 4.0	46.8 ± 5.9	0.002*
- LVEF (%)	54.3 ± 6.2	44.7 ± 8.1	<0.001*
Nephropathy Markers			
- S. Creatinine (mg/dL)	1.4 ± 0.8	2.3 ± 1.4	0.01*
Insulin Use (%)	25.0%	72.7%	<0.001*

Table 11 explores the relationship between the duration of diabetes and the severity of complications. Notably, the longer duration of diabetes (greater than 10 years) is associated with more severe diabetic complications. The incidence of retinopathy, including both NPDR and proliferative diabetic retinopathy (PDR), increases significantly with the duration of diabetes (p-value <0.001), with only 12.5% of those with more than 10 years of diabetes having a normal fundus compared to 65.2% of those with less than 10 years. Cardiac remodeling indicators such as left ventricular diastolic diameter (LVDD) and LVEF also show significant deterioration with longer diabetes duration (p-values 0.002 and <0.001, respectively). Nephropathy markers such as serum creatinine levels are higher in the group with a longer diabetes history (p-value = 0.01), and significantly more patients in this group require insulin (72.7% vs. 25.0%, p-value <0.001).

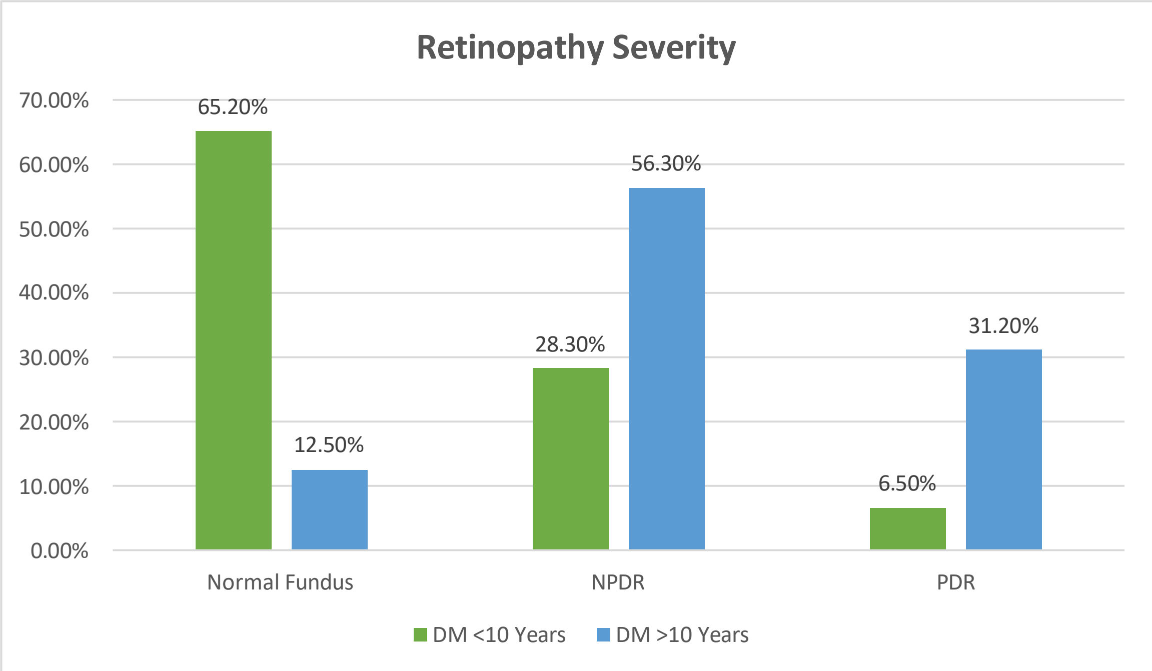

Figure 15: Distribution of Retinopathy Severity Based on Duration of Diabetes Mellitus (DM).

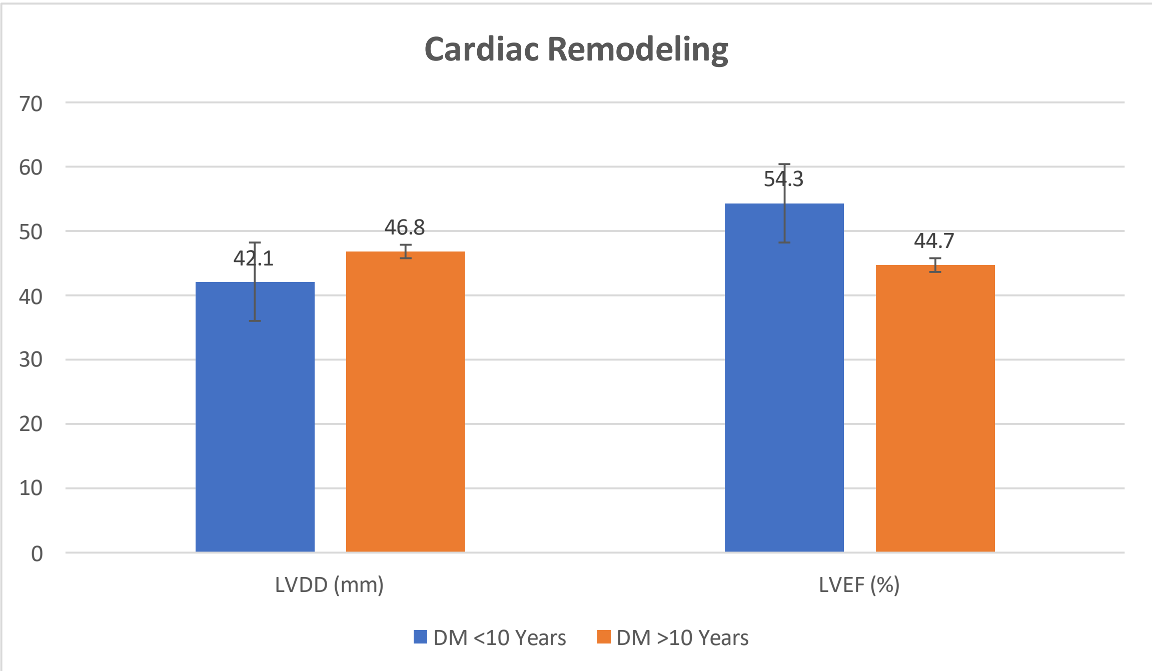

Figure 16: Cardiac Remodeling Parameters in Patients with Diabetes Mellitus (DM) Based on Duration

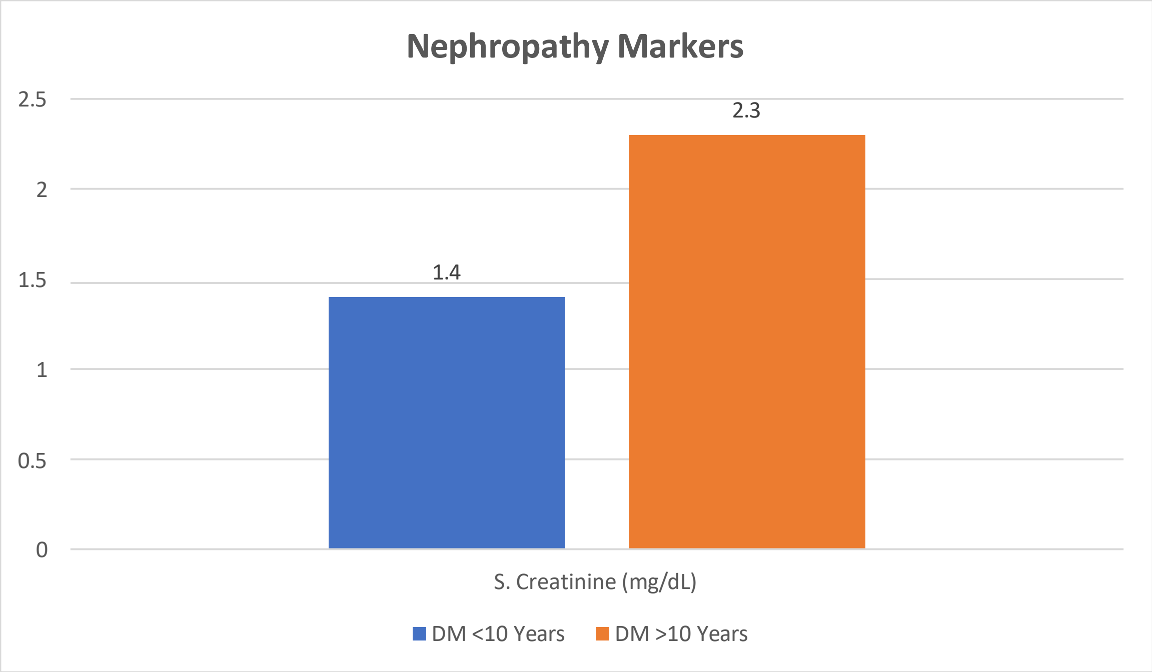

Figure 17: Serum Creatinine Levels in Diabetic Patients Based on Duration of Diabetes

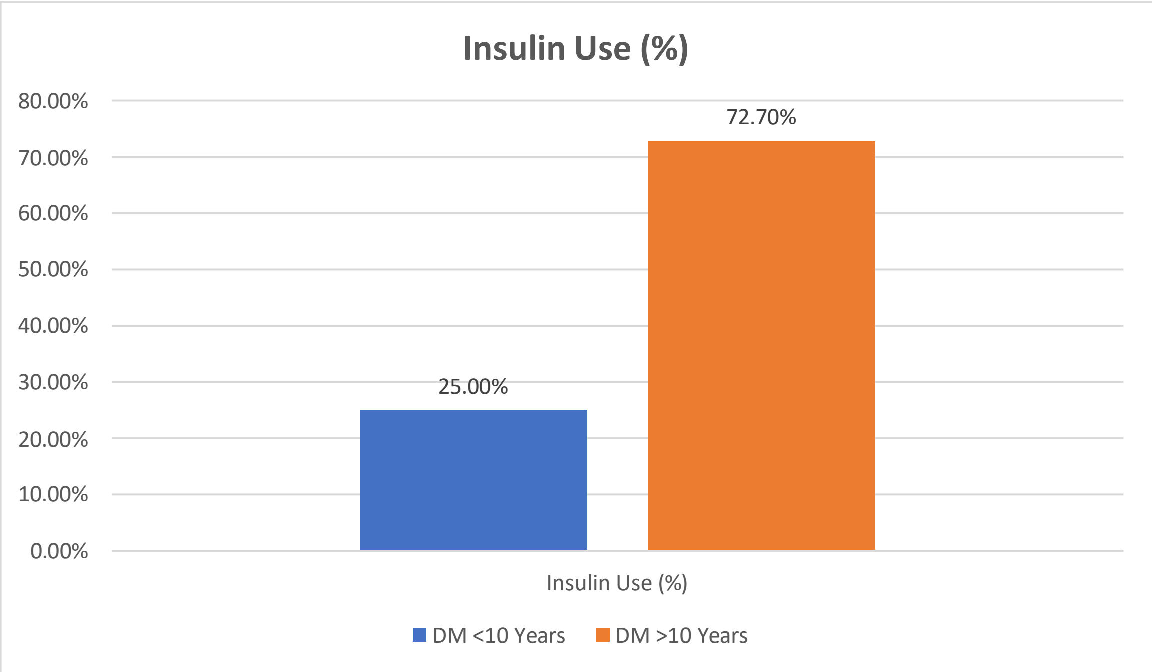

Figure 18: Insulin Usage in Diabetic Patients by Duration of Diabetes

DISCUSSION

DISCUSSION

The results of our study indicate that as people age, the severity of NPDR significantly increases. In particular, the percentage of patients with severe NPDR increased to 13.2% in those aged 61–70 and above 70, whereas just 2.6% of those under 40 had moderate NPDR. This age-related trend correlates with research by Moshfeghi A et al. (2020)[66], who discovered that the prevalence and severity of diabetic retinopathy (DR) increases significantly with age, probably because the condition lasts longer and people are exposed to more hyperglycemic situations over time.

The gender distribution of NPDR severity showed that males had a higher incidence of NPDR (57.9%) than females (42.1%). Severe NPDR was more commonly seen in men than in women (18.4% vs. 10.5%). Research by Nakayama Y, et al. (2021)[67], found that men are more likely to experience severe DR, which is consistent with the study. This could be due to differences in treatment compliance or comorbid conditions between the sexes.

BLOOD PRESSURE, GLYCEMIC CONTROL, AND RENAL FUNCTION

Systolic blood pressure (BP) was seen to be increased as the severity of NPDR progressed; the group with the severe NPDR had the highest BP (129.2 ± 15.3 mmHg). This trend, was statistically significant (p=0.01), aligns with findings from Noroozi M et al. (2024)[68] who showed a link between higher systolic blood pressure and the onset of diabetic retinopathy in people with type II diabetes. The importance of tight blood sugar control was clear, as both post-prandial blood sugar (PPBS) and fasting blood sugar (FBS) levels remained steady across all NPDR severity groups. This echoes research by Chatziralli IP et al. (2018)[69], which emphasized the importance of stable glycaemic control in the management of diabetic complications.

Hemoglobin A1c (HbA1c) levels varied slightly but did not show an upward or a downward trend suggests that HbA1c alone may not be a good indicator of the severity of DR. Chen et al. (2018)[70] agreed, stating that other factors, like blood pressure and the duration of diabetes, might be more significant factors. Blood urea and serum creatinine levels were constant across all the groups, despite minimum rise in serum creatinine in the severe NPDR group, which can be an early sign of diabetic nephropathy, according to a study by Shiferaw WS et al. (2020).[71]

ECHOCARDIOGRAPHIC PARAMETERS

The lack of consistent trends in left ventricular end-diastolic diameter (LVDD) and left atrial (LA) diameter with NPDR severity suggests that myocardial remodelling may not always be clearly correlated with NPDR stages. These findings contrast with those of Chen et al. (2018)[70], who found that left ventricular systolic performance reduced with worsening DR. However, our study found no significant decrease in left ventricular ejection fraction (LVEF) with increasing NPDR severity.

DEMOGRAPHIC CHARACTERISTICS

According to the demographic statistics, the prevalence of DR rises with age, which is especially evident in the groups above 60, when PDR frequency sharply rises. Although statistically insignificant (p=0.12), this tendency is consistent with the findings of Sharif A et al. (2024)[72], who found that the longer duration of diabetes and cumulative damage increase the chance of getting PDR as people age. Research by Kautzky-Willer A et al. (2023)[73] identified no significant gender difference in DR advancement, and the gender distribution did not demonstrate a significant difference in DR prevalence, suggesting that DR affects both males and females similarly.

Clinical and Biochemical Parameters: Clinically, individuals with PDR had systolic blood pressure (BP) that was considerably higher (135.4 ± 18.6 mmHg) than those with a normal fundus (124.3 ± 12.8 mmHg, p=0.03*), indicating a clear link between DR severity and elevated systolic BP. Chaurasia S et al. (2023)[74] found that hypertension is a significant risk factor for the advancement of DR because it affects microvascular integrity, which supports this conclusion.

Fasting blood sugar (FBS) and postprandial blood sugar (PPBS), which are indicators of glucose management, gradually deteriorated from normal fundus to PDR. The investigations of Akil H et al. (2022)[75], which highlighted the need of strict glycaemic management in avoiding DR, were supported by the concomitant rise in HbA1c levels, which showed poor long-term glucose control as a strong predictor of DR severity. Blood urea and serum creatinine, are the two indicators of renal function that increased in proportion to the severity of DR, suggesting the advancement of both retinopathy and nephropathy. According to Kulkarni A et al. (2024)[76], this co-progression illustrates the systemic character of diabetes problems, highlighting the necessity of comprehensive diabetes treatment to prevent both renal and retinal issues.

As diabetic retinopathy progresses from normal fundus to non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR), data from Table 7 show a progressive increase in left ventricular end-diastolic diameter (LVDD) and left ventricular systolic diameter (LVDS). In particular, as DR advances, LVDD and LVDS rise noticeably, with PDR showing the greatest readings. These results are consistent with Chen Y. et al.'s work from 2023 [77], which showed that microvascular damage brought on by diabetes plays a role in alterations in heart remodelling.

Additionally, the same groups' left ventricular ejection fraction (LVEF) significantly decreases, indicating a decline in cardiac function with advanced stages of DR. These findings support those of Tan Y et al. (2020)[78], who reported that long-term uncontrolled blood glucose levels frequently result in diabetic cardiomyopathy.

ECG CHANGES AND QUALITY OF LIFE

The frequency of ECG anomalies is seen in Table 8 and rises dramatically with the severity of DR: 30% in normal fundus, 62.5% in NPDR, and 90% in PDR. Given that cardiovascular illness is a prevalent comorbidity among people with severe diabetes and DR, this trend ($p<0.001$*) indicates that cardiac electrical activity is progressively compromised as DR worsens. This validates the results of Becker S et al. (2020)[79], who found that individuals with more severe diabetic problems had a greater incidence of ventricular arrhythmias.

From normal fundus to PDR, there is a noticeable drop in quality of life. 40% of patients with PDR reported having a high quality of life, compared to 70% of those with a normal fundus. According to Mahobia A et al. (2021)[80], who highlighted the psychosocial effects of chronic diabetes complications on patients' general well-being, this decline in quality of life may be linked to the rise in symptom burden and the psychological toll of having several chronic conditions.

ASSOCIATION AMONG TREATMENT MODALITIES

As DR progresses, Table 9 shows a notable transition from oral hypoglycemic medications (OHAs) to insulin treatment, which is more common in the PDR group (75% on insulin). The findings of Meece J et al. (2016)[81], who pointed out that more severe diabetes problems necessitate rigorous glycaemic management, frequently requiring the administration of insulin, are supported by this trend, which is statistically significant ($p<0.001$). The increase from OHAs to insulin as DR advances appears to account for the bulk of variances, as the disparities in the combination therapy and no treatment groups did not achieve statistical significance despite these changes.

ASSOCIATION AMONG NPDR SUBGROUP ANALYSIS

As NPDR severity rises from mild to severe, Table 10 shows a distinct gradient in clinical parameters. Notably, there are notable declines in left ventricular ejection fraction (LVEF), systolic blood pressure, and HbA1c levels, all of which are associated with more severe NPDR. These results are consistent with those of Xiao X et al. (2025)[82], who emphasised that systemic deteriorations including elevated blood pressure and worsened glycaemic management frequently accompany deteriorating DR. Furthermore, the multiple effects of diabetes on general health are shown by the rise in ECG abnormalities and decrease in quality of life associated with more severe NPDR.

ASSOCIATION OF DIABETES DURATION

According to Table 11, people who have had diabetes for more than ten years have retinopathy that is noticeably more severe, cardiac remodelling that is more noticeable, and nephropathy indicators that are greater than those who have had diabetes for less than ten years. The research by Poonoosamy J et al. (2023)[83]showed that continuous exposure to hyperglycemia contributes to a variety of microvascular and macrovascular problems, and these relationships are statistically significant. Notably, people with diabetes who have had the disease for more than ten years require insulin significantly more often, which emphasises the necessity for more intensive therapy.

LIMITATIONS OF STUDY

1. **Cross-Sectional Design**: One of the primary limitations is the cross-sectional nature of the study. This design restricts the ability to infer causality between diabetic retinopathy, myocardial remodeling, and quality of life changes. Longitudinal studies would be more effective in tracking these changes over time and establishing a causal relationship.
2. **Sample Size and Variety:** The study group was fairly small, so the findings might not apply to everyone. Most participants came from a particular area and had similar socioeconomic backgrounds, which may not capture the diversity of people overall.
3. **Self-Reported Quality of Life:** Quality of life assessment was done by self-reported measures, but people's feelings about their lives can differ a lot and might be affected by things like their mood, support from family, or personal ways of dealing with challenges, which were not fully explored.
4. **Lack of detailed Lifestyle Information:** The study did not look into lifestyle habits like diet, exercise, or their compliance for medications. These things can play a major role in how diabetic retinopathy and heart health progress, which play a crucial role in understanding the correlation.
5. **Single Centre Study:** Since the study was conducted at just one hospital, the results are limited to that particular hospital itself. Hence a multicentred approach could have gotten us stronger and more varied data.
6. **Confounding Variables:** Potential confounding variables such as genetic predispositions and undiagnosed conditions could affect the outcomes. Not accounting for these could effect the results and interpretations.
7. **Analysis Methods:** The analytical methods used provided significant insights but we might benefit from more advanced statistical techniques that handle interactions and non-linear relationships better.

CONCLUSION

CONCLUSION

- **Cardiac Remodeling and DR Progression**: As diabetic retinopathy (DR) progressed from normal fundus to proliferative diabetic retinopathy (PDR), the study found that cardiac remodelling markers, including left ventricular diameters, dramatically increased and left ventricular ejection fraction (LVEF) decreased. This suggests a bidirectional relationship where worsening DR negatively impacts cardiac function, necessitating cardiovascular monitoring in diabetic patients.

- **ECG Abnormalities and Cardiovascular Risk**: As DR worsened, there was a noticeable rise in ECG abnormalities, particularly in PDR patients. This underscores the elevated cardiovascular risk associated with severe retinopathy, highlighting the need for integrated ophthalmic and cardiac care in diabetes management.

- **Shift in Treatment Modalities**: The study revealed a shift from oral hypoglycemic agents (OHAs) to insulin therapy as DR severity increased, reflecting the growing challenge in glycemic control. Early insulin intervention could help prevent the progression to severe stages of DR and associated myocardial risks.

- **Quality of Life Decline**: The quality of life drastically decreased as DR severity rose because of the increasing heart symptoms, eyesight loss, and cumulative load of managing diabetes. This emphasizes the importance of comprehensive care, including mental health support, for diabetes patients.

- **Impact of Diabetes Duration**: There was a clear correlation between the length of diabetes and poorer outcomes in terms of nephropathy indicators, cardiac remodelling, and DR severity. Improving patient outcomes and reducing long-term consequences need early identification and careful blood glucose control.

SUMMARY

SUMMARY

Heart Changes Linked to DR Worsening: As diabetic retinopathy (DR) advanced to proliferative diabetic retinopathy (PDR), heart remodelling worsened significantly. Markers like larger left ventricular diameters increased, while left ventricular ejection fraction (LVEF) reduced. This correlation suggests that worsening DR worsens the heart function, highlighting the need for regular cardiac evaluation in diabetic patients.

Rising ECG Issues and Heart Risks: Especially in PDR cases, a higher rate of ECG abnormalities was seen. This points to a greater risk of heart disease in patients with advanced retinopathy, stressing the importance of combining eye and heart care in diabetes treatment plans.

Switch to Insulin Therapy: As DR changes progressed , the patients increasingly shifted from oral medications to insulin to manage blood sugar levels. This shift reflects the difficulty of controlling diabetes as the disease progresses. Starting insulin treatment earlier might help slow down the progression of DR and reduce cardiomyopathy changes.

Declining Quality of Life: Worsening DR led to a sharp decline in quality of life, driven by heart problems, vision loss, and the ongoing burden of managing diabetes. This highlights the need for holistic approach, including mental health support, to help patients cope better.

Longer Diabetes, Worse Outcomes: Longer the duration of diabetes, the worse their outcomes were for kidney issues, cardiac remodelling, and DR severity. Early diagnosis and strict blood sugar control are crucial to improving long-term health and preventing complications.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. *Standards of Medical Care in Diabetes—2022* Abridged for Primary Care Providers. Clinical Diabetes. 2022 Jan 1;40(1):10–38.

2. Wang W, Lo ACY. Diabetic Retinopathy: Pathophysiology and Treatments. Int J Mol Sci. 2018 Jun 20;19(6):1816.

3. Chong CR, Clarke K, Levelt E. Metabolic remodelling in diabetic cardiomyopathy. Cardiovasc Res. 2017 Mar 15;113(4):422–30.

4. ElSayed NA, Aleppo G, Bannuru RR, Bruemmer D, Collins BS, Ekhlaspour L, et al. 2. Diagnosis and Classification of Diabetes: *Standards of Care in Diabetes—2024*. Diabetes Care. 2024 Jan 1;47(Supplement_1):S20–42.

5. Sun H, Saeedi P, Karuranga S, Pinkepank M, Ogurtsova K, Duncan BB, et al. IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. Diabetes Res Clin Pract. 2022 Jan;183:109119.

6. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. Diabetes Res Clin Pract. 2019 Nov;157:107843.

7. Ritchie ND, Sauder KA, Kaufmann PG, Perreault L. Patient-Centered Goal-Setting in the National Diabetes Prevention Program: A Pilot Study. Diabetes Care. 2021 Nov 1;44(11):2464–9.

8. Chen GC, Qi Q. Lifestyle for the prevention of type 2 diabetes: what is the role of genetic risk information? Am J Clin Nutr. 2020 Mar;111(3):491–2.

9. Standl E, Khunti K, Hansen TB, Schnell O. The global epidemics of diabetes in the 21st century: Current situation and perspectives. Eur J Prev Cardiol. 2019 Dec 26;26(2_suppl):7–14.

10. Filla LA, Edwards JL. Metabolomics in diabetic complications. Mol Biosyst. 2016;12(4):1090–105.

11. Eid S, Sas KM, Abcouwer SF, Feldman EL, Gardner TW, Pennathur S, et al. New insights into the mechanisms of diabetic complications: role of lipids and lipid metabolism. Diabetologia. 2019 Sep 25;62(9):1539–49.

12. Rask-Madsen C, King GL. Vascular Complications of Diabetes: Mechanisms of Injury and Protective Factors. Cell Metab. 2013 Jan;17(1):20–33.

13. Chawla A, Chawla R, Jaggi S. Microvasular and macrovascular complications in diabetes mellitus: Distinct or continuum? Indian J Endocrinol Metab. 2016;20(4):546.

14. Wong TY, Tan TE. The Diabetic Retinopathy ―Pandemic‖ and Evolving Global Strategies: The 2023 Friedenwald Lecture. Investigative Opthalmology & Visual Science. 2023 Dec 28;64(15):47.

15. Wu L. Classification of diabetic retinopathy and diabetic macular edema. World J Diabetes. 2013;4(6):290.

16. Klein R, Klein BEK, Moss SE, Davis MD, DeMets DL. The Wisconsin Epidemiologic Study of Diabetic Retinopathy. Ophthalmology. 1984 Dec;91(12):1464–74.

17. Kusuhara S, Fukushima Y, Ogura S, Inoue N, Uemura A. Pathophysiology of Diabetic Retinopathy: The Old and the New. Diabetes Metab J. 2018;42(5):364.

18. Gupta N, Mansoor S, Sharma A, Sapkal A, Sheth J, Falatoonzadeh P, et al. Diabetic Retinopathy and VEGF. Open Ophthalmol J. 2013 Feb 1;7(1):4–10.

19. Nellaiappan K, Preeti K, Khatri DK, Singh SB. Diabetic Complications: An Update on Pathobiology and Therapeutic Strategies. Curr Diabetes Rev. 2022 Jan;18(1).

20. Al-Kharashi AS. Role of oxidative stress, inflammation, hypoxia and angiogenesis in the development of diabetic retinopathy. Saudi Journal of Ophthalmology. 2018 Oct;32(4):318–23.

21. Shah S, Feher M, McGovern A, Sherlock J, Whyte MB, Munro N, et al. Diabetic retinopathy in newly diagnosed Type 2 diabetes mellitus: Prevalence and predictors of progression; a national primary network study. Diabetes Res Clin Pract. 2021 May;175:108776.

22. Dinesen S, Stokholm L, Subhi Y, Henriksen JE, Savarimuthu TR, Peto T, et al. Retinal main vessel calibers and systemic markers for long-term development of proliferative diabetic retinopathy. Acta Ophthalmol. 2024 Jun 7;102(4):448–54.

23. Jia G, Hill MA, Sowers JR. Diabetic Cardiomyopathy. Circ Res. 2018 Feb 16;122(4):624–38.

24. Pan KL, Hsu YC, Chang ST, Chung CM, Lin CL. The Role of Cardiac Fibrosis in Diabetic Cardiomyopathy: From Pathophysiology to Clinical Diagnostic Tools. Int J Mol Sci. 2023 May 11;24(10):8604.

25. Bugger H, Abel ED. Molecular mechanisms of diabetic cardiomyopathy. Diabetologia. 2014 Apr 30;57(4):660–71.

26. Binu AJ, Kapoor N. Understanding Diabetic Cardiomyopathy: Insulin Resistance and Beyond. Heart Int. 2024 Dec 19;18(2):7–13.

27. Naruke T, Hashimura-Kakogawa M, Suzuki Y, Ikeda Y, Kitasato L, Nabeta T, et al. The Role of Esaxerenone in the Continuum of Heart Failure With Preserved Ejection Fraction: Insights From a Prospective Observational Study. Clin Cardiol. 2025 Apr 24;48(4).

28. Jia G, DeMarco VG, Sowers JR. Insulin resistance and hyperinsulinaemia in diabetic cardiomyopathy. Nat Rev Endocrinol. 2016 Mar 18;12(3):144–53.

29. Negishi K. Echocardiographic feature of diabetic cardiomyopathy: where are we now? Cardiovasc Diagn Ther. 2018 Feb;8(1):47–56.

30. Hassooni FRH, Aljubori E, Abbas GMA, Dheyab AYD, Abdulatif MQA, Sadeq Sadeq MA, et al. Advances in diabetic cardiomyopathy: current and potential management strategies and emerging biomarkers. Annals of Medicine & Surgery. 2025 Jan;87(1):224–33.

31. Seferović PM, Paulus WJ. Clinical diabetic cardiomyopathy: a two-faced disease with restrictive and dilated phenotypes. Eur Heart J. 2015 Jul 14;36(27):1718–27.

32. Radovits T, Korkmaz S, Mátyás C, Oláh A, Németh BT, Páli S, et al. An Altered Pattern of Myocardial Histopathological and Molecular Changes Underlies the Different Characteristics of Type-1 and Type-2 Diabetic Cardiac Dysfunction. J Diabetes Res. 2015;2015:1–12.

33. Chen X, Xie N, Feng L, Huang Y, Wu Y, Zhu H, et al. Oxidative stress in diabetes mellitus and its complications: From pathophysiology to therapeutic strategies. Chin Med J (Engl). 2025 Jan 5;138(1):15–27.

34. Nathan DM. The Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Study at 30 Years: Overview. Diabetes Care. 2014 Jan 1;37(1):9–16.

35. Funck KL, Knudsen JS, Hansen TK, Thomsen RW, Grove EL. Real-world use of cardioprotective glucose-lowering drugs in patients with type 2 diabetes and cardiovascular disease: A Danish nationwide cohort study, 2012 to 2019. Diabetes Obes Metab. 2021 Feb 17;23(2):520–9.

36. Kramer CM, Barkhausen J, Bucciarelli-Ducci C, Flamm SD, Kim RJ, Nagel E. Standardized cardiovascular magnetic resonance imaging (CMR) protocols: 2020 update. Journal of Cardiovascular Magnetic Resonance. 2020 Jan;22(1):17.

37. Lopez-Candales A, Hernandez-Suarez DF. Strain Imaging Echocardiography: What Imaging Cardiologists Should Know. Curr Cardiol Rev. 2017 Mar 1;13(2).

38. Nakanishi R, Hashimoto H, Ikeda T. Improving Quality of Clinical Diagnosis Report with Single-Photon Emission Computed Tomography Myocardial Perfusion Imaging and Coronary Computed Tomography Angiography. Ann Nucl Cardiol. 2020;6(1):86–90.

39. Puntmann VO, Peker E, Chandrashekhar Y, Nagel E. T1 Mapping in Characterizing Myocardial Disease. Circ Res. 2016 Jul 8;119(2):277–99.

40. Zare F, Ameri H, Madadizadeh F, Reza Aghaei M. Health-related quality of life and its associated factors in patients with type 2 diabetes mellitus. SAGE Open Med. 2020 Jan 26;8.

41. Teli M, Thato R, Rias YA. Predicting Factors of Health-Related Quality of Life Among Adults With Type 2 Diabetes: A Systematic Review. SAGE Open Nurs. 2023 Jan 10;9.

42. Abbas Q, Latif S, Ayaz Habib H, Shahzad S, Sarwar U, Shahzadi M, et al. Cognitive behavior therapy for diabetes distress, depression, health anxiety, quality of life and treatment adherence among patients with type-II diabetes mellitus: a randomized control trial. BMC Psychiatry. 2023 Feb 3;23(1):86.

43. Mousavizadeh SN, Banazadeh Z. Loss of Time in the Treatment Adherence Process: A Qualitative Study in a Sample of Iranian People with Diabetes. J Med Life. 2020 Jul;13(3):293–9.

44. Nicolucci A, Kovacs Burns K, Holt RIG, Comaschi M, Hermanns N, Ishii H, et al. Diabetes Attitudes, Wishes and Needs second study (DAWN2™): Cross-national benchmarking of diabetes-related psychosocial outcomes for people with diabetes. Diabetic Medicine. 2013 Jul 23;30(7):767–77.

45. Selim AJ, Rothendler JA, Qian SX, Bailey HM, Kazis LE. The History and Applications of the Veterans RAND 12-Item Health Survey (VR-12). Journal of Ambulatory Care Management. 2022 Jul;45(3):161–70.

46. Devlin N, Pan T, Kreimeier S, Verstraete J, Stolk E, Rand K, et al. Valuing EQ-5D-Y: the current state of play. Health Qual Life Outcomes. 2022 Dec 6;20(1):105.

47. Bujang MA, Adnan TH, Mohd Hatta NKB, Ismail M, Lim CJ. A Revised Version of Diabetes Quality of Life Instrument Maintaining Domains for Satisfaction, Impact, and Worry. J Diabetes Res. 2018 Jul 22;2018:1–10.

48. AlOtaibi AA, Almesned M, Alahaideb TM, Almasari SM, Alsuwayt SS. Assessment of diabetes-related distress among type 2 diabetic patients, Riyadh, Saudi Arabia. J Family Med Prim Care. 2021 Sep;10(9):3481–9.

49. Chung YR, Park SJ, Moon KY, Choi SA, Lim HS, Park SW, et al. Diabetic retinopathy is associated with diastolic dysfunction in type 2 diabetic patients with non-ischemic dilated cardiomyopathy. Cardiovasc Diabetol. 2017 Dec 6;16(1):82.

50. Seo H, Park SJ, Song M. Diabetic Retinopathy (DR): Mechanisms, Current Therapies, and Emerging Strategies. Cells. 2025 Mar 4;14(5):376.

51. Chawla S, Trehan S, Chawla A, Jaggi S, Chawla R, Kumar V, et al. Relationship between diabetic retinopathy microalbuminuria and other modifiable risk factors. Prim Care Diabetes. 2021 Jun;15(3):567–70.

52. Lingineni VB, Mangudkar S, Gokhale VS, Malik S, Yadav P. Linking Diabetic Retinopathy Severity to Coronary Artery Disease Risk Factors in Type 2 Diabetic Patients. Cureus. 2024 Jul 21;

53. Hsu CY, Lee CM, Chou KY, Lee CY, Chen HC, Chiou JY, et al. The Association of Diabetic Retinopathy and Cardiovascular Disease: A 13-Year Nationwide Population-Based Cohort Study. Int J Environ Res Public Health. 2021 Jul 30;18(15):8106.

54. Zhao Y, Singh RP. The role of anti-vascular endothelial growth factor (anti-VEGF) in the management of proliferative diabetic retinopathy. Drugs Context. 2018 Aug 13;7:1–10.

55. Cyr AR, Huckaby L V., Shiva SS, Zuckerbraun BS. Nitric Oxide and Endothelial Dysfunction. Crit Care Clin. 2020 Apr;36(2):307–21.

56. Aguilar D, Hallman DM, Piller LB, Klein BEK, Klein R, Devereux RB, et al. Adverse association between diabetic retinopathy and cardiac structure and function. Am Heart J. 2009 Mar;157(3):563–8.

57. Herman WH, Ye W, Griffin SJ, Simmons RK, Davies MJ, Khunti K, et al. Early Detection and Treatment of Type 2 Diabetes Reduce Cardiovascular Morbidity and Mortality: A Simulation of the Results of the Anglo-Danish-Dutch Study of Intensive Treatment in People

With Screen-Detected Diabetes in Primary Care (ADDITION-Europe). Diabetes Care. 2015 Aug 1;38(8):1449–55.

58. Zhen Z, Chen Y, Shih K, Liu JH, Yuen M, Wong DSH, et al. Altered myocardial response in patients with diabetic retinopathy: an exercise echocardiography study. Cardiovasc Diabetol. 2015 Dec 18;14(1):123.

59. Ernande L, Bergerot C, Girerd N, Thibault H, Davidsen ES, Gautier Pignon-Blanc P, et al. Longitudinal Myocardial Strain Alteration Is Associated with Left Ventricular Remodeling in Asymptomatic Patients with Type 2 Diabetes Mellitus. Journal of the American Society of Echocardiography. 2014 May;27(5):479–88.

60. Ben ÂJ, Souza CF de, Locatelli F, Rosses APO, Szortika A, Araujo AL de, et al. Health-related quality of life associated with diabetic retinopathy in patients at a public primary care service in southern Brazil. Arch Endocrinol Metab. 2020 Mar 30;

61. Zayed MG, Karsan W, Peto T, Saravanan P, Virgili G, Preiss D. Diabetic Retinopathy and Quality of Life. JAMA Ophthalmol. 2024 Mar 1;142(3):199.

62. Poonoosamy J, Lopes P, Huret P, Dardari R, Penfornis A, Thomas C, et al. Impact of Intensive Glycemic Treatment on Diabetes Complications—A Systematic Review. Pharmaceutics. 2023 Jun 22;15(7):1791.

63. Horton WB, Barrett EJ. Microvascular Dysfunction in Diabetes Mellitus and Cardiometabolic Disease. Endocr Rev. 2021 Jan 28;42(1):29–55.

64. Luneva EB, Vasileva AA, Karelkina E V., Boyarinova MA, Mikhaylov EN, Ryzhkov A V., et al. Simple Predictors for Cardiac Fibrosis in Patients with Type 2 Diabetes Mellitus: The Role of Circulating Biomarkers and Pulse Wave Velocity. J Clin Med. 2022 May 18;11(10):2843.

65. Saucedo L, Pfister IB, Schild C, Garweg JG. Association of inflammation-related markers and diabetic retinopathy severity in the aqueous humor, but not serum of type 2 diabetic patients. PLoS One. 2023 Oct 26;18(10):e0293139.

66. Moshfeghi A, Garmo V, Sheinson D, Ghanekar A, Abbass I. <p>Five-Year Patterns of Diabetic Retinopathy Progression in US Clinical Practice</p>. Clinical Ophthalmology. 2020 Oct;Volume 14:3651–9.

67. Nakayama Y, Yamaguchi S, Shinzato Y, Okamoto S, Millman JF, Yamashiro K, et al. Retrospective exploratory analyses on gender differences in determinants for incidence and progression of diabetic retinopathy in Japanese patients with type 2 diabetes mellitus. Endocr J. 2021;68(6):655–69.

68. Noroozi M. Visit-to-visit variability of blood pressure and risk of diabetic retinopathy: a systematic review and meta-analysis. Am J Cardiovasc Dis. 2024;14(5):281–94.

69. Chatziralli IP. The Role of Glycemic Control and Variability in Diabetic Retinopathy. Diabetes Therapy. 2018 Feb 30;9(1):431–4.

70. Shiferaw WS, Akalu TY, Desta M, Kassie AM, Petrucka PM, Assefa HK, et al. Glycated hemoglobin A1C level and the risk of diabetic retinopathy in Africa: A systematic review and meta-analysis. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. 2020 Nov;14(6):1941–9.

71. Rajalakshmi R, Shanthi Rani CS, Venkatesan U, Unnikrishnan R, Anjana RM, Jeba Rani S, et al. Correlation between markers of renal function and sight-threatening diabetic retinopathy in type 2 diabetes: a longitudinal study in an Indian clinic population. BMJ Open Diabetes Res Care. 2020 May 31;8(1):e001325.

72. Sharif A, Smith DR, Hellgren K, Jendle J. Diabetic retinopathy among the elderly with type 2 diabetes: A Nationwide longitudinal registry study. Acta Ophthalmol. 2024 Sep 10;102(6).

73. Kautzky-Willer A, Leutner M, Harreiter J. Sex differences in type 2 diabetes. Diabetologia. 2023 Jun 10;66(6):986–1002.

74. Chaurasia S, Thool AR, Ansari KK, Saifi AI. Advancement in Understanding Diabetic Retinopathy: A Comprehensive Review. Cureus. 2023 Nov 21;

75. Akil H, Burgess J, Nevitt S, Harding SP, Alam U, Burgess P. Early Worsening of Retinopathy in Type 1 and Type 2 Diabetes After Rapid Improvement in Glycaemic Control: A Systematic Review. Diabetes Therapy. 2022 Jan 20;13(1):1–23.

76. Kulkarni A, Thool AR, Daigavane S. Understanding the Clinical Relationship Between Diabetic Retinopathy, Nephropathy, and Neuropathy: A Comprehensive Review. Cureus. 2024 Mar 21;

77. Chen Y, Li M, Wang Y, Fu J, Liu X, Zhang Y, et al. Association between Severity of Diabetic Retinopathy and Cardiac Function in Patients with Type 2 Diabetes. J Diabetes Res. 2023 Jun 7;2023:1–10.

78. Tan Y, Zhang Z, Zheng C, Wintergerst KA, Keller BB, Cai L. Mechanisms of diabetic cardiomyopathy and potential therapeutic strategies: preclinical and clinical evidence. Nat Rev Cardiol. 2020 Sep 20;17(9):585–607.

79. Becker S, Carroll LS, Vinberg F. Diabetic photoreceptors: Mechanisms underlying changes in structure and function. Vis Neurosci. 2020 Oct 6;37:E008.

80. Mahobia A, Sahoo SR, Maiti N, Sathyanarayanan R, Aravinth R, Pandraveti RR, et al. Diabetic Retinopathy and Its Effect on Quality of Life. J Pharm Bioallied Sci. 2021 Nov;13(Suppl 2):S1365–8.

81. Meece J. Basal Insulin Intensification in Patients with Type 2 Diabetes: A Review. Diabetes Therapy. 2018 Jun 24;9(3):877–90.

82. Xiao X, Zhao J, Lin S, Yang Y, Li W, Zhou Y, et al. Relationships Between Retinal Vascular Characteristics and Systemic Indicators in Patients With Diabetes Mellitus. Invest Ophthalmol Vis Sci. 2025 Apr 24;66(4):72.

83. Poonoosamy J, Lopes P, Huret P, Dardari R, Penfornis A, Thomas C, et al. Impact of Intensive Glycemic Treatment on Diabetes Complications—A Systematic Review. Pharmaceutics. 2023 Jun 22;15(7):1791.

84. Yu W, Zhang X, Li Y, Ma Q, Zhang L, Liu Y, et al. Diabetic retinopathy and cardiovascular disease: a literature review. Diabetes Metab Syndr Obes. 2023;16:4247-61. doi:10.2147/DMSO.S438111.

85. Arcadu, F., Benmansour, F., Maunz, A. *et al.* Deep learning algorithm predicts diabetic retinopathy progression in individual patients. *npj Digit. Med.* 2, 92 (2019).

ANNEXURE

ANNEXURE-1

CASE PROFORMA	
Name:	Case No:
Age:	Date:
Sex:	1P No:
Occupation:	DOS:
Address:	

Chief complaints:

Past history:

DM / HTN / BA / Epilepsy

Family history:

Personal history:

Appetite – Sleep – Bowel –

Diet – Habits – Bladder –

GPE:

Pallor / Edema /Icterus / Cyanosis / Clubbing / Lymphadenopathy

Vital signs:

HR – c) RR –

BP – d) Temp –

Systemic examination:

CVS – c. RS –

PA – d. CNS –

OCULAR EXAMINATION		
	RE	**LE**
Head Posture **Ocular Posture** **Facial Symmetry**		
Ocular Movements		
Visual Acuity		
Anterior Segment		
Fundus (IDO)		
Lab Investigations FBS PPBS HbA1c Blood urea Serum Creatinine Urine		
ECG 10. **2D ECHO** Ejection fraction – LVH – Left ventricular strain pattern – Left atrial enlargement – Posterior wall thickness in diastole – Inter-ventricular septal thickness in diastole –		

ANNEXURE-II

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Case no:

IP no:

TITLE: "CORRELATION OF DIABETIC RETINOPATHY WITH MYOCARDIAL REMODELING AND QUALITY OF LIFE IN TYPE II DIABETES MELLITUS PATIENTS"

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

I understand the purpose of this study, the risks and benefits of the technique 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 the participation from this study at any time and this will not change the future care.

Participation in this study does not involve any financial burden to me.

Name	Signature	Date	Time
Patient:			
Witness:			
Primary Investigator/ Doctor:			

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನಾ ಸಂಸ್ಥೆ, ಟಮಕ, ಕೋಲಾರ - 563101.

ತಿಳಿವಳಿಕೆ ಸಮ್ಮತಿ ನಮೂನೆ

ಶೀರ್ಷಿಕೆ : " ಮಯೋಕಾರ್ಡಿಯಲ್ ಮರುರೂಪಿಸುವಿಕೆ ಮತ್ತು ಟೈಪ್ II ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ ರೋಗಿಗಳಲ್ಲಿ ಜೀವನದ ಗುಣಮಟ್ಟದೊಂದಿಗೆ ಡಯಾಬಿಟಿಕ್ ರೆಟಿನೋಪತಿಯ ಪರಸ್ಪರ ಸಂಬಂಧ "

ಈ ಸಂಶೋಧನೆಗೆ ರೋಗಿಯ ಗುರುತಿನ ಸಂಖ್ಯೆ:

ಐಪಿ ಸಂಖ್ಯೆ:

ಅಂಗೀಕರಿಸಿದ ನಾನು, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ಈ ಸಮ್ಮತಿಯ ರೂಪದಲ್ಲಿ ವಿವರಿಸಿರುವಂತೆ ನನ್ನ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನು ದೃಢೀಕರಿಸುತ್ತೇನೆ.

ನಾನು ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ತಂತ್ರಗಳ ಅಪಾಯಗಳು ಮತ್ತು ಪ್ರಯೋಜನಗಳನ್ನು ಮತ್ತು ಅಧ್ಯಯನದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿದ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವ ಮಾಹಿತಿಯ ಗೌಪ್ಯತೆಗೆ ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳನ್ನು ಕುರಿತು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ ಮತ್ತು ನನ್ನ ತೃಪ್ತಿಗೆ ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರ ನೀಡಲಾಗಿದೆ.

ಈ ಸಂಶೋಧನೆಯಿಂದ ಹೊರಬರುವ ಮಾಹಿತಿಯನ್ನು ವೈದ್ಯರು ಯಾವುದೇ ಜರ್ನಲ್ನಲ್ಲಿ ಅಥವಾ ಕಾನ್ಫರೆನ್ಸ್ನಲ್ಲಿ ಪ್ರಕಟಿಸಲು ಅನುಮತಿ ಸೂಚಿಸಿರುತ್ತೇನೆ

ನಾನು ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಲು ಮುಕ್ತವಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ನನ್ನ ಮುಂದಿನ ಕಾಳಜಿಯನ್ನು ಬದಲಿಸುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯ ಭಾಗವಹಿಸುವಿಕೆ ನನಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಹೊರೆ ಒಳಗೊಂಡಿರುವುದಿಲ್ಲ.

ಹೆಸರು	ಸಹಿ	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿಯ:			
ಸಾಕ್ಷಿ 1:			
ಸಾಕ್ಷಿ 2:			
ಪ್ರಾಥಮಿಕ ತನಿಖೆದಾರ / ಡಾಕ್ಟರ್:			

14

ANNEXURE-III

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

PATIENT INFORMATION SHEET

TITLE: "CORRELATION OF DIABETIC RETINOPATHY WITH CARDIAC REMODELLING AND QUALITY OF LIFE IN PATIENTS WITH TYPE II DIABETIES MELLITUS"

This information is to help you understand the purpose of the study titled ―Correlation of Diabetic Retinopathy and Cardiac Remodeling in Type II Diabetes Mellitus‖. As you're invited to take part voluntarily in this research study, it is important that you read and understand the purpose, procedure, benefits and discomforts of the study.

Diabetes mellitus affects not only the heart, kidneys, brain, and large arteries but also the eyes. Early detection of these adverse cardiac manifestations would help identify patients at risk of future development of heart failure for more intensive treatment.

The eye is the only organ where the blood vessels can be observed directly. Retinal arterioles are similar to blood vessels in the heart and Brain regarding the make and function. The evaluation of retinal circulation provides further information of the changes in the microvasculature in the body, which may provide additional information about the risk associated in the heart and brain blood vessels in diabetic patients.

Absolutely no risks are associated with the various investigations to be done which are Random Blood Sugar, Fasting Blood Sugar, Post Prandial Blood Sugar, HbA1c, retinal examination, Electrocardiography & 2D Echocardiography.

There is no compulsion to participate in this study and will not change the final outcome of your eye condition. You may refuse to take part in the study or you may stop your participation in the study at any time, without a penalty or loss of any benefits to which you were otherwise entitled before taking part in this study. However, patients in the future may benefit as a result of knowledge gained from this study.

CONFIDENTIALITY

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. All information collected from you will be strictly confidential and will not be disclosed to any outsider except if it is required by the law. The information collected will be used only for research. This information will not reveal your identity and the original records may be reviewed by your doctor or ethics review board. This study seeks ethical committee approval and will be started only after their formal approval.

For further information, /clarification please contact the below mentioned resident at Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar – 563101.

CONTACT DETAILS:

DR. ANUNITHA RAYAPURAJU, MBBS, (MS)

1st Year Resident

Department of Ophthalmology,

SDUMC, Kolar – 563101

Contact no: 9711156577

Mail ID: ranunitha10@gmail.com

ಶೀರ್ಷಿಕೆ : "ಮಯೋಕಾರ್ಡಿಯಲ್ ಮರುರೂಪಿಸುವಿಕೆ ಮತ್ತು ಟೈಪ್ II ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ ರೋಗಿಗಳಲ್ಲಿ ಜೀವನದ ಗುಣಮಟ್ಟದೊಂದಿಗೆ ಡಯಾಬಿಟಿಕ್ ರೆಟಿನೋಪತಿಯ ಪರಸ್ಪರ ಸಂಬಂಧ"

"ಅಸೋಸಿಯೇಶನ್ ಆಫ್ ಹೈಪರ್ಟೆನ್ಸಿವ್ ರೆಟಿನೋಪತಿ ಮತ್ತು ಕಾರ್ಡಿಯಾಕ್ ರಿಮೋಡೆಲಿಂಗ್ ಇನ್ ಸಿಸ್ಟಮಿಕ್ ಹೈಪರ್ ಟೆನ್ಶನ್" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳಲು ಈ ಮಾಹಿತಿಯು ನಿಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ. ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಪಾಲ್ಗೊಳ್ಳಲು ನಿಮ್ಮನ್ನು ಆಹ್ವಾನಿಸಲಾಗಿದೆ, ನೀವು ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಕಾರ್ಯವಿಧಾನ, ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಅನಾನುಕೂಲಗಳನ್ನು ಓದುವುದು ಮತ್ತು ಅರ್ಥಮಾಡಿಕೊಳ್ಳುವುದು ಮುಖ್ಯವಾಗಿದೆ.

ಅಧಿಕ ರಕ್ತದೊತ್ತಡವು ಹೃದಯ, ಮೂತ್ರಪಿಂಡಗಳು, ಮೆದುಳು ಮತ್ತು ದೊಡ್ಡ ಅಪಧಮನಿಗಳ ಮೇಲೆ ಮಾತ್ರವಲ್ಲದೆ ಕಣ್ಣುಗಳ ಮೇಲೂ ಪರಿಣಾಮ ಬೀರುತ್ತದೆ. ಈ ಪ್ರತಿಕೂಲ ಹೃದಯದ ಅಭಿವ್ಯಕ್ತಿಗಳ ಆರಂಭಿಕ ಪತ್ತೆ ಹೆಚ್ಚು ತೀವ್ರವಾದ ಚಿಕಿತ್ಸೆಗಾಗಿ ಹೃದಯಾಘಾತದ ಭವಿಷ್ಯದ ಬೆಳವಣಿಗೆಯ ಅಪಾಯದಲ್ಲಿರುವ ರೋಗಿಗಳನ್ನು ಗುರುತಿಸಲು ಸಹಾಯ ಮಾಡುತ್ತದೆ.

ರಕ್ತನಾಳಗಳು ನೇರವಾಗಿ ವೀಕ್ಷಿಸಬಹುದಾದ ಏಕೈಕ ಅಂಗ ಕಣ್ಣು. ರೆಟಿನಲ್ ಅಪಧಮನಿಗಳು ಮೇಕಪ್ ಮತ್ತು ಕಾರ್ಯಚಟುವಟಿಕೆಗೆ ಸಂಬಂಧಿಸಿದಂತೆ ಹೃದಯ ಮತ್ತು ಮೆದುಳಿನಲ್ಲಿರುವ ರಕ್ತನಾಳಗಳಂತೆಯೇ ಇರುತ್ತವೆ. ರೆಟಿನಲ್ ರಕ್ತಪರಿಚಲನೆಯ ಮೌಲ್ಯಮಾಪನವು ದೇಹದ ಸೂಕ್ಷ್ಮವಸ್ತುವಿನಲ್ಲಿನ ಬದಲಾವಣೆಗಳ ಹೆಚ್ಚಿನ ಮಾಹಿತಿಯನ್ನು ಒದಗಿಸುತ್ತದೆ, ಇದು ಅಧಿಕ ಒತ್ತಡದ ರೋಗಿಗಳಲ್ಲಿ ಹೃದಯ ಮತ್ತು ಮಿದುಳಿನ ರಕ್ತನಾಳಗಳಲ್ಲಿ ಕಂಡುಬರುವ ಅಪಾಯದ ಬಗ್ಗೆ ಹೆಚ್ಚಿನ ಮಾಹಿತಿಯನ್ನು ಒದಗಿಸುತ್ತದೆ.

ಯಾದೃಚ್ಛಿಕ ರಕ್ತ ಸಕ್ಕರೆ, ಉಪವಾಸ ರಕ್ತ ಸಕ್ಕರೆ, ಪ್ರಸವದ ನಂತರ ರಕ್ತ ಸಕ್ಕರೆ, ರೆಟಿನಲ್ ಪರೀಕ್ಷೆ, ಎಲೆಕ್ಟ್ರೋಕಾರ್ಡಿಯೋಗ್ರಫಿ ಮತ್ತು 2 ಡಿ ಇಕೋಕಾರ್ಡಿಯೋಗ್ರಫಿ ಸೇರಿದಂತೆ ವಿವಿಧ ತನಿಖೆಗಳಿಗೆ ಯಾವುದೇ ಅಪಾಯವಿಲ್ಲ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ ಮತ್ತು ನಿಮ್ಮ ಕಣ್ಣಿನ ಸ್ಥಿತಿಯ ಅಂತಿಮ ಫಲಿತಾಂಶವನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಮೊದಲು ನೀವು ಅರ್ಹರಾಗಿದ್ದ ಯಾವುದೇ ಪ್ರಯೋಜನಗಳ ದಂಡ ಅಥವಾ ನಷ್ಟವಿಲ್ಲದೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ನಿಲ್ಲಿಸಬಹುದು. ಆದಾಗ್ಯೂ, ಈ ಅಧ್ಯಯನದಿಂದ ಪಡೆದ ಜ್ಞಾನದ ಪರಿಣಾಮವಾಗಿ ಭವಿಷ್ಯದಲ್ಲಿ ರೋಗಿಗಳು ಪ್ರಯೋಜನ ಪಡೆಯಬಹುದು.

<u>ಗೌಪ್ಯತೆ</u>

ನಿಮ್ಮ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯನ್ನು ಅಧ್ಯಯನ ವೈದ್ಯರು ಮತ್ತು ಸಿಬ್ಬಂದಿ ಗೌಪ್ಯವಾಗಿಡುತ್ತಾರೆ ಮತ್ತು ಸಾರ್ವಜನಿಕವಾಗಿ ಲಭ್ಯವಾಗುವಂತೆ ಮಾಡಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯು ಕಟ್ಟುನಿಟ್ಟಾಗಿ ಗೌಪ್ಯವಾಗಿರುತ್ತದೆ ಮತ್ತು ಕಾನೂನಿನ ಅಗತ್ಯವಿದ್ದಲ್ಲಿ ಹೊರತುಪಡಿಸಿ ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಸಂಗ್ರಹಿಸಿದ

ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಮಾಹಿತಿಯು ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ ಮತ್ತು ಮೂಲ ದಾಖಲೆಗಳನ್ನು ನಿಮ್ಮ ವೈದ್ಯರು ಅಥವಾ ನೈತಿಕ ಪರಿಶೀಲನಾ ಮಂಡಳಿಯು ಪರಿಶೀಲಿಸಬಹುದು. ಈ ಅಧ್ಯಯನವು ನೈತಿಕ ಸಮಿತಿಯ ಅನುಮೋದನೆಯನ್ನು ಬಯಸುತ್ತದೆ ಮತ್ತು ಅವರ ಔಪಚಾರಿಕ ಅನುಮೋದನೆಯ ನಂತರವೇ ಪ್ರಾರಂಭಿಸಲಾಗುವುದು.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ./ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ದಯವಿಟ್ಟು ಕೆಳಗೆ ನಮೂದಿಸಿದ ನಿವಾಸಿಗಳನ್ನು ಶ್ರೀ ದೇವರಾಜ್ ಅರ್ಸ್ ಅಕಾಡೆಮಿ ಅಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ ಅಂಡ್ ರಿಸರ್ಚ್, ತಮಕ, ಕೋಲಾರ - 563101 ನಲ್ಲಿ ಸಂಪರ್ಕಿಸಿ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ

ಡಾ. ಅನುನಿತಾ ರಾಯಪುರರಾಜು, ಎಂ ಬಿ ಬಿ ಎಸ್, (ಎಂ ಎಸ್)
1 ನೇ ವರ್ಷದ ನಿವಾಸಿ
ನೇತ್ರಶಾಸ್ತ್ರ ವಿಭಾಗ,
SDUMC, ಕೋಲಾರ - 563101
ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 9711156577
ಮೇಲ್ ಐಡಿ: ranunitha10@gmail.com

NATIONAL EYE INSTITUTE

VISUAL FUNCTIONING QUESTIONNAIRE – 25 (VFQ-25)

PART 1 - GENERAL HEALTH AND VISION

1. In general, would you say your overall health is: (Circle One)

Excellent..............................1

Very Good 2

Good................................. 3

Fair 4

Poor 5

2. At the present time, would you say your eyesight using both eyes (with glasses or contact lenses, if you wear them) is excellent, good, fair, poor, or very poor or are you completely blind? (Circle One)

Excellent...............................1

Good.................................. 2

Fair 3

Poor 4

Very Poor..............................5

Completely Blind 6

3. How much of the time do you worry about your eyesight? (Circle One)

None of the time 1

A little of the time 2

Some of the time 3

Most of the time 4

All of the time? 5

4. How much pain or discomfort have you had in and around your eyes (for example, burning, itching, or aching)? Would you say it is: (Circle One)

None 1

Mild................................. 2

Moderate 3

Severe, or 4

Very severe? 5

PART 2 - DIFFICULTY WITH ACTIVITIES

The next questions are about how much difficulty, if any, you have doing certain activities wearing your glasses or contact lenses if you use them for that activity.

5. How much difficulty do you have reading ordinary print in newspapers? Would you say you have: (Circle One)

No difficulty at all 1

A little difficulty.. 2

Moderate difficulty..3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this...................6

6. How much difficulty do you have doing work or hobbies that require you to see well up close, such as cooking, sewing, fixing things around the house, or using hand tools? Would you say: (Circle One)

No difficulty at all 1

A little difficulty.. 2

Moderate difficulty..3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this....................6

7. Because of your eyesight, how much difficulty do you have finding something on a crowded shelf? (Circle One)

No difficulty at all 1

A little difficulty.. 2

Moderate difficulty..3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this 6

8. How much difficulty do you have reading street signs or the names of stores? (Circle One)

No difficulty at all... 1

A little difficulty.. 2

Moderate difficulty...3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this 6

9. Because of your eyesight, how much difficulty do you have going down steps, stairs, or curbs in dim light or at night? (Circle One)

No difficulty at all 1

A little difficulty.. 2

Moderate difficulty...3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this 6

10. Because of your eyesight, how much difficulty do you have noticing objects off to the side while you are walking along? (Circle One)

No difficulty at all 1

A little difficulty 2

Moderate difficulty...3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this 6

11. Because of your eyesight, how much difficulty do you have seeing how people react to things you say? (Circle One)

No difficulty at all 1

A little difficulty 2

Moderate difficulty...3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this 6

12. Because of your eyesight, how much difficulty do you have picking out and matching your own clothes? (Circle One)

No difficulty at all 1

A little difficulty... 2

Moderate difficulty..3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this......................6

13. Because of your eyesight, how much difficulty do you have visiting with people in their homes, at parties, or in restaurants ? (Circle One)

No difficulty at all 1

A little difficulty... 2

Moderate difficulty..3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this.................6

14. Because of your eyesight, how much difficulty do you have going out to see movies, plays, or sports events? (Circle One)

No difficulty at all 1

A little difficulty... 2

Moderate difficulty..3

Extreme difficulty 4

Stopped doing this because of your eyesight 5

Stopped doing this for other reasons or not interested in doing this.................6

15. Are you currently driving, at least once in a while? (Circle One)

Yes .... 1 Skip To Q 15c

No....................... 2

15. a) IF NO: Have you never driven a car or have you given up driving? (Circle One)

Never drove .... 1 Skip To Part 3, Q 17

Gave up. 2

15. b) IF YOU GAVE UP DRIVING: Was that mainly because of your eyesight, mainly for some other reason, or because of both your eyesight and other reasons? (Circle One)

Mainly eyesight 1 SkipToPart3,Q17

Mainly other reasons 2 SkipToPart3,Q17

Both eyesight and other reasons 3 SkipToPart3,Q17

15. c) IF CURRENTLY DRIVING: How much difficulty do you have driving during the daytime in familiar places? Would you say you have: (Circle One)

No difficulty at all 1

A little difficulty................................ 2

Moderate difficulty............................3

Extreme difficulty 4

16. How much difficulty do you have driving at night? Would you say you have: (Circle One)

No difficulty at all.. 1

A little difficulty.. 2

Moderate difficulty...3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or are you not interested in doing this. 6

16. A) How much difficulty do you have driving in difficult conditions, such as in bad weather, during rush hour, on the freeway, or in city traffic? Would you say you have: (Circle One)

No difficulty at all.. 1

A little difficulty.. 2

Moderate difficulty..3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or are you not interested in doing this. 6

PART 3: RESPONSES TO VISION PROBLEMS

The next questions are about how things you do may be affected by your vision. For each one, please circle the number to indicate whether for you the statement is true for you all, most, some, a little, or none of the time.

READ CATEGORIES:	All of the time	Most of the time	Some of the time	A little of the time	None of the time
Do you accomplish less than you would like because of your vision?	1	2	3	4	5
Are you limited in how long you can work or do other activities because of your vision?	1	2	3	4	5
How much does pain or discomfort in or around your eyes, for example, burning, itching, or aching, keep you from doing what you'd like to bedoing? Would you say:	1	2	3	4	5

For each of the following statements, please circle the number to indicate whether for you the statement is definitely true, mostly true, mostly false, or definitely false for you or you are not sure.

Sl No	Read Categories:	Definitely true	Mostly true	Not sure	Mostly false	Definitely false
20	I stay home most of the time because of my eyesight.....	1	2	3	4	5
21	I feel frustrated a lot of the time because of my eyesight.	1	2	3	4	5
22	I have much less control over what I do, because of my eyesight	1	2	3	4	5
23	Because of my eyesight, I have to rely too much on what other people tell me.	1	2	3	4	5
24	I need a lot of help from others because of my eyesight.	1	2	3	4	5
25	I worry about doing things that will embarrass myself or others, because of my eyesight	1	2	3	4	5

APPENDIX OF OPTIONAL ADDITIONAL QUESTIONS

SUBSCALE: GENERAL HEALTH

A1. How would you rate your overall health, on a scale where zero is as bad as death and 10 is best possible health? (Circle One)

Worst 0 1 2 3 4 5 6 7 8 9 10 Best

SUBSCALE: GENERAL VISION

A2. How would you rate your eyesight now (with glasses or contact lens on, if you wear them), on a scale of from 0 to 10, where zero means the worst possible eyesight, as bad or worse than being blind, and 10 means the best possible eyesight? (Circle One)

Worst 0 1 2 3 4 5 6 7 8 9 10 Best

A3. Wearing glasses, how much difficulty do you have reading the small print in a telephone book, on a medicine bottle, or on legal forms? Would you say: (Circle One)

No difficulty at all.. 1

A little difficulty 2

Moderate difficulty.. 3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or not interested in doing this. 6

A4. Because of your eyesight, how much difficulty do you have figuring out whether bills you receive are accurate? (Circle One)

No difficulty at all. 1

A little difficulty 2

Moderate difficulty... 3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or not interested in doing this. 6

A5. Because of your eyesight, how much difficulty do you have doing things like shaving, styling your hair, or putting on makeup? (Circle One)

No difficulty at all. 1

A little difficulty 2

Moderate difficulty... 3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or not interested in doing this. 6

SUBSCALE: DISTANCE VISION

A6. Because of your eyesight, how much difficulty do you have recognizing people you know from across a room? (Circle One)

No difficulty at all. 1

A little difficulty 2

Moderate difficulty..3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or not interested in doing this. 6

A7. Because of your eyesight, how much difficulty do you have taking part in active sports or other outdoor activities that you enjoy (like golf, bowling, jogging, or walking)?

No difficulty at all. 1

A little difficulty 2

Moderate difficulty...3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or not interested in doing this. 6

A8. Because of your eyesight, how much difficulty do you have seeing and enjoying programs on TV?

No difficulty at all. 1

A little difficulty 2

Moderate difficulty...3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or not interested in doing this. 6

SUBSCALE: SOCIAL FUNCTION

A9. Because of your eyesight, how much difficulty do you have entertaining friends and family in your home?

No difficulty at all. 1

A little difficulty 2

Moderate difficulty... 3

Extreme difficulty 4

Have you stopped doing this because of your eyesight 5

Have you stopped doing this for other reasons or not interested in doing this. 6

SUBSCALE: DRIVING

A10. [This item, ―driving in difficult conditions‖, has been included as part of the base set of 25 items as item 16a.]

SUBSCALE: ROLE LIMITATIONS

A11. The next questions are about things you may do because of your vision. For each item, please circle the number to indicate whether for you this is true for you all, most, some, a little, or none of the time. (Circle One On Each Line)

READ CATEGORIES:	All of the time	Most of the time	Some of the time	A little of the time	None of the time
Do you have more help from others because of your vision?	1	2	3	4	5
Are you limited in the kinds of things you can do because of your vision?	1	2	3	4	5

SUBSCALES: WELL-BEING/DISTRESS (#A12) and DEPENDENCY (#A13)

The next questions are about how you deal with your vision. For each statement, please circle the number to indicate whether for you it is definitely true, mostly true, mostly false, or definitely false for you or you don't know.

Sl No	Read Categories:	Definitely true	Mostly true	Not sure	Mostly false	Definitely false
A12	I am often irritable because of my eyesight	1	2	3	4	5
A13	I don't go out of my home alone, because of my eyesight	1	2	3	4	5

ANNEXURE-IV

IMAGE 9 : Fundus photograph of a study patient with normal fundus study

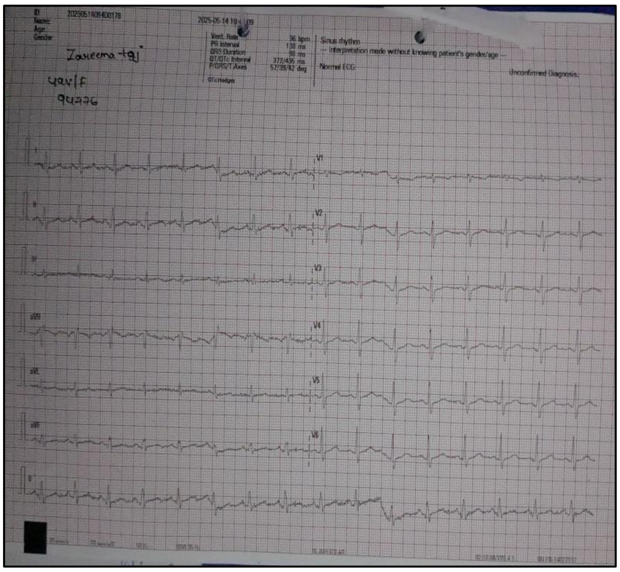

IMAGE 10: ECG of the same study patient showing normal ECG

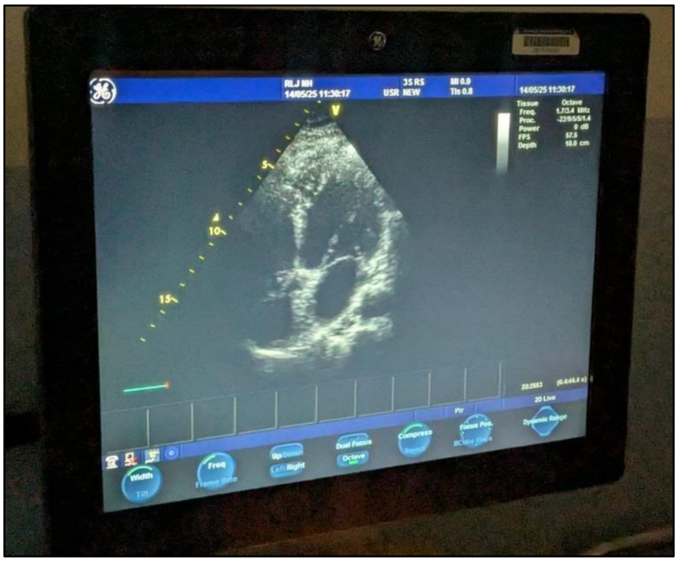

IMAGE 11: Echocardiography of the same study patient with Normal LVDD and EF.

IMAGE 12: Fundus photograph of a study patient with Mild NPDR

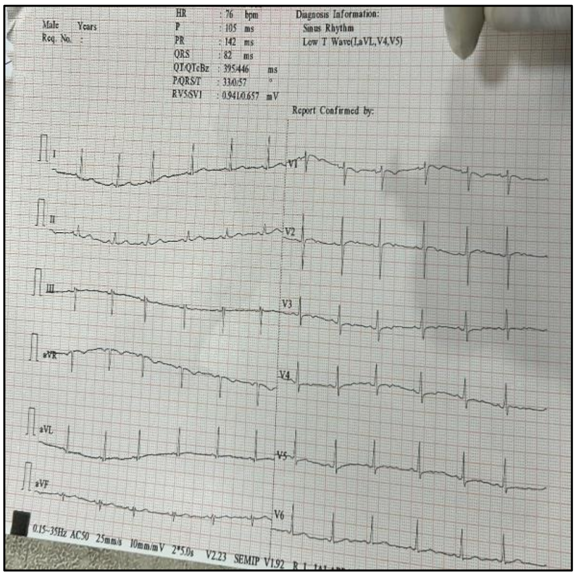

IMAGE 13: ECG of the same study patient showing Low T wave abnormality

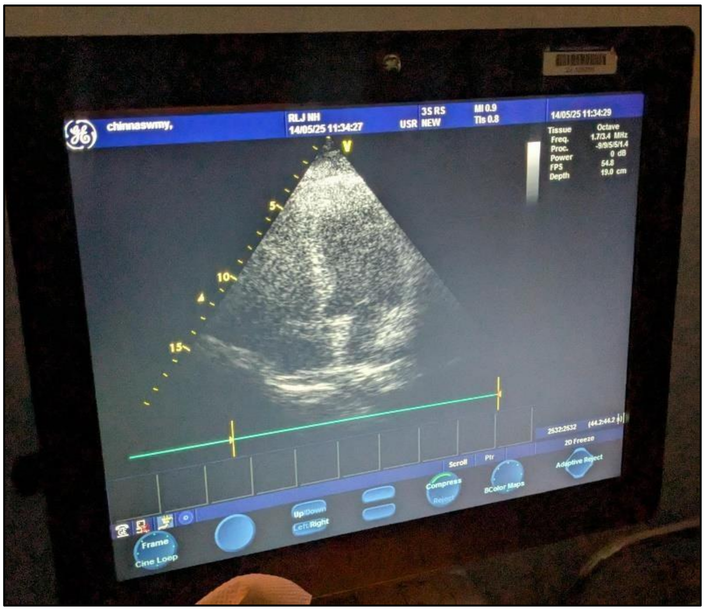

IMAGE 14: Echocardiography of the same study patient with reduced LVDD and 34 %EF.

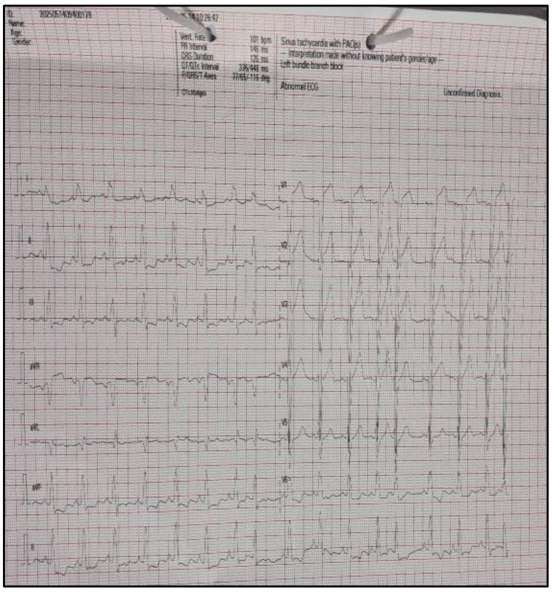

IMAGE 15: Fundus photograph of a study patient with Moderate NPDR

IMAGE 16: ECG of the same study patient showing left bundle branch block

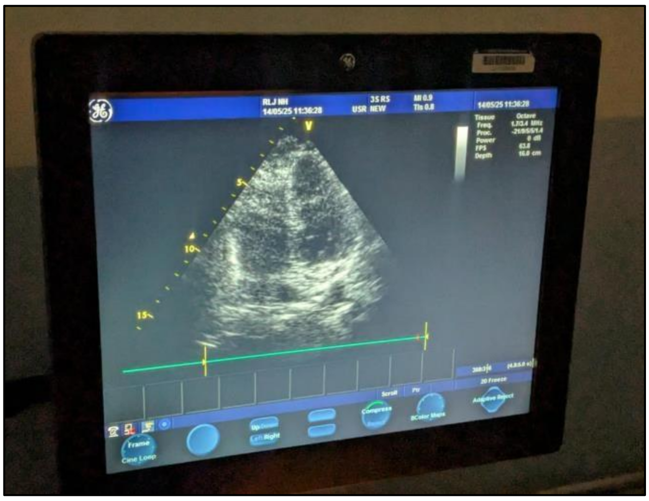

IMAGE 17: Echocardiography of the same study patient with LVH and 27% EF

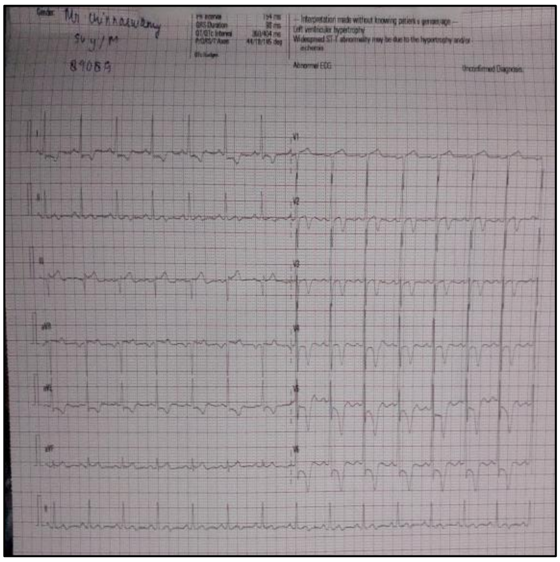

IMAGE 18: Fundus photograph of a study patient with Severe NPDR

IMAGE 19: ECG of the same study patient showing left ventricular hypertrophy- abnormal ECG

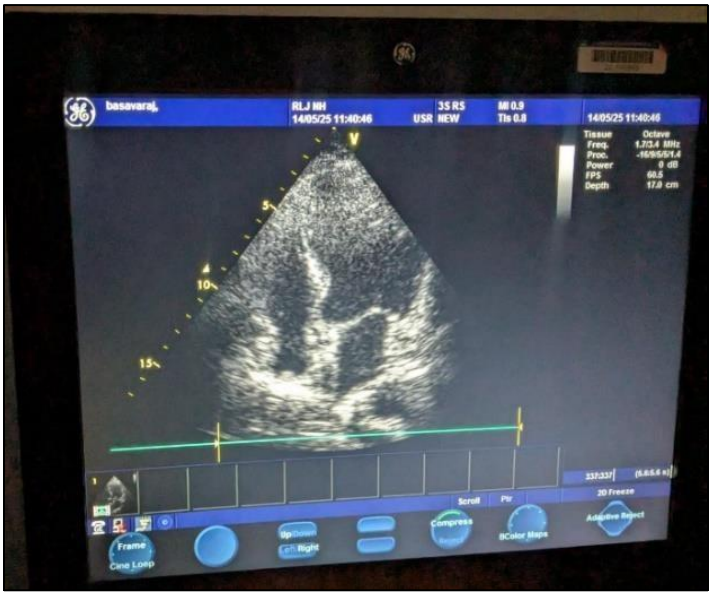

IMAGE 20: Echocardiography of the same study patient with Left Ventricular Hypertrophy and 20% EF

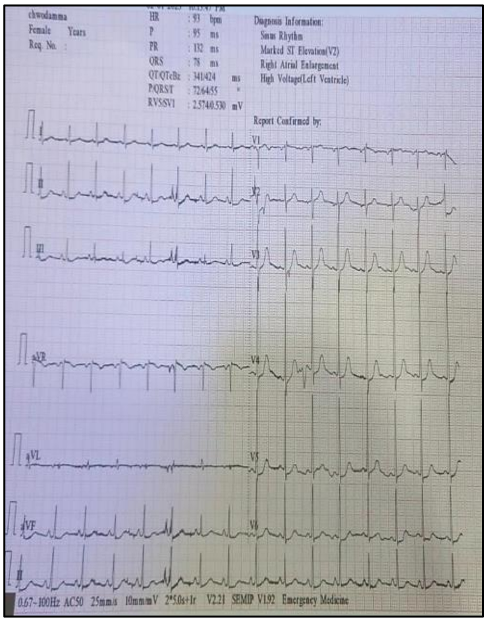

IMAGE 21: Fundus photograph of a study patient with Proliferative Diabetic Retinopathy

IMAGE 22: ECG of the same study patient showing left ventricular hypertrophy- abnormal ECG

IMAGE 23: Echocardiography of the same study patient with Left Ventricular Hypertrophy and 20% EF

IMAGE 24: Slit lamp examination

IMAGE 25: 90D examination

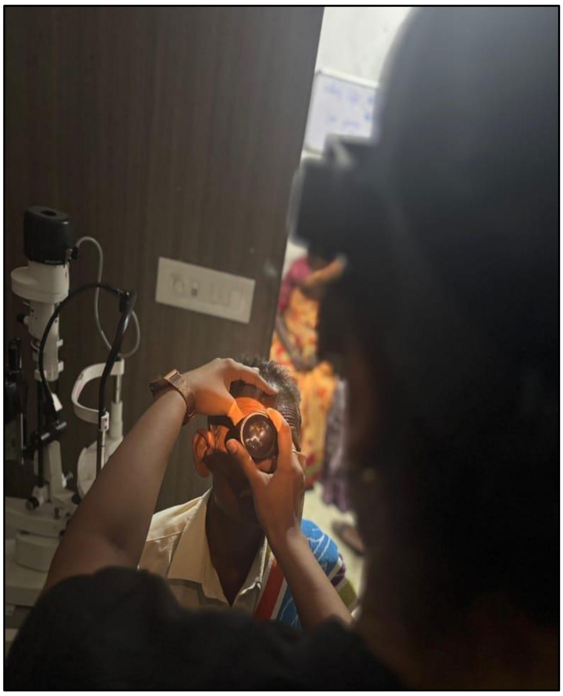

IMAGE 26 and 27: IDO examination

IMAGE 28: ECHOCARDIOGRAPHY PERFORMED ON A PATIENT

IMAGE 28: TOPCON FUNDUS CAMERA

MASTER CHART

Sl No	UHID NO	Age	Gender	Duration of DM		Fundus Finding	BP		FBS	PPBS	HbA1c	B. Urea	S. Creatinine	2 D ECHO				ECG changes	Quality of life score	TREATMENT			
				≤ 10	> 10		Systolic	Diastolic						LVDD	LVDS	LVEF	LA Diameter			No Rx	OHA	Insulin	Combined
1	241121	45	Male	6		BE -Moderate NPDR	90	60	150	176	8.6	17	2.6	45 MM	28 MM	60%	30	Abnormal	Good	No treatment			
2	244794	51	Male		15	BE: HIGH RISK PDR	160	98	162	204	10.5	15	2.4	22	12	45%	20	Suggestive old LVF	Poor			Actrapid 20-20-15 + Lantus 0-0-10	
3	236810	65	Female		12	BE: Mild NPDR	130	90	94	178	9.9	12	0.9	46	30	50%	30	Sinus arrythmia	Excellent			Actrapid + Lantus + Mixtard	
4	213707	55	Male	6		BE- Normal Fundus Study	130	80	90	158	5.8	16	0.8	45	30	55%	26	Acute MI	Excellent	No treatment			
5	223179	53	Male	5		OU- Normal fundus	122	90	116	200	8.6	17	0.7	40	30	60%	30	Right bundle branch block	Good			INJ. H Actrapid 12-12-10	
6	246795	40	Female	8		BE- Severe NPDR	110	70	125	302	8.4	15	3.2	53	35	25%	32	Sinus tachycardia	Good			H. Actrapid , Lantus	
7	142362	70	Male		15	BE- High risk PDR	140	70	195	352	12.6	52	2.3	45	30	60%	32	Sinus tachycardia	Good			H. Actrapid , Lantus	
8	290742	46	Male		12	BE- Moderate Non Proliferative DR	120	80	171	223	10.7	46	2	28	20	45%	25	Sinus arrythmia	Good				Tab Glimi m2 + Inj H Actrapid 8-8-6
9	334226	70	Female	8		BE- Normal Fundus Study	120	90	126	200	12.4	13	0.4	45	30	54%	30	Sinus tachycardia	Good		Glimipride		
10	391916	48	Male	5		BE- Normal Fundus Study	130	80	168	198	15.8	55	0.8	35	25	55%	32	Sinus rhythm	Excellent		Metformin OD		
11	301173	73	Female	10		BE- Mild NPDR	122	88	124	180	8.2	20	0.8	42	25	35%	25	Premature atrial contraction	Good		Tenegliptin, Glimipride		
12	308206	76	Male	9		BE- Normal Fundus Study	130	70	112	140	9.2	28	0.8	33	20	48%	20	Sinus tachycardia	Good			H. Actrapid , Lantus	
13	400515	68	Female	10		BE- Normal Fundus Study	124	88	140	158	8.6	49	0.7	42	25	55%	25	ST depression	Good				Tab Glimi m2 + Inj H Actrapid 8-8-6
14	404079	83	Male	8		BE- Normal Fundus Study	110	88	116	136	8	26	0.7	45	30	50%	32	ST depression	Fair		Glimipride		
15	294260	74	Male	9		BE- Normal Fundus Study	130	90	130	169	7.9	27	0.5	42	25	55%	25	Premature ventricular contraction	Good		Metformin 500 mg 1-0-0		
16	286079	78	Male	10		BE- Normal Fundus Study	120	70	119	150	7.8	25	0.7	45	30	55%	33	Right axis deviation	Good			INJ. H Actrapid 10-10-8 + Lantus 0-0-10	
17	519052	71	Female		20	BE- Moderate NPDR	130	70	146	200	6.4	48	2.2	38	28	45%	44	Sinus tachy cardia	Good		Metformin 500 mg 1-0-0		
18	519651	56	Male		15	RE- Moderate NPDR LE- PDR	100	90	134	154	10	75	2.6	55	45	20%	45	Slight ST Depression	Good		Tab Tenegliptin + Metformin		
19	358647	62	Female	2		BE: Severe NPDR	110	60	169	227	11.1	17	1	32	44	55%	43	Slight S T elevation	Fair			INJ. H Actrapid 10-10-8 + Lantus 0-0-10	
20	91078	75	Male	8		BE: Moderate NPDR	120	70	154	189	8.9	22	0.8	30	32	55%	33	Sinus Bradycardia	Good		Tab Metformin 1-0-0		
21	91076	57	Male	10		BE: Mild NPDR	150	90	112	154	9	33	0.6	36	42	35%	45	Poor r wave progression	Good		Tab Glycomet GP1		
22	92380	63	Female	5		BE: Normal Fundus Study	110	70	159	200	7.8	41	0.9	42	25	55%	40	Sinus Bradycardia	Good		Tab -Glimistar PM2 1-0-1/2		
23	555933	68	Female	10		BE: Normal Fundus Study	140	82	247	309	11.2	21	0.6	42	25	60%	42	Sinus Rhythm	Good	No treatment			
24	546807	48	Female	10		BE: High Risk PDR	130	70	200	304	12	34	0.7	45	30	45%	34	Sinus Tachycardia	Fair			INJ. H Actrapid 12-12-10	
25	90066	64	Female	8		BE: Normal Fundus Study	124	76	124	156	8.6	32	1	45	29	40%	42	Sinus Rhythm	Good			H. Actrapid , Lantus	
26	338574	71	Female	4		BE: High Risk PDR	140	80	238	320	10.9	42	0.4	53	32	20%	43	Left BBB	Fair			H. Actrapid , Lantus	
27	593952	45	Female	3		BE: Normal Fundus Study	128	68	112	134	5.6	17	0.4	42	25	60%	45	Sinus Bradycardia	Good				Tab Glimi m2 + Inj H Actrapid 8-8-6
28	569736	45	Male	7		BE: Normal Fundus Study	142	80	143	187	6.5	44	1.2	45	25	55%	40	S T Depression	Good			H. Actrapid , Lantus	
29	591037	72	Male	3		BE: Severe NPDR	100	70	98	110	5.8	42	1.9	52	40	25%	39	Inverted T wave	Fair		Tab Glimipride + Metformin 1-0-1		
30	593026	80	Female	4		BE: Normal Fundus Study	120	76	216	235	7.2	11	0.5	45	25	55%	46	Poor r wave progression	Good		Tab Metformin 1-0-0		
31	275914	74	Female	5		BE: Mild NPDR	116	72	118	178	6.9	32	0.9	45	28	50%	42	Normal ECG	Good			H. Actrapid , Lantus	
32	584670	64	Female	3		BE: Severe NPDR	120	74	129	189	8.2	41	0.8	50	40	15%	45	Short PR interval	Good		Tab -Glimistar PM2 1-0-1/2		
33	372528	65	Female	8		BE: PDR	120	80	346	487	12.8	42	0.7	42	25	30%	39	Sinus Tachycardia	Good				Tab Glimi m2 + Inj H Actrapid 8-8-6
34	587514	68	Male		12	BE: High Risk PDR	140	90	271	380	11.4	42	1.9	36	28	25%	46	Left axis deviation	Fair		Tab Metformin 1-0-0		
35	501481	70	Male	2		BE: Normal Fundus Study	136	74	218	313	10.6	31	0.9	38	23	40%	37	Sinus Tachycardia	Good			H. Actrapid , Lantus	
36	248271	65	Male	6		BE- No DR changes	130	76	124	160	8.1	22	2.8	42	34	52%	45	Sinus Tachycardia	Good		Tab Tenegliptin + Metformin		
37	248241	60	Male	6		BE- Moderate Non Proliferative DR	110	80	90	130	10.2	28	4.5	29	16	20%	34	Short PR interval	Good			INJ. H Actrapid 10-10-8 + Lantus 0-0-10	
38	191290	67	Male	5		BE- mild NPDR	140	98	116	158	8.6	24	0.7	38	21	47%	42	Tall T waves	Excellent		Tab Metformin 1-0-0		
39	191562	65	Male	5		RE: PDR, LE : Severe NPDR	120	80	166	189	9.9	28	2.3	22	17	32%	45	T waves abnormality	Fair		Tab Glycomet GP1		
40	191566	72	Male	6		BE- Severe NPDR	140	80	122	197	12.2	23	3.7	24	15	29%	46	Sinus Tachycardia	Fair		Tab -Glimistar PM2 1-0-1/2		

41	191477	60	Male	7		BE- Mild NPDR	160	100	113	147	8.2	45	2.4	32	22	40%	39	Sinus Bradycardia	Good		Tab Glycomet GP1		
42	191773	67	Female	10		RE- Moderate NPDR LE- Mild DR	130	70	197	230	10.2	33	1.5	29	18	27%	30	Short PR interval	Fair		Tab . Glimicer + Vildagliptin 1-0-1		
43	191752	51	Male	7		BE- Normal Fundus Study	140	80	224	218	9.9	21	1.6	29	31	44%	32	Poor r wave progression	Good		Tab Glimipride + Metformin 1-0-1		
44	253376	30	Female	6		BE: Normal fundus study	128	76	122	160	6.9	18	0.9	42	27	53%	37	Sinus Rhythm	Excellent		Glimipride		
45	191741	87	Male		20	BE- Mild NPDR	140	78	100	134	10.6	32	0.6	37	22	38%	45	Inverted T wave	Fair		Tab Glimipride + Metformin 1-0-1		
46	196480	55	Male		16	BE- High risk PDR	130	74	259	208	13.2	23	0.9	20	14	33%	43	Sinus Tachycardia	Good		Tab -Glimistar PM2 1-0-1/2		
47	239554	65	Male	7		OU- Normal fundus	134	76	114	152	6.6	36	1.4	40	30	49%	44	Short PR interval	Good	No meds			
48	239566	79	Male	10		BE- Normal Fundus Study	128	74	90	120	6.9	51	1.1	45	32	56%	41	T waves abnormality	Good	No meds			
49	244408	65	Female	5		BE- Moderate Non Proliferative DR	136	78	102	68	6.5	27	0.8	30	20	38%	37	T waves abnormality	Fair	No meds			
50	519241	67	Female	5		BE: Normal fundus study	130	70	170	193	6.4	28	0.7	45	30	30%	34	Sinus Tachycardia	Excellent		Tab Tenegliptin + Metformin		
51	515487	57	Male	10		BE: Moderate NPDR	126	82	170	213	11.3	21	0.8	45	28	40%	32	Short PR interval	Good			INJ. H Actrapid 10-10-8 + Lantus 0-0-10	
52	109258	45	Male	2		BE: Normal fundus study	132	76	121	236	7.8	25	1.2	35	27	40%	34	Sinus Tachycardia	Good		Tab Metformin 1 0-0		
53	543782	39	Male	2		BE: Normal fundus study	120	70	110	140	7	18	1	55	45	25%	43	T wave abnormality	Fair		Tab Tenegliptin + Metformin		
54	536848	60	Male	7		BE: Mild NPDR	120	72	168	190	8.2	30	1	55	45	20%	45	Arrhythmia	Fair			INJ. H Actrapid 10-10-8 + Lantus 0-0-10	
55	382976	63	Male		12	BE: Moderate NPDR	110	72	186	230	8.9	29	0.8	45	28	40%	42	Sinus Tachycardia	Good		Tab Metformin 1 0-0		
56	432672	57	Male		11	BE: Severe NPDR	130	82	156	172	9.2	21	0.6	48	32	35%	38	Tachycardia	Good		Tab Glycomet GP1		
57	372456	70	Male	2		BE: PDR	140	70	310	312	14.1	28	0.6	52	35	20%	34	T wave abnormality	Fair		Tab -Glimistar PM2 1-0-1/2		
58	511009	49	Female	5		BE: Normal fundus study	110	82	112	120	5.8	30	0.8	55	45	40%	38	Short PR interval	Good		Tab Glycomet GP1		
59	515528	73	Male	7		BE: Normal fundus study	120	72	126	154	7	38	1	45	30	60%	38	T wave abnormality	Excellent		Tab . Glimicer + Vildagliptin 1-0-1		
60	521569	64	Female	6		BE: Normal fundus study	142	92	85	121	7.3	28	1.3	45	30	55%	45	Short PR interval	Good		Tab Glimipride + Metformin 1-0-1		
61	593952	49	Male		14	BE: Moderate NPDR	120	70	146	178	9.2	40	1.2	42	25	35%	39	T wave abnormality	Good		Glimipride		
62	569736	56	Female	4		BE: Mild NPDR	110	60	120	145	8.3	34	0.5	53	25	30%	56	Short PR interval	Fair	No meds			
63	591037	59	Female	8		BE: Normal fundus study	120	78	216	327	12.9	38	0.7	42	25	40%	37	Sinus Tachycardia	Good	No meds			
64	593026	70	Female		20	BE: Moderate NPDR	130	70	168	217	8.9	42	1.2	45	29	50%	38	Short PR interval	Good		Tab Tenegliptin + Metformin		
65	275914	59	Male		15	BE: PDR	110	70	125	190	6.9	59	1.5	45	35	20%	35	Tall T waves	Good		Tab Glycomet GP1		
66	584670	65	Male	2		BE: Mild NPDR	136	78	103	142	9.1	42	1.2	45	25	45%	43	Tall T waves	Good			Tab Glimipride + Metformin 1-0-1	
67	372528	73	Male	2		BE: Normal fundus study	120	68	96	110	6.7	19	0.2	42	25	60%	42	T wave abnormality	Excellent			Glimipride	
68	587514	80	Female	4		BE: Normal fundus study	120	70	107	215	7.3	8	0.7	45	30	55%	34	S T elevation	Good			Metformin OD	
69	252190	61	Male		20	BE: High Risk PDR	130	80	301	336	8.7	52	1.5	45	26	45%	38	Left Axis deviation	Good		Tab Tenegliptin + Metformin		
70	445414	69	Male	7		BE: High Risk PDR	120	70	142	167	8.2	26	1.7	45	28	40%	51	Short PR interval	Good			Actrapid 20-20-15 + Lantus 0-0-10	
71	464458	71	Female		20	BE: Moderate NPDR	110	70	108	270	11.5	79	6.3	55	45	25%	42	Anterior MI	Fair			Actrapid + Lantus + Mixtard	
72	449517	64	Male	3		BE: Normal fundus study	100	70	109	160	12.5	22	1.2	45	28	60%	34	Small Inferior Q wave	Good	No treatment			
73	518721	47	Male		15	BE: High Risk PDR	120	74	124	142	6.7	17	0.7	46	28	40%	39	Left Axis deviation	Good			INJ. H Actrapid 12-12-10	
74	402538	65	M	6		BE -Moderate NPDR	90	60	150	176	8.6	17	2.6	45	28	60%	30	Abnormal	Good		Tab Glimipride + Metformin 1-0-1		
75	290742	46	Male		15	BE: HIGH RISK PDR	160	98	162	204	10.5	15	2.4	22	12	45%	20	Suggestive old LVF	Poor			Actrapid 20-20-15 + Lantus 0-0-10	
76	378258	62	M		12	BE: Mild NPDR	130	90	94	178	9.9	12	0.9	46	30	50%	30	Sinus arrythmia	Excellent			Actrapid + Lantus + Mixtard	
77	500625	65	Male	6		BE- Normal Fundus Study	130	80	90	158	5.8	16	0.8	45	30	55%	26	Acute MI	Excellent	No treatment			
78	256159	85	F	5		OU- Normal fundus	122	90	116	200	8.6	17	0.7	40	30	60%	30	Right bundle branch block	Good			INJ. H Actrapid 12-12-10	
79	251445	53	M	8		BE- Severe NPDR	110	70	125	302	8.4	15	3.2	53	35	25%	32	Sinus tachycardia	Good			H. Actrapid , Lantus	
80	461682	64	M		15	BE- High risk PDR	140	70	195	352	12.6	52	2.3	45	30	60%	32	Sinus tachycardia	Good			H. Actrapid , Lantus	
81	250231	62	F		12	BE- Moderate Non Proliferative DR	120	80	171	223	10.7	46	2	28	20	45%	25	Sinus arrythmia	Good		Tab Glimipride + Metformin 1-0-1		
82	381625	67	F	8		BE- Normal Fundus Study	120	90	126	200	12.4	13	0.4	45	30	54%	30	Sinus tachycardia	Good		Glimipride		
83	247236	73	M	5		BE- Normal Fundus Study	130	80	168	198	15.8	55	0.8	35	25	55%	32	Sinus rhythm	Excellent		Metformin OD		

84	514698	78	F	10		BE- Mild NPDR	122	88	124	180	8.2	20	0.8	42	25	35%	25	Premature atrial contraction	Good		Tenegliptin, Glimipride		
85	344506	75	male	9		BE- Normal Fundus Study	130	70	112	140	9.2	28	0.8	33	20	48%	20	Sinus tachycardia	Good		Tab Glimipride + Metformin 1-0-1		
86	236810	59	Female	10		BE- Normal Fundus Study	124	88	140	158	8.6	49	0.7	42	25	55%	25	ST depression	Good				Tab Glimi m2 + Inj H Actrapid 8-8-6
87	361112	80	Female	8		BE- Normal Fundus Study	110	88	116	136	8	26	0.7	45	30	50%	32	ST depression	Fair		Tab Glimipride + Metformin 1-0-1		
88	507619	87	F	9		BE- Normal Fundus Study	130	90	130	169	7.9	27	0.5	42	25	55%	25	Premature ventricular contraction	Good		Metformin 500 mg 1-0-0		
89	398617	70	F	10		BE- Normal Fundus Study	120	70	119	150	7.8	25	0.7	45	30	55%	33	Right axis deviation	Good			INJ. H Actrapid 10-10-8 + Lantus 0-0-10	
91	235339	61	F		20	BE- Moderate NPDR	130	70	146	200	6.4	48	2.2	38	28	45%	44	Sinus tachy cardia	Good			Actrapid 20-20-15 + Lantus 0-0-10	
92	381625	67	F		15	RE- Moderate NPDR LE- PDR	100	90	134	154	10	75	2.6	55	45	20%	45	Slight ST Depression	Good			Actrapid + Lantus + Mixtard	
94	496146	68	F	2		BE: Severe NPDR	110	60	169	227	11.1	17	1	32	44	55%	43	Slight S T elevation	Fair	No treatment			
95	233825	76	M	8		BE: Moderate NPDR	120	70	154	189	8.9	22	0.8	30	32	55%	33	Sinus Bradycardia	Good			INJ. H Actrapid 12-12-10	
96	442873	75	F	10		BE: Mild NPDR	150	90	112	154	9	33	0.6	36	42	35%	45	Poor r wave progression	Good			H. Actrapid , Lantus	
97	508834	78	F	5		BE- Normal Fundus Study	110	70	159	200	7.8	41	0.9	42	25	55%	40	Sinus Bradycardia	Good			H. Actrapid , Lantus	