"ASSOCIATION OF HYPERTENSIVE RETINOPATHY AND CARDIAC REMODELLING IN SYSTEMIC HYPERTENSION"

By

DR. ATHMIKA.R,

MBBS

Dissertation submitted to the

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

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

Under the guidance of

DR. SANGEETHA.T

MBBS, MS , FPRS

DEPARTMENT OF OPHTHALMOLOGY,

SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR

2025

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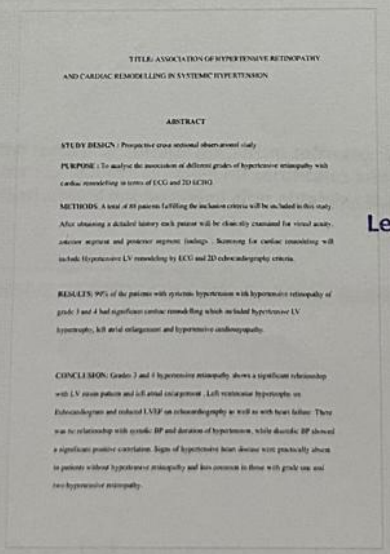

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ABSTRACT

STUDY DESIGN : Prospective cross sectional observational study

PURPOSE : To analyse the association of different grades of hypertensive retinopathy with cardiac remodelling in terms of ECG and 2D ECHO.

METHODS: A total of 88 patients fulfilling the inclusion criteria will be included in this study. After obtaining a detailed history each patient will be clinically examined for visual acuity, anterior segment and posterior segment findings . Screening for cardiac remodeling will include Hypertensive LV remodeling by ECG and 2D echocardiography criteria.

RESULTS: 90% of the patients with systemic hypertension with hypertensive retinopathy of grade 3 and 4 had significant cardiac remodelling which included hypertensive LV hypertrophy, left atrial enlargement and hypertensive cardiomyopathy.

CONCLUSION: Grades 3 and 4 hypertensive retinopathy shows a significant relationship with LV mass pattern and left atrial enlargement . Left ventricular hypertrophy on Echocardiogram and reduced LVEF on echocardiography as well as with heart failure. There was no relationship with systolic BP and duration of hypertension, while diastolic BP showed a significant positive correlation. Signs of hypertensive heart disease were practically absent in patients without hypertensive retinopathy and less common in those with grade one and two hypertensive retinopathy.

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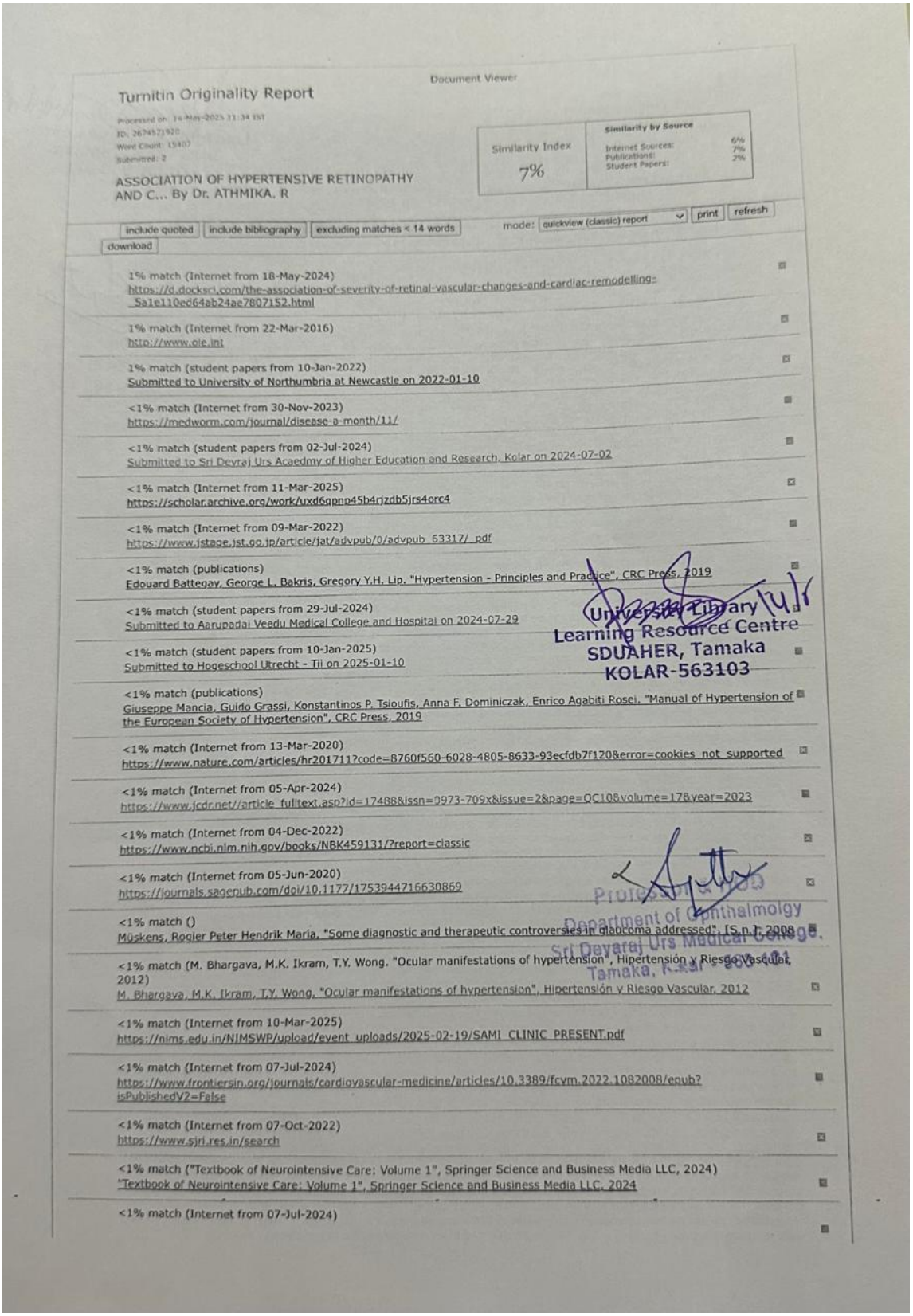

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TITLE: ASSOCIATION OF HYPERTENSIVE RETINOPATHY AND CARDIAC REMODELLING IN SYSTEMIC HYPERTENSION

ABSTRACT STUDY DESIGN : Prospective cross sectional observational study PURPOSE : To analyse the association of different grades of hypertensive retinopathy with cardiac remodelling in terms of ECG and 2D ECHO. METHODS: A total of 88 patients fulfilling the inclusion criteria will be included in this study. After obtaining a detailed history each patient will be clinically examined for visual acuity, anterior segment and posterior segment findings . Screening for cardiac remodelling will include Hypertensive LV remodeling by ECG and 2D echocardiography criteria. RESULTS: 90% of the patients with systemic hypertension with hypertensive retinopathy of grade 3 and 4 had significant cardiac remodelling which included hypertensive LV hypertrophy, left atrial enlargement and hypertensive cardiomyopathy. CONCLUSION: Grades 3 and 4 hypertensive retinopathy shows a significant relationship with LV strain pattern and left atrial enlargement , Left ventricular hypertrophy on Echocardiogram and reduced LVEF on echocardiography as well as with heart failure. There was no relationship with systolic BP and duration of hypertension, while diastolic BP showed a significant positive correlation. Signs of hypertensive heart disease were practically absent in patients without hypertensive retinopathy and less common in those with grade one and two hypertensive retinopathy. INTRODUCTION Hypertensive retinopathy (HR) is a retinal condition caused by long-term increased blood pressure, which leads to damage to the blood vessels in the retina. As a major consequence of poorly controlled hypertension, hypertensive retinopathy is a important cause of ocular morbidity, and in severe cases, it can lead to permanent vision loss. The retinal vasculature is particularly vulnerable to the effects of high blood pressure.1 "Hypertension has profound effects on various parts of the eye. Classically, elevated blood pressure results in a series of retinal microvascular changes called hypertensive retinopathy, comprising of generalized and focal retinal arteriolar narrowing, arteriovenous nicking, retinal haemorrhages, microaneurysms and, in severe cases, optic disc and macular oedema".2 "Hypertensive retinopathy signs are associated with other indicators of end-organ damage (for example, left ventricular hypertrophy, renal impairment) and may be a risk marker of future clinical events, such as stroke, congestive heart failure and cardiovascular mortality".3 "Hypertensive retinopathy is graded based on the severity and four grades of the disease are defined, grade 1 which is known as mild hypertensive retinopathy, grade 2 is moderate hypertensive retinopathy, grade 3 is a combination of grade 1 and grade 2 and finally grade 4 known as accelerated hypertensive retinopathy".4 Figure 1-Grades of Hypertensive Retinopathy "Hypertension is a prominent clinical condition that promotes widespread cardiac remodelling, functioning as a contributing factor in both systolic and diastolic dysfunction, arrhythmias, and symptomatic heart failure, which are among the leading causes of death globally".5 "Although the effect of hypertension on the left ventricle is well studied, that on the left atrium (LA) is less well defined. Electrocardiographic (ECG) or echocardiographic LA enlargement occurs commonly in hypertensive patients. In one study 21% of hypertensive patients without ECG evidence of LVH had LA enlargement of greater than 4 cm. In subjects with ECG LVH, the prevalence of LA enlargement is higher (56% in women and 38% in men)".6 Cardiac remodelling in arterial hypertension causes left ventricular dysfunction and reduced coronary flow reserve. Antihypertensive treatment should aim to correct this by reversing myocytes hypertrophy, recovering myocardial structure and coronary flow reserve. Hydralazine, felodipine, and lisinopril have been shown in studies to improve coronary reserve in spontaneously hypertensive rats while preventing LVH regression. Long-term antihypertensive medication can improve the coronary flow reserve in hypertensive patients with microvascular dysfunction7. Figure 2-Myocardial remodelling factors AIM AND OBJECTIVES Aim Association of Hypertensive Retinopathy and Cardiac Remodelling in Systemic Hypertension. Objectives of Study To analyse the association of different grades of hypertensive retinopathy with cardiac remodelling in terms of ECG and 2D ECHO. REVIEW OF LITERATURE "Hypertension is frequently associated with asymptomatic target organ damage. Ideally, screening should look for ECG abnormalities such as LVH, microalbuminuria, carotid intima- media thickness, and arterial pulse wave velocity. Hypertensive retinopathy, a target organ damage, is frequently related with other target organ damage which assists in the prognosis of hypertension".8 "Hypertensive retinopathy is widely recognized as a sign and/or prediction of vascular disease and death. Because the retinal and cerebrovascular circulations share anatomical, physiological, histological, and embryological characteristics, it is not surprising that hypertensive retinopathy is closely linked to stroke or lacunar infarction. In a three-year population-based cohort study with atherosclerotic risk, exudates (cotton-wool spots), retinal hemorrhages, and microaneurysms were linked with a 2- to 4-fold increased risk of incident stroke, cognitive decline, white matter lesions, cerebral atrophy, and stroke death".9 HR can predict CHD in high-risk individuals, regardless of other risk variables such as blood pressure. Most worldwide HTN care guidelines underline that HR accompanied with LVH and renal impairment represents target organ damage, necessitating a more aggressive management approach for these hypertensive patients.10 "Hypertensive retinopathy is distinguished by increased vascular permeability, which results from the collapse of the blood-retinal barrier. This lack of barrier integrity allows proteins and fluids to flow into the retinal tissues, resulting in edema and hemorrhages. These alterations weaken the retinal microvasculature, causing ischemia and increased oxidative stress. The remodelling of smooth muscle cells within retinal arterioles, along with endothelial dysfunction, aggravates the condition by increasing vascular stiffness and resistance". Retinal examination using SLDF (Scanning Laser Doppler Flowmetry) offers a high sensitivity for detecting structural vascular abnormalities associated with heart injury. "Lumen constriction of retinal arterioles was found to be independently related to intraventricular septum thickness, left ventricular mass, and left atrial volume. Wall lumen ratio was independently related with the intraventricular septum and left atrial volume".11 Structural changes in retinal arteries may resemble those in small resistance arteries and that systemic blood pressure influences vascular structure in retinal arterioles similar to that in peripheral arteries. This is supported by the observations that, in the treated patients with well- controlled hypertension, media: lumen ratio was less than that in patients with uncontrolled hypertension, similar to data reported by others in small arteries from subcutaneous tissue.8 "LVH (Left Ventricular Hypertrophy) may be associated with increased mortality by a number of mechanisms. Activation of the renin-angiotensin system not only leads to LVH but also may promote the progression of atherosclerosis by the effects of angiotensin II on vasomotor tone, coagulation, and vascular smooth muscle cell proliferation. Additionally, LVH may predispose to arrhythmic death. Myocardial fibrosis occurs in LVH and may provide the substrate for re- entrant ventricular arrhythmias".12 "Generalized retinal arteriolar narrowing and arteriovenous nicking were associated with an elevation in blood pressure that had been documented six to eight years before the retinal

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ACKNOWLEDGEMENT

My sincere appreciation to my respected guide **Dr. SANGEETHA T.** for her unwavering guidance.

Her valuable suggestions, and kind encouragement throughout this study were immeasurable. Her constant support, wise guidance, and prudent admonitions have empowered me to cultivate a profound comprehension of the subject. Her steadfast guidance, keen scientific insight, practicality, a knack for solving the impossible, and ability to break complex ideas into simple terms taught me to think beyond the box. I wish to imprint her teachings throughout my career.

My genuine and profound appreciation to **Dr B.O.HANUMANTHAPPA .** for his timely assistance and support. His scholarly suggestions, intellectual stimulation, genuine interest, affection and comfort have been a constant source of inspiration. Besides, his insightful advice and dedication have been instrumental in completing this post-graduate program.

I am sincerely thankful to the esteemed faculty members: **DR. MANJULA T.R , DR. USHA B.R, DR. RASHMI.G, DR. CHAITRA M.C, DR. INCHARA.N, DR.NAVEENA.A,DR.APOORVA.N,DR.PADMINI.S,DR.ATHIRA.K,DR.NARENDRA N.B.S,DR.SHWETHA** for their insightful discussions during seminars and valuable suggestions. Their expertise and wisdom have significantly contributed to my personal and professional advancement.

My acknowledgement would be incomplete without mentioning my dearest and beloved family, especially my Husband **DR.LAKWAN SAKTHI D R** and parents **MR.RAMASAMY C** and **MRS.JAYANTHI R ,** my dear brother **MR.ADWAITH RAHUL R** for all the support and belief they had in me.

I thank my fellow post graduates and my friends **DR. ANUNITHA, DR. ALISHA, DR. SANJANA, DR. BHAVISHYA, DR. HITHESH , DR.HANEELA , DR.RAVEENA , DR.PRAMUKH , DR. HIMA TEJA** for their support throughout.

Thank you for everything. This journey wouldn't have been easy without you .

Lastly, I would like to extend my gratitude to the nursing staff and hospital workers for their assistance in conducting the study. My humble acknowledgement to the patients and their cooperation during this research.

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ABBREVIATIONS

HR	HYPERTENSIVE RETINOPATHY
LA	LEFT ATRIUM
ECG	ELECTROCARDIOGRAPHY
LVH	LEFT VENTRICULAR HYPERTROPHY
ECHO	ECHOCARDIOGRAPHY
CHD	CONGESTIVE HEART DISEASE
SLDF	SCANNING LASER DOPPLER FLOWMETRY
WCSA	WALL CROSS SECTIONAL AREA
CHF	CONGESTIVE HEART FAILURE
TOD	TARGET ORGAN DAMAGE
LVMI	LEFT VENTRICULAR MASS INDEX
RWT	RELATIVE WALL THICKNESS
IMT	INTIMA MEDIA THICKNESS
UAE	URINE ALBUMIN EXCRETION
IOP	INTRAOCULAR PRESSURE
CVD	CARDIOVASCULAR DISEASE
VD	VENULAR DENSITY
RNFL	RETINAL NERVE FIBER LAYER
OCTA	OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY

LVEF	LEFT VENTRICULAR EJECTION FRACTION
RCF	RETINAL CAPILLARY FLOW
WLR	WALL TO LUMEN RATIO
HF	HEART FAILURE
RI	REMODELLING INDEX
NT-PROBNP	N TERMINAL PRO B TYPE NATRIURETIC PEPTIDE
ASE	AMERICAN SOCIETY OF ECHOCARDIOGRAPHY

ASSOCIATION OF HYPERTENSIVE RETINOPATHY AND CARDIAC REMODELLING IN SYSTEMIC HYPERTENSION

ABSTRACT

TITLE: ASSOCIATION OF HYPERTENSIVE RETINOPATHY AND CARDIAC REMODELLING IN SYSTEMIC HYPERTENSION

STUDY DESIGN : Prospective cross sectional observational study

PURPOSE : The analysis of the association between different grades of hypertensive retinopathy and cardiac remodelling through ECG (Electrocardiogram) and 2D ECHO (Two-Dimensional Echocardiography) involves exploring how progressive retinal changes due to hypertension correlate with structural and functional alterations in the heart. Hypertensive retinopathy, a complication of chronic hypertension, is graded based on the severity of retinal vascular changes, ranging from mild narrowing of retinal arterioles to advanced exudative and hemorrhagic changes. These retinal changes are reflective of systemic vascular damage, including the cardiovascular system. ECG can detect electrical changes such as left ventricular hypertrophy (LVH) and arrhythmias, which are common in hypertensive heart disease. 2D ECHO provides a detailed assessment of cardiac structure and function, including measurements of left ventricular mass, wall thickness, ejection fraction, and diastolic function. By analyzing the relationship between the severity of retinopathy and cardiac findings on ECG and 2D ECHO, researchers can better understand how microvascular damage in the retina reflects or predicts macrovascular and myocardial remodelling, potentially aiding in early cardiovascular risk stratification in hypertensive patients.

METHODS: A total of 88 patients fulfilling the inclusion criteria will be included in this study. After obtaining a detailed history each patient will be clinically examined for visual acuity, anterior segment and posterior segment findings . Screening for cardiac remodeling

will include Hypertensive LV remodeling by ECG and 2D echocardiography criteria.

RESULTS: 90% of the patients with systemic hypertension with hypertensive retinopathy of grade 3 and 4 had significant cardiac remodelling which included hypertensive LV hypertrophy, left atrial enlargement and hypertensive cardiomyopathy.

CONCLUSION: Grades 3 and 4 hypertensive retinopathy shows a significant relationship with LV strain pattern and left atrial enlargement , Left ventricular hypertrophy on Echocardiogram and reduced LVEF on echocardiography as well as with heart failure. There was no relationship with systolic BP and duration of hypertension, while diastolic BP showed a significant positive correlation. Signs of hypertensive heart disease were practically absent in patients without hypertensive retinopathy and less common in those with grade one and two hypertensive retinopathy.

KEY WORDS : Hypertensive retinopathy , Cardiac remodelling , Hypertension , Left ventricular hypertrophy , Echocardiogram

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INTRODUCTION

INTRODUCTION

Hypertensive retinopathy (HR) is a retinal condition caused by long-term increased blood pressure, which leads to damage to the blood vessels in the retina. As a major consequence of poorly controlled hypertension, hypertensive retinopathy is a important cause of ocular morbidity, and in severe cases, it can lead to permanent vision loss. The retinal vasculature is particularly vulnerable to the effects of high blood pressure.[1]

Hypertension has profound effects on various parts of the eye. Classically, elevated blood pressure results in a series of retinal microvascular changes called hypertensive retinopathy, comprising of generalized and focal retinal arteriolar narrowing, arteriovenous nicking, retinal haemorrhages, microaneurysms and, in severe cases, optic disc and macular oedema.[2]

Hypertensive retinopathy signs are associated with other indicators of end-organ damage (for example, left ventricular hypertrophy, renal impairment) and may be a risk marker of future clinical events, such as stroke, congestive heart failure and cardiovascular mortality.[3]

Hypertensive retinopathy is graded based on the severity and four grades of the disease are defined, grade 1 which is known as mild hypertensive retinopathy, grade 2 is moderate hypertensive retinopathy, grade 3 is a combination of grade 1 and grade 2 and finally grade 4 known as accelerated hypertensive retinopathy.[4]

Hypertension is a prominent clinical condition that promotes widespread cardiac remodelling, functioning as a contributing factor in both systolic and diastolic dysfunction, arrhythmias, and symptomatic heart failure, which are among the leading causes of death globally.[5]

Although the effect of hypertension on the left ventricle is well studied, that on the left atrium (LA) is less well defined. Electrocardiographic (ECG) or echocardiographic LA enlargement

occurs commonly in hypertensive patients. In one study 21% of hypertensive patients without ECG evidence of LVH had LA enlargement of greater than 4 cm. In subjects with ECG LVH, the prevalence of LA enlargement is higher (56% in women and 38% in men).[6]

Cardiac remodelling in arterial hypertension causes left ventricular dysfunction and reduced coronary flow reserve. Antihypertensive treatment should aim to correct this by reversing myocytes hypertrophy, recovering myocardial structure and coronary flow reserve. Hydralazine, felodipine, and lisinopril have been shown in studies to improve coronary reserve in spontaneously hypertensive rats while preventing LVH regression. Long-term antihypertensive medication can improve the coronary flow reserve in hypertensive patients with microvascular dysfunction[7].

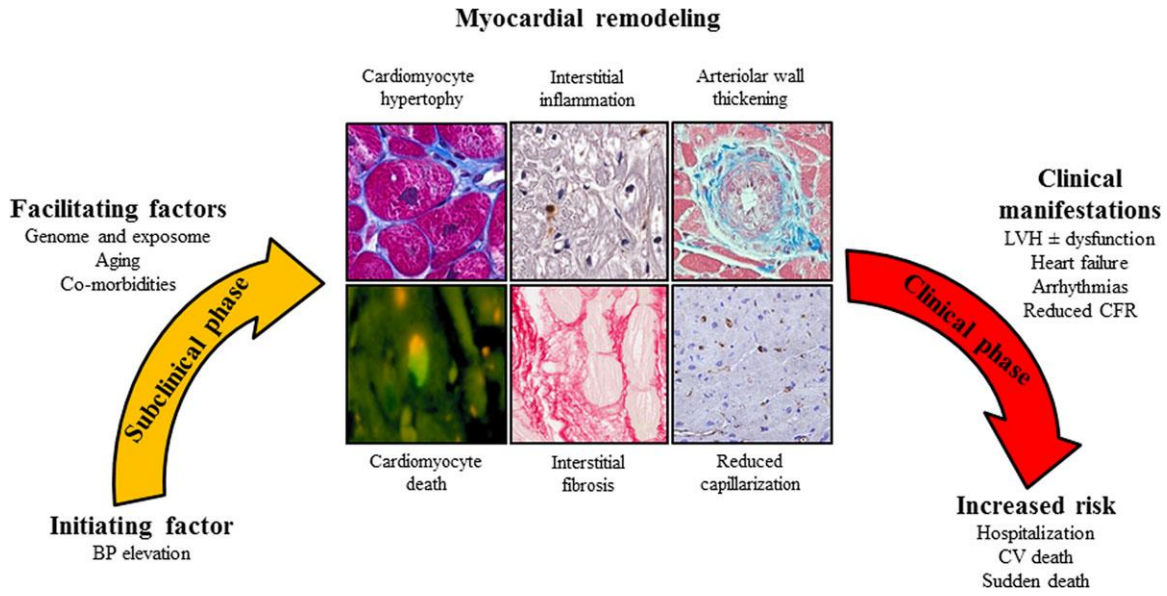

Figure 1-Myocardial remodelling factors

AIM AND OBJECTIVES

AIM AND OBJECTIVES

Aim

Association of Hypertensive Retinopathy and Cardiac Remodelling in Systemic Hypertension.

Objectives of Study

To analyse the association of different grades of hypertensive retinopathy with cardiac remodelling in terms of ECG and 2D ECHO.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Hypertension is frequently associated with asymptomatic target organ damage. Ideally, screening should look for ECG abnormalities such as LVH, microalbuminuria, carotid intima-media thickness, and arterial pulse wave velocity. Hypertensive retinopathy, a target organ damage, is frequently related with other target organ damage which assists in the prognosis of hypertension.[8]

Hypertensive retinopathy is widely recognized as a sign and/or prediction of vascular disease and death. Because the retinal and cerebrovascular circulations share anatomical, physiological, histological, and embryological characteristics, it is not surprising that hypertensive retinopathy is closely linked to stroke or lacunar infarction. In a three-year population-based cohort study with atherosclerotic risk, exudates (cotton-wool spots), retinal hemorrhages, and microaneurysms were linked with a 2- to 4-fold increased risk of incident stroke, cognitive decline, white matter lesions, cerebral atrophy, and stroke death.[9]

HR can predict CHD in high-risk individuals, regardless of other risk variables such as blood pressure. Most worldwide HTN care guidelines underline that HR accompanied with LVH and renal impairment represents target organ damage, necessitating a more aggressive management approach for these hypertensive patients.[10]

Hypertensive retinopathy is distinguished by increased vascular permeability, which results from the collapse of the blood-retinal barrier. This lack of barrier integrity allows proteins and fluids to flow into the retinal tissues, resulting in edema and hemorrhages. These alterations weaken the retinal microvasculature, causing ischemia and increased oxidative stress. The remodelling of smooth muscle cells within retinal arterioles, along with endothelial dysfunction, aggravates the condition by increasing vascular stiffness and resistance.

Retinal examination using SLDF (Scanning Laser Doppler Flowmetry) offers a high sensitivity for detecting structural vascular abnormalities associated with heart injury. Lumen constriction of retinal arterioles was found to be independently related to intraventricular septum thickness, left ventricular mass, and left atrial volume. Wall lumen ratio was independently related with the intraventricular septum and left atrial volume.[11]

Structural changes in retinal arteries may resemble those in small resistance arteries and that systemic blood pressure influences vascular structure in retinal arterioles similar to that in peripheral arteries. This is supported by the observations that, in the treated patients with well-controlled hypertension, media: lumen ratio was less than that in patients with uncontrolled hypertension, similar to data reported by others in small arteries from subcutaneous tissue.[8]

LVH (Left Ventricular Hypertrophy) may be associated with increased mortality by a number of mechanisms. Activation of the renin-angiotensin system not only leads to LVH but also may promote the progression of atherosclerosis by the effects of angiotensin II on vasomotor tone, coagulation, and vascular smooth muscle cell proliferation. Additionally, LVH may predispose to arrhythmic death. Myocardial fibrosis occurs in LVH and may provide the substrate for re-entrant ventricular arrhythmias.[12]

Generalized retinal arteriolar narrowing and arteriovenous nicking were associated with an elevation in blood pressure that had been documented six to eight years before the retinal assessment; the studies were controlled for concurrent blood-pressure levels. This association suggests that generalized narrowing and arteriovenous nicking are markers of vascular damage from chronic hypertension. In contrast, other signs (focal arteriolar narrowing, retinal hemorrhages, microaneurysms, and cotton-wool spots) were related to current but not previous blood-pressure levels and may therefore be more indicative of the severity of recent

hypertension.[13]

Patients with concentric hypertrophy (increased relative wall thickness and mass) had the highest likelihood of dying or having a cardiovascular event. Among patients with eccentric hypertrophy (normal relative wall thickness despite increased mass), a smaller percentage of patients had several, frequently nonfatal, morbid events". "Excess risk associated with increased left ventricular mass at baseline persisted for more than 10 years, despite the use of conventional antihypertensive treatment in most patients.

Narrower retinal arteriolar diameter and wider retinal venular diameter are both associated with the incidence of hypertension in the multiethnic population. As wider retinal venular diameter is associated with inflammation markers and the metabolic syndrome, these findings could explain at least partly the association of wider venules with the development of hypertension. However, in our current study, further adjustment of C-reactive protein, interleukin-6, and plasma fibrinogen level did not alter this association. Wider retinal arteriolar diameter was associated with regression of hypertension. The pathophysiological mechanisms underlying these associations are not entirely clear. Retinal arteriolar narrowing has been associated with reduced large artery compliance, which contributes to systolic hypertension.[14]

An increase in both wall thickness and WCSA (Wall Cross-Sectional Area) indicates a predominant growth response of retinal arterioles in subjects with CHF. The increase in both vessel and lumen diameter suggests a dilative component of structural alterations of retinal arterioles in subjects with CHF, as can be observed echocardiographically in subjects with CHF. Evaluation of retinal arteriolar abnormalities by means of SLDF represents an exceptional opportunity for assessment of the microcirculation non-invasively in vivo in patients with cardiac diseases. Microvascular structure and function are important surrogate

parameters for macrovascular damage and may thereby represent interesting treatment targets for new CHF therapies[15].

Treatment with ACE inhibition has been proven to increase coronary reserve during dipyridamole induced vasodilatation in essential hypertensive patients, with a parallel improvement in the structure of subcutaneous small arteries whereas β-blockade induced a marked increase in minimal myocardial vascular resistance and no change in micro vessels remodelling. Moreover, long-term therapy with the ACE inhibitor perindopril was able to induce a regression of periarteriolar fibrosis in coronary arterioles, which was associated with improvement in coronary reserve in hypertensive heart disease and a modest reduction of arteriolar wall area.[16]

Changes in the retinal vasculature, reflected as retinopathy signs, also have been shown to predict heart failure risk in the general population and in patients after coronary artery bypass surgery, further supporting a link between the microvascular process and the development of clinical heart failure. The presence of narrower retinal arterioles was associated with LV concentric remodelling. This association was consistently present across different ethnic groups; was independent of blood pressure, smoking, and other risk factors; and was seen in men and women, and even in those without diabetes, without hypertension, and with minimal coronary calcification. An association of retinopathy signs with LV concentric remodelling, but the associations were less consistent and were stronger in persons with diabetes, with hypertension, and with significant coronary calcification. The association of wider retinal venules and concentric remodelling was significant only in women[17].

The prevalence of early retinal microvascular abnormalities (arteriolar narrowing and arteriovenous crossings) is much higher than that of other validated clinical markers of TOD, such as LVH, carotid wall alterations and microalbuminuria. The relationship between retinal

microvascular abnormalities and other types of hypertension-dependent TOD, as we could show that patients with arteriovenous crossings do not have more cardiac, carotid and renal alterations compared with those without this retinal pattern. In fact, LVMI (Left Ventricular Mass Index), carotid IMT (Common Carotid Intima–Media Thickness)and UAE (Urinary Albumin Excretion) rate both when analysed as continuous variables and when calculated categorically were superimposable in patients with and without arteriovenous crossings, despite the fact that in the former group mean age was slightly but significantly higher than in the latter one[18]

In human, hypertension is associated with reduced retinal capillary density and increased retinal arterial and venous narrowing, significantly reduced thickness of the macular and ganglion cell complex but no change in the retinal nerve fiber layer thickness. Retinal nerve fiber layer thickness is reduced in hypertensive patients. That both the macular and retinal nerve fiber layer are thinner in patients with hypertension for more than five years. These results suggest that hypertension-induced retinal thinning, when noted, may possibly be mediated, at least in part, by IOP-independent mechanisms[19].

Advice regarding adequate therapy for elevated BP as well as the choice of anti-hypertensive medication is pertinent both for ophthalmologists and primary care physicians. It is possible that improved BP control in critically IOP susceptible individuals could translate into better IOP control or at least prevention of related small vessel disease. It is possible that patients currently on anti-hypertensive medications would consult a primary care physician more frequently than those without hypertension and thus be referred to an ophthalmologist and have their glaucoma diagnosed[19].

Mild cognitive impairment as an early clinical manifestation of target organ damage corresponds to left ventricular hypertrophy in hypertensive patients. Hypertensive patients

with left ventricular hypertrophy should be screened for MCI. Deterioration of cognitive functioning may be a prerequisite for poor compliance to treatment and further deterioration of hypertension control and cognitive functioning.

Retinal arteriolar narrowing is associated with higher systolic and diastolic BP but only diastolic BP was independently associated with retinal arteriolar narrowing. This indicates the importance of diastolic BP as a determinant of microvascular health in children. In adults, arteriolar narrowing is associated with incidence of hypertension and risk of coronary artery disease, and it predicts cardiovascular morbidity and mortality. Therefore, retinal arteriolar narrowing is a preclinical marker of cardiovascular risk and disease manifestation in adults[20].

Persons with hypertensive retinopathy were not only at an increased risk of developing incident stroke, but also cognitive decline, cerebral white matter lesions and cerebral atrophy, even after controlling for traditional risk factors. Additionally, generalized arteriolar narrowing and venular widening is associated with lacunar stroke, while retinopathy signs are linked to nonlacunar thrombotic and cardioembolic strokes. Retinal arteriolar narrowing is also found to be closely linked to decreased myocardial blood flow and perfusion reserve[21].

A correlation between small artery structure and both clinic and 24-hour systolic and diastolic BP, and the correlation was more strict after exclusion of patients with renovascular hypertension to rule out the possible influence of other factors, such as circulating neurohormonal factors, on remodelling in subcutaneous resistance arteries. In addition to BP, several neurohormonal factors have been suggested to impact arterial remodelling, including dietary salt, angiotensin II, endothelin-1, and insulin; this might explain why the correlation between 24-hour BP and retinal arteriolar structure was not very strict, albeit statistically significant.[22]

Hypertension is associated with retinal microvascular abnormalities including arteriolar narrowing and rarefaction. Retinal microvascular abnormalities also predict cardiovascular disease (CVD) and are associated with an increased number of cerebral white matter lesions and cognitive impairment. In view of this, it is surprising that little is known regarding the effect of antihypertensive treatment on quantitative aspects of the retinal microvasculature. Assessment of the retinal microvasculature provides a simple and non-invasive means of assessing the impact of antihypertensive treatment on the microcirculation in humans.

Arterial and venous diameters (near the optic disc) to be increased both in essential and in renovascular hypertension, and to be positively correlated with mean arterial pressure during the night and with an attenuated nocturnal blood pressure decline". Vascular density, in particular venular density, was decreased only in the patients with essential hypertension compared with age-matched normotensive control subjects and patients with renovascular hypertension, and was not correlated with blood pressure. Vascular rarefaction is an important factor in the pathogenesis of essential hypertension. However, rarefaction can also be the result of long-term hypertension. Venular density was decreased only in patients with essential hypertension and not in those with renovascular hypertension.[23]

Among patients without hypertensive retinopathy, macula VD was found to decrease in those with good BP control (< 140 mmHg), with the presence of hypertension for more than 5 years. Hypertensive patients with standard and poor BP control had lower retinal VD in some measured regions, and that only hypertensive patients with intensive BP control could maintain retinal VD consistent with that of normal subjects with 5 years of hypertension duration. SBP was correlated with reduced RNFL (Retinal Nerve Fiber Layer) thickness and inside disc capillary density, suggesting that reduced SBP may be beneficial for reducing the risk of glaucoma.[24]

Chobanian AV, Bakris GL et al., Had studied that The National High Blood Pressure Education Program presents the complete Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Like its predecessors, the purpose is to provide an evidence-based approach to the prevention and management of hypertension. The key messages of this report are these: in those older than age 50, systolic blood pressure (BP) of greater than 140 mmHg is a more important cardiovascular disease (CVD) risk factor than diastolic BP; beginning at 115/75 mm Hg, CVD risk doubles for each increment of 20/10 mm Hg; those who are normotensive at 55 years of age will have a 90% lifetime risk of developing hypertension; prehypertensive individuals (systolic BP 120 –139 mm Hg or diastolic BP 80 – 89mm Hg) require health-promoting lifestyle modifications to prevent the progressive rise in blood pressure and CVD [8]

Bhargava, M., Ikram, M. & Wong, T, Studied on hypertension has profound effects on various parts of the eye. Classically, elevated blood pressure results in a series of retinal microvascular changes called hypertensive retinopathy, comprising of generalized and focal retinal arteriolar narrowing, arteriovenous nicking, retinal hemorrhages, microaneurysms and, in severe cases, optic disc and macular edema. Studies have shown that mild hypertensive retinopathy signs are common and seen in nearly 10% of the general adult non-diabetic population. In management of patients with hypertension, physicians should be aware of the full spectrum of the relationship of blood pressure and the eye.[25]

Harjasouliha A, Raiji V et al., Had found that hypertension is the leading risk factor for the development of cardiovascular disease in 32.5% of adults over the age of 20 years. Roughly, one-third of adults in the United States have a diagnosis of hypertension and only roughly half (52%) of these individuals have their blood pressure under control. Hypertension can

damage the retinal, choroidal, and optic nerve circulations, thereby affecting both the anatomic and physiologic function of the eye.[26]

Narasimhan K, Neha VC et al., Studied that hypertensive retinopathy is a disease that damages the retina of the eye and results in loss of vision and is closely associated with high blood pressure. Severe case of hypertensive retinopathy causes systematic aliments that may cause cardiovascular diseases, heart and renal failure, loss of vision and finally death. Thus, the timely diagnosis and treatment of the disease is vital. Arteriovenous ratio is used to diagnose hypertensive retinopathy. In this paper we proposed an algorithm in which, the blood vessels are segmented out initially, from the pre-processed retinal images. Vessel width estimation method is used to measure the arteriovenous ratio from which various stages of hypertensive retinopathy can be identified. Retinal images were obtained from the VICAVR database, along with images collected form Deepam eye hospital Chennai. From the images that were collected 25 were normal images and 76 images of hypertensive retinopathy.[27]

J Physiol, Had studied that hypertension induces considerable cardiac remodelling, such as hypertrophy, interstitial fibrosis, and abnormal activity of cardiac sympathetic nervous system, which are established risk factors in several highly dangerous heart diseases, such as ventricular fibrillation and congestive heart failure. All these risk factors and heart diseases are studied extensively in isolation, but to our knowledge, there is no comprehensive review of their interactions. Finally, we show why the described mechanisms are relevant not only in hypertension, but also in the case of healed myocardial infarction.[28]

S. Kenchaiah, Pfeffer, Studied that the cardiac chambers adapt in configuration and mass to the external work requirements. In utero right and left ventricular size and configuration are relatively similar. With an infant's first breath dramatic changes in pulmonary and systemic vascular resistances occur so that the workload of the right ventricle becomes proportionally

lower than that of the left ventricle. After birth, the proportional growth of the left ventricle exceeds the right in concert with their respective evolving hemodynamic patterns. The differential growth pattern of these chambers reflects the adaptive biologic hypertrophic response that continues to match structure to workload. So too across various species of adult animals a close relationship exists between cardiac chamber weight and stroke work. These relationships between organ size and function underscore one of the basic biologic compensatory properties: inherent ability to increase or decrease mass (hypertrophy or atrophy) and to alter tissue configuration in direct relationship to functional requirements.[29]

Motz, W. Strauer, Studied that arterial hypertension, cardiac remodeling comprises myocyte hypertrophy, interstitial fibrosis, and functional and structural alterations of the coronary microcirculation. This leads to diastolic and systolic dysfunction of the left ventricle and impairment of coronary flow reserve. Consequently, antihypertensive treatment should aim at repairing hypertensive cardiac remodeling through reversing myocyte hypertrophy, restoring myocardial structure, and improving coronary flow reserve along with blood pressure normalization. Although it has been shown that regression of left ventricular hypertrophy (LVH) can be achieved by suitable antihypertensive therapy, more insight regarding the ability to repair coronary microcirculation is needed. In spontaneously hypertensive rats (SHRs), it has been shown that coronary reserve was enhanced after hydralazine administration without concomitant regression of LVH. Likewise, administration of the calciumchannel blocker felodipine led to a reversal of medial hypertrophy in coronary resistance vessels. First clinical data indicate that, after prolonged antihypertensive treatment, coronary flow reserve can be improved in hypertensive patients with microvascular disease. Further studies are warranted to elucidate whether improved coronary flow reserve after medical treatment for arterial hypertension is due to an influence of myocardial factors, such as LVH or myocardial fibrosis or to repair of the structurally remodelled microcirculation.[30]

Donald J. Weaver et al., Studied that hypertension is a well-characterized risk factor for the development of cardiovascular disease in adults. More recently, data obtained from autopsy studies as well as epidemiological longitudinal studies have demonstrated that similar end organ changes occur in children and adolescents with even mild elevations in blood pressure. These changes in early life predict the development of adult end organ disease. Specifically, chronic elevations in blood pressure in paediatric patients lead to alteration in left atrial and ventricular structure. Hypertension is also associated with impaired ventricular relaxation and decreased compliance. These cardiac changes occur in parallel with alterations in the vascular system and development of atherosclerosis. For these reasons, children with elevated blood pressures must be identified and treated appropriately in order to improve their longterm outcomes.[31]

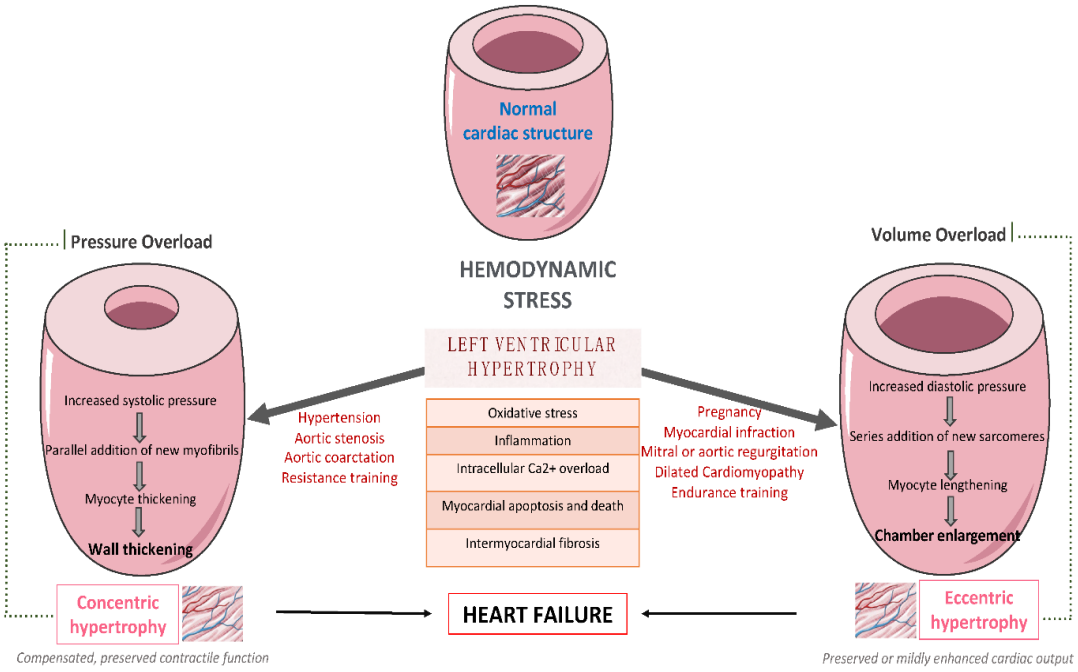

Figure 2 – Left ventricular hypertrophy causes

Cesare Cuspidi et al., Studied that the clinical value of left ventricular hypertrophy (LVH), a cardinal manifestation of hypertensive organ damage, in predicting cardiovascular (CV) events, independently of blood pressure (BP) and other accompanying risk factors, has been widely documented and its role in CV stratification indisputability recognized. Although the examination of the fundus oculi provides a unique opportunity to evaluate retinal microvascular abnormalities, which may mirror systemic arteriolar damage due to high BP, no consistent evidence exists, on the prognostic value of mild degrees of retinopathy, encompassing the vast majority of uncomplicated hypertensive subjects. Personal and literature data indicate that: (1) there is a tight association between advanced retinopathy and LVH suggesting the existence of a parallel involvement of retinal tree and cardiac damage in severe untreated or poorly controlled hypertension; (2) in contrast, a firm conclusion about the relationship between early or nonspecific retinal changes (narrowing or arteriovenous crossing) and cardiac damage is not allowed by the majority of the studies; (3) future investigations, based on computer-assisted methods, are further required to document the relation between initial retinal changes with organ damage and more importantly to test their predictive value for clinical outcomes.[32]

Chua et al., This study examined the associations between quantitative optical coherence tomography angiography (OCTA) parameters and myocardial abnormalities as documented on cardiovascular magnetic resonance imaging in patients with systemic hypertension. We conducted a cross-sectional study of 118 adults with hypertension (197 eyes). Patients underwent cardiovascular magnetic resonance imaging and OCTA (PLEX Elite 9000, Carl Zeiss Meditec). Associations between OCTA parameters (superficial and deep retinal capillary density) and adverse cardiac remodelling (left ventricular mass, remodelling index, interstitial fibrosis, global longitudinal strain, and presence of left ventricular hypertrophy) were studied using multivariable linear regression analysis with generalized estimating

equations. Of the 118 patients with hypertension enrolled (65% men; median [interquartile range] age, 59 [13] years), 29% had left ventricular hypertrophy. After adjusting for age, sex, systolic blood pressure, diabetes, and signal strength of OCTA scans, patients with lower superficial capillary density had significantly higher left ventricular mass (β=−0.150; 95% CI, −0.290 to −0.010), higher interstitial volume (β=−0.270; 95% CI, −0.535 to −0.0015), and worse global longitudinal strain (β=−0.109; 95% CI, −0.187 to −0.032). Lower superficial capillary density was found in patients with hypertension with replacement fibrosis versus no replacement fibrosis (16.53±0.64 mm-1 versus 16.96±0.64 mm-1; P=0.003). We showed significant correlations between retinal capillary density and adverse cardiac remodelling markers in patients with hypertension, supporting the notion that the OCTA could provide a non-invasive index of microcirculation alteration for vascular risk stratification in people with hypertension[11].

Mary Varghese et al., The aim of the study was to explore the association between hypertensive retinopathy, grades of retinopathy and cardiac remodelling. This was a cross-sectional observational study. A total of 500 consecutive hypertensive adults from the in-patient population were studied for the presence of hypertensive retinopathy by dilated fundoscopy. The presence of cardiac remodelling due to hypertension was studied both by electrocardiography (ECG) and echocardiography. Hypertensive target organ damage in other organs was also screened. In addition, the association of grades of hypertensive retinopathy with target organ damage was also analysed. Systolic blood pressure (BP) at presentation and duration of hypertension showed no relationship with markers of hypertensive heart disease. However, diastolic BP was significantly higher in patients with retinopathy. Hypertensive retinopathy was diagnosed in 324 subjects of whom 90 had grades 3 and 4 retinopathy. Patients with grades 3 and 4 retinopathy had significant associations with ECG evidence of left ventricular (LV) strain pattern and left atrial enlargement, and a weaker association with

left ventricular hypertrophy (LVH) using QRS voltage criteria (Sokolov–Lyon). On echocardiography, grades 3 and 4 retinopathy were significantly associated with LVH, left atrial enlargement and reduced left ventricular ejection fraction (LVEF), as well as with higher creatinine values. A large number of these patients presented with heart failure. Cardiac remodelling was not seen in patients without retinopathy and was uncommon in patients with grades 1 and 2 retinopathy. Grades 3 and 4 retinopathy demonstrated a significant association with LV strain pattern and left atrial enlargement on ECG, LVH and reduced LVEF on echocardiography as well as with heart failure. There was no relationship with systolic BP and duration of hypertension, while diastolic BP showed a significant positive correlation. Signs of hypertensive heart disease were practically absent in patients without hypertensive retinopathy and uncommon in those with grade 1–2 alterations.[14]

Tan W et al., Studied that structural and functional alterations in the microcirculation by systemic hypertension can cause significant organ damage at the eye, heart, brain, and kidneys. As the retina is the only tissue in the body that allows direct imaging of small vessels, the relationship of hypertensive retinopathy signs with development of disease states in other organs have been extensively studied; large-scale epidemiological studies using fundus photography and advanced semi-automated analysis software have reported the association of retinopathy signs with hypertensive end-organ damage includes the following: stroke, dementia, and coronary heart disease. Although yielding much useful information, the vessels assessed from fundus photographs remain limited to the larger retinal arterioles and venules, and abnormalities observed may not be that of the earliest changes. Newer imaging modalities such as optical coherence tomography angiography and adaptive optics technology, which allow a greater precision in the structural quantification of retinal vessels, including capillaries, may facilitate the assessment and management of these patients. In the future, combining deep learning systems with the imaging precision offered by optical

coherence tomography angiography and adaptive optics could pave way for systems that are able to predict adverse clinical outcomes even more accurately.[33]

González A et al., Studied that arterial hypertension has been related to multiple outcomes, including cardiac, cerebral, and renal. The burden of arterial hypertension remains high, despite the availability of preventive interventions and low-cost, effective antihypertensive medications. Therefore, to hypertension burden, beyond new effective health strategies, novel pathophysiological and clinical approaches are also required.[2]

Di Marco E et al., Studied that hypertensive retinopathy (HR) is the most common ocular manifestation of systemic arterial hypertension. This paper aims to summarize the current knowledge of HR, reviewing its classical features, such as epidemiology, pathophysiology, clinical manifestations, classifications, management and the most significant systemic correlations. We also provide an update on the latest advances in new technologies focusing on novel instrumental classifications. HR signs have a significative association with cardiovascular, cerebrovascular and other systemic diseases. Patients with arteriosclerotic changes and, at the same time, severe HR, are at increased risk for coronary disease, peripheral vascular disease, stroke and dementia. HR is even now diagnosed and classified by its clinical appearance on a fundoscopic exam that is limited by interobserver variability. New technologies, like OCT, OCTA, AO and artificial intelligence may be used to develop a new instrumental classification that could become an objective and quantitative method for the evaluation of this disease. They could be useful to evaluate the subclinical retinal microvascular changes due to hypertension that may reflect the involvement of other vital organs. The eye is the only organ in the human body where changes in the blood vessels due to systemic hypertension can be studied in vivo.[34]

Wong TY, Mitchell P, Studied that hypertensive retinopathy is a condition characterized by a spectrum of retinal vascular signs in people with elevated blood pressure. The detection of hypertensive retinopathy with the use of an ophthalmoscope has long been regarded as part of the standard evaluation of persons with hypertension. This clinical practice is supported by both previous and current reports of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC), which list retinopathy as one of several markers of target organ damage in hypertension. On the basis of the JNC criteria, the presence of retinopathy may be an indication for initiating antihypertensive treatment, even in people with stage 1 hypertension (blood pressure, 140 to 159/90 to 99 mm Hg) who have no other evidence of target-organ damage.[35]

Koren MJ et al., To assess the prognostic significance of left ventricular mass and geometry in initially healthy persons with essential hypertension. An observational study of a prospectively identified cohort. University medical center. Two hundred and eighty patients with essential hypertension and no pre-existing cardiac disease were evaluated using echocardiography between 1976 and 1981. Two hundred and fifty-three subjects or their family members (90%) were contacted for a follow-up interview an average of 10.2 years after the initial echocardiogram was obtained; the survival status of 27 patients lost to follow-up was ascertained using National Death Index data. Left ventricular mass exceeded 125 g/m2 in 69 of 253 patients (27%). Cardiovascular events occurred in a higher proportion of patients with than without left ventricular hypertrophy (26% compared with 12%; P = 0.006). Patients with increased ventricular mass were also at higher risk for cardiovascular death (14% compared with 0.5%; P < 0.001) and all-cause mortality (16% compared with 2%; P = 0.001). Electrocardiographic left ventricular hypertrophy did not predict risk. Patients with normal left ventricular geometry had the fewest adverse outcomes (no cardiac deaths; morbid events in 11%), and those with concentric hypertrophy had the most (death in 21%; morbid

events in 31%). In a multivariate analysis, only age and left ventricular mass—but not gender, blood pressure, or serum cholesterol level—independently predicted all three outcome measures.[36]

Kawasaki R et al., To describe the prospective relationship of retinal vessel diameters with risk of hypertension in a multiethnic population-based cohort The Multi-Ethnic Study of Atherosclerosis is a population-based study of subclinical cardiovascular disease among white, African–American, Hispanic, and Chinese American adults aged 45–84 years. Retinal vessel diameters were measured using a standardized imaging software at the second examination (considered baseline in this analysis) and summarized as the central retinal artery/vein equivalent. Presence of retinopathy and retinal focal arteriolar narrowing and arteriovenous nicking was assessed by trained graders. Incidence of hypertension was defined among participants at risk as systolic blood pressure at least 140 mmHg, diastolic blood pressure at least 90 mmHg, or use of an antihypertensive medication. Of the initial 6237 participants at baseline, 2583 were at risk of hypertension.[37]

Lehmann MV, Schmieder RE, Studied that vascular dysfunction due to elevated blood pressure constitutes an early step in the pathogenesis of atherosclerotic disease. A better understanding of the pathophysiology and of clinical correlates of vascular remodeling in retinal arteries and arterioles offers the opportunity for a better risk stratification and treatment. In vivo vascular changes can be best detected by direct imaging techniques. In this review, we summarize the main findings of several recent studies analyzing retinal-arteriolar parameters, such as outer diameter (OD) and lumen diameter (LD), retinal capillary flow (RCF), wall-to-lumen-ratio, and wall cross-sectional area by using scanning laser Doppler flowmetry (SLDF). Blood pressure emerged as an independent determinant of the wall-to-lumen ratio (WLR) of retinal arterioles.[38]

Jung S et al., Studied that analysis of microvascular parameters in the retinal circulation—known to reflect those in the systemic circulation —allows us to differentiate between eutrophic and hypertrophic remodelling of small arteries. Applying a matched pair approach, we compared this group with reference values of age-matched controls from a random sample in the population of Pilsen, Czech Republic. There was no significant difference in RCF and WLR between the groups (RCF: P = 0.513; WLR: P = 0.106). In contrast, wall thickness and WCSA, indicators of hypertrophic remodelling, were higher in CHF subjects (WT: 15.0 ± 4.2 vs. 12.7 ± 4.2 μm, P = 0.021; WCSA: 4437.6 ± 1314.5 vs. 3615.9 ± 1567.8 μm2 , P = 0.014). Similarly, vessel (109.4 ± 11.1 vs. 100.5 ± 14.4 μm, P = 0.002) and lumen diameter (79.0 ± 7.9 vs. 75.2 ± 8.5 μm, P = 0.009) were increased in CHF.[39]

Rizzoni D et al., Studied that although the gold-standard method for the assessment of structural alteration in small resistance arteries is the evaluation of the MLR by micromyography in bioptic tissues, new, noninvasive techniques are presently under development, focusing mainly on the evaluation of WLR in retinal arterioles. These approaches represent a promising and interesting future perspective.[40]

Sadowski J et al., Stated that current data indicate that heart failure (HF) is associated with inflammation and microvascular dysfunction and remodelling. These mechanisms could be involved in HF development and progression, especially in HF with preserved ejection fraction (HFpEF). We aimed to compare structural changes in retinal arterioles and carotid arteries between HF patients and patients without heart failure. This preliminary, retrospective, case-control study included 28 participants (14 patients with HFpEF and 14 age- and sex-matched healthy controls). Carotid intima-media thickness to lumen ratio (cIMTLR) was assessed using B-mode ultrasonography. Retinal arterioles wall- to-lumen ratio (rWLR) was assessed by adaptive optics camera rtx1. The HF patients had higher

IMTLR (Δmedian [HFpEF–control group] 0.07, p = 0.01) and eWLR (Δmedian 0.03, p = 0.001) in comparison to patients without HF. In the whole study group, rWLR correlated significantly with IMTLR (r = 0.739, p = 0.001). Prevalence of arterial hypertension was similar in both groups, however, patients with HF had a significantly lower office, central and 24-h ambulatory blood pressure (systolic Δmedian −21 to −18 mmHg; diastolic Δmedian −23 to −10 mmHg).[41]

Cuspidi C et al., Studied that cardiac and extracardiac hypertensive target organ damage (TOD) is recognized as an intermediate step in the continuum of cardiovascular disease and a powerful independent predictor of cardiovascular morbidity, mortality and all-cause deaths. Furthermore, regression or reduction of TOD is increasingly regarded as a useful intermediate endpoint for assessing the efficacy of blood pressure (BP)-lowering medications.[42]

Goh VJ et al., Studied that hypertensive left ventricular hypertrophy (HTN-LVH) is a leading cause of heart failure. Conventional patterns of cardiac geometry do not adequately risk-stratify patients with HTN-LVH. Using cardiovascular magnetic resonance, we developed a novel Remodelling Index (RI) that was designed to detect an exaggerated hypertrophic response to hypertension and tested its potential to risk-stratify hypertensive patients". "The RI was derived using LaPlace's Law, and normal RI ranges were established in 180 healthy volunteers. The utility of the RI was examined in 256 asymptomatic hypertensive patients and 10 patients with heart failure with preserved ejection fraction. Hypertensive patients underwent multimodal cardiac assessment: contrast-enhanced cardiovascular magnetic resonance, echocardiograms, 24-hour blood pressure monitoring, and cardiac biomarkers (high-sensitivity cardiac troponins, NT-proBNP [N-terminal pro-B-type natriuretic peptide], and galectin-3). Blood pressure accounted for only 20% of the variance observed in LV mass. Although there was no association between blood pressure and

myocardial fibrosis, LV mass was independently associated with fibrosis. Compared with hypertensive patients without LVH (n=191; 74.6%) and those with HTN-LVH and normal RI (n=50; 19.5%), patients with HTN-LVH and low RI (HTN-LVH/low RI; n=15, 5.9%) had an amplified myocardial response: elevated indexed LV masses (83±24 g/m^2), more fibrosis (73%), and higher biomarkers of myocardial injury and dysfunction (P<0.05 for all). RI was similar in HTN-LVH/low RI and heart failure with preserved ejection fraction (4.1 [3.4-4.5] versus 3.7 [3.4-4.0], respectively; P=0.15). We suggest that RI provides an approach for stratifying hypertensive patients and is suitable for testing in other disease cohorts to assess its clinical utility.[43]

Feihl F et al., In the present review, microvascular remodelling refers to alterations in the structure of resistance vessels contributing to elevated systemic vascular resistance in hypertension. We start with some historical aspects, underscoring the importance of Folkow's contribution made half a century ago. We then move to some basic concepts on the biomechanics of blood vessels, and explicit the definitions proposed by Mulvany for specific forms of remodelling, especially inward eutrophic and inward hypertrophic. The available evidence for the existence of remodelled resistance vessels in hypertension comes next, with relatively more weight given to human, in comparison with animal data. Mechanisms are discussed". "The impact of antihypertensive drug treatment on remodelling is described, again with emphasis on human data. Some details are given on the three studies to date which point to remodelling of subcutaneous resistance arteries as an independent predictor of cardiovascular risk in hypertensive patients. We terminate by considering the potential role of remodelling in the pathogenesis of end organ damage and in the perpetuation of hypertension.[44]

Cheung N et al., This study sought to examine the relationships of retinal vascular signs with left ventricular (LV) mass, volume, and concentric remodelling. Microvascular disease, reflected as retinopathy lesions, has been shown to predict clinical congestive heart failure. Whether these retinal vascular changes are related to early structural alterations and remodelling of the heart in asymptomatic individuals is unknown. A cross-sectional, population-based study of 4,593 participants ages 45 to 85 years, free of clinical cardiovascular disease. Retinal vascular calibers and retinopathy were graded from retinal photographs according to standardized protocols. The LV mass and volume were measured from cardiac magnetic resonance imaging. Extent of LV concentric remodelling was determined by the ratio of LV mass to end-diastolic volume (M/V ratio). After controlling for age, gender, race, center, past and current systolic blood pressure, body mass index, smoking, antihypertensive medications, diabetes, diabetes duration, glycosylated hemoglobin, lipid profile, and C-reactive protein, narrower retinal arteriolar caliber was associated with concentric (highest quintile of M/V ratio) remodelling (odds ratio [OR] 2.06, 95% confidence interval 1.57 to 2.70). This association was seen in men and women, and was present even in those without diabetes, without hypertension, and without significant coronary calcification. In multivariate analysis, the presence of retinopathy (OR 1.31, 95% confidence interval 1.08 to 1.61) was also associated with concentric remodelling. Narrower retinal arteriolar caliber is associated with LV concentric remodelling independent of traditional risk factors and coronary atherosclerotic burden, supporting the hypothesis that microvascular disease may contribute to cardiac remodelling[45].

Cuspidi C et al., The clinical and prognostic significance of initial retinal alterations in hypertensive patients remains controversial. Therefore, we assessed the relationship of microvascular abnormalities with prognostically validated markers of target organ damage (TOD), such as left ventricular mass (LVM), carotid intima– media thickness (IMT) and

microalbuminuria, in early stages of untreated essential hypertension. A total of 437 consecutive, never-treated patients with grade 1 or 2 essential hypertension, referred to our outpatient clinic, underwent the following procedures: (1) clinical and routine laboratory examinations, (2) 24-h ambulatory blood pressure monitoring, (3) 24-h urine collection for microalbuminuria, (4) echocardiography, (5) carotid ultrasonography, (6) non-mydriatic retinography. Patients were divided into group I, with either a normal retinal pattern (n 65, 14.9%) or arteriolar narrowing (n 185, 42.4%) and group II with arteriovenous crossings (n 187, 42.7%). The two groups were similar for gender, body mass index, smoking habit, heart rate, clinic and ambulatory blood pressure (BP) values, while mean age was slightly but significantly higher in group II than in group I (47.6 6 10.7 versus 44.5 6 12.5 years, P 0.008). No differences occurred between the two groups in LVM index (101.8 6 18.5 versus 99.9 6 20.4 g/m2), carotid IMT (0.67 6 0.12 versus 0.66 6 0.20 mm), urinary albumin excretion rate (14.4 6 27.7 versus 13.3 6 27.7 mg/24 h) as well as in the prevalence of LV hypertrophy (14.3 versus 14.0%), IM thickening and/or plaques (26.5 versus 27.2%) (both defined according to 2003 ESH-ESC guidelines) and microalbuminuria (10.1 versus 8.7%). Furthermore, the three different retinal artery patterns were similarly distributed among tertiles of LV mass index, IMT and urinary albumin excretion rate. These results show that: (1) a very large fraction (more than 80%) of untreated, recently diagnosed hypertensive patients have initial retinal microvascular abnormalities detectable by non-mydriatic retinography, (2) the presence of arteriovenous crossings is not associated with more prominent cardiac and extracardiac TOD, (3) fundoscopic examination has a limited clinical value to detect widespread organ involvement in early phases of grade 1 and 2 hypertension.[45]

Gallo A et al., To research a retinal arterioles wall-to-lumen ratio or lumen diameter cut-off that would discriminate hypertensive from normal subjects using adaptive optics camera. One thousand and five hundred subjects were consecutively recruited and Adaptive Optics

Camera rtx1TM (ImagineEyes, Orsay, France) was used to measure wall thickness, internal diameter, to calculate wall-to-lumen ratio (WLR) and wall cross-sectional area of retinal arterioles. Sitting office blood pressure was measured once, just before retinal measurements and office blood pressure was defined as systolic blood pressure > = 140 mmHg and diastolic blood pressure > = 90 mmHg. ROC curves were constructed to determine cut-off values for retinal parameters to diagnose office hypertension. In another population of 276 subjects office BP, retinal arterioles evaluation and home blood pressure monitoring were obtained. The applicability of retinal WLR or diameter cut-off values were compared in patients with controlled, masked, white-coat and sustained hypertension. In 1500 patients, a WLR> 0.31 discriminated office hypertensive subjects with a 0.57 sensitivity and 0.71 specificity. Lumen diameter < 78.2m discriminated office hypertension with a 0.73 sensitivity and a 0.52 specificity. In the other 276 patients, WLR was higher in sustained hypertension vs normotensive patients (0.330 ± 0.06 vs 0.292 ± 0.05; P < 0.001) and diameter was narrower in masked hypertensive vs normotensive subjects (73.0 ± 11.2 vs 78.5 ± 11.6m; P < 0.005). A WLR higher than 0.31 is in favour of office arterial hypertension; a diameter under < 78m may indicate a masked hypertension. Retinal arterioles analysis through adaptive optics camera may help the diagnosis of arterial hypertension, in particular in case of masked hypertension[46].

Cui Y et al.,Systemic hypertension or hypertension is a very common chronic age-related disease worldwide. It is typically characterized by a sustained elevation of blood pressure, particularly when the systolic blood pressure and/or diastolic blood pressure are of more than 140 mmHg and 90 mmHg, respectively. If hypertension is not well controlled, it may lead to an increased risk of stroke and heart attack. It has been shown that hypertension is linked with various ocular diseases, including cataract, diabetic retinopathy, age-related macular degeneration, and glaucoma. Glaucoma is the leading cause of irreversible blindness

worldwide. Primary open angle glaucoma is the most common form of the disease and is usually characterized by an increase in intraocular pressure (IOP). This condition, together with normal tension glaucoma, constitutes open angle glaucoma. Systemic hypertension has been identified as a risk factor for open angle glaucoma. It is speculated that blood pressure is involved in the pathogenesis of open angle glaucoma by altering IOP or ocular blood flow, or both. Recent evidence has shown that both extremely high and low blood pressure are associated with increased risk of open angle glaucoma. Additional pathogenic mechanisms, including increased inflammation likely to be involved in the development and progression of these two diseases, are discussed[46].

MATERIALS AND METHODS

MATERIALS AND METHODS

This prospective cross sectional observational study was conducted on 88 patients fulfil the inclusion criteria in the Department of Ophthalmology, R. L. Jalappa Hospital and Research, Kolar from May 2023 to November 2024, after obtained ethical clearance from Institutional Ethical Committee of Sri Devaraj URS Medical College and written informed consent from the subjects.

PLACE OF STUDY:

The present study was carried out in the Department of Ophthalmology, R. L. Jalappa Hospital and Research, Kolar.

TYPE OF STUDY:

The present study was prospective cross sectional observational study.

DURATION OF STUDY:

The study was carried out for a period of 18 months.

SAMPLE SIZE:

The study was conducted on 88 patients.

INCLUSION CRITERIA:

Detailed clinical assessment of patients including:

All patients who will be diagnosed to have hypertension according to the Joint National Commission 7 guidelines.

EXCLUSION CRITERIA:

Patients meeting the following criteria were excluded from the study.

Patients with other causes of retinopathy like:

1. Diabetic retinopathy
2. Anaemia
3. Leukaemia
4. Hyper coagulable states
5. Radiation

METHOD OF COLLECTION OF DATA:

A total of 88 patients who fulfil the inclusion criteria were included in this study. After obtaining a detailed history each patient was clinically examined for the following:

1. Visual acuity assessment by using Snellen chart
2. Slit lamp biomicroscopy for evaluation of anterior segment
3. Posterior segment evaluation by indirect ophthalmoscopy and +90D biomicroscopy by two ophthalmologists who will be blinded to the findings of hypertensive target organ damage. Hypertensive retinopathy is graded according to the Keith–Wagener–Barker classification.

Grade	Features
I	Slight narrowing, sclerosis, and tortuosity of retinal arterioles
II	Definite narrowing, focal constriction, sclerosis of arterioles & AV nicking
III	Retinal hemorrhages, exudates and cotton wool spots
IV	Severe grade III and papilledema

4. Body mass index will be calculated as weight (kg) divided by height (m2).
5. Blood investigations: Random blood sugar, lipid profile (serum total cholesterol, high-density lipoprotein cholesterol), creatine urea, creatinine and urine tests for casts and albumin. The estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease equation modified by the Japanese coefficient (0.881). [14]

   eGFR =0.881×186×(age)-0.203×(serum creatinine)-1.154×(0.742 for women)
6. Recording blood pressure (BP)
7. Screening for cardiac remodeling will include as:

- Hypertensive LV remodeling by ECG and 2D echocardiography criteria. 2D echocardiography will be carried out in all patients according to American Society of Echocardiography (ASE) recommendations. [15]
- LV dimensions in systole and diastole
- LV end diastolic volume and end systolic volume
- Left atrial dimension.
- The left ventricular ejection fraction (LVEF) will be calculated according to Simpson's rule (measurement of LVEF by tracing the endocardial border in both the apical four-chamber and two-chamber views in end-systole and end-diastole).
- Other target organ damage will also be noted.

INFORMED CONSENT

All the patients fulfilling selection criteria were explained about the details of the disease process, options of treatment, ultimate outcome, possible effects, complications and chances of recurrence in both procedure and a written informed consent was obtained before enrolment. They were informed of their right to withdraw from the study at any stage.

SAMPLE SIZE ESTIMATION

Sample size was estimated by using the proportion of who had hypertensive retinopathy in subjects who had systemic hypertension was 64.8% from the study by Mary Varghese et al.[15] using the formula

Sample Size = $Z_{1-\alpha/2}{}^2 \, P\,(1-P) \,/\, d^2$

$Z_{1-\alpha/2}$ = is standard normal variate (at 5% type 1 error (P<0.05) it is 1.96 and at 1% type1 error(P<0.01) it is 2.58). As in majority of studies P values are considered significant below 0.05 hence 1.96 is used in formula.

P= Expected proportion in population based on previous studies or pilot studies

d= Absolute error or precision

P = 64.8% or 0.648

q = 35.2 or 0.352

d = 10% or 0.10

Using the above values at 95% Confidence level a sample size of **88** subjects will be included in the study.

STATISTICAL ANALYSIS

The collected data was entered into Microsoft Excel Worksheet-2010 and data was taken into IBM SPSS Statistic for windows, version 24 (IBM Corp., Armonk, N.Y., USA) software for calculation of frequency, percentage, mean, standard deviation and probability value.

Qualitative data was represented in the form of frequency and percentage.

- Association between qualitative variables was assessed by Chi Square test with continuity correction for 2 x 2 tables and
- Fisher's exact test for all 2 x 2 tables, where P value of chi square test was not valid due to small counts.

Quantitative data was represented using mean and standard deviation.

- Analysis of quantitative data within the groups was done using paired t test if data passes 'Normality test'.
- One Way Analysis (ANOVA) was used to compare more than two groups.

A **'P'** value of <0.05 was considered statistically significant.

RESULTS

RESULTS

Table 1: Distribution of subjects based on fundus changes Vs age group.

Age group (years)	Frequency	Percentage
21-30	5	5.68
31-40	36	40.91
41-50	27	30.68
51-60	20	22.73
Total	**88**	**100**

Table 1 summarizes the age distribution of study participants diagnosed with systemic hypertension. The majority of patients fall within the 31–40 years age group, accounting for 40.91% of the study population, followed by 41–50 years (30.68%), 51–60 years (22.73%), and a smaller proportion in the 21–30 years group (5.68%). This distribution suggests that hypertensive changes, including potential retinopathy and cardiac remodeling, are more prevalent or detected more frequently in middle-aged adults, highlighting the need for early cardiovascular and ocular screening in this age bracket.

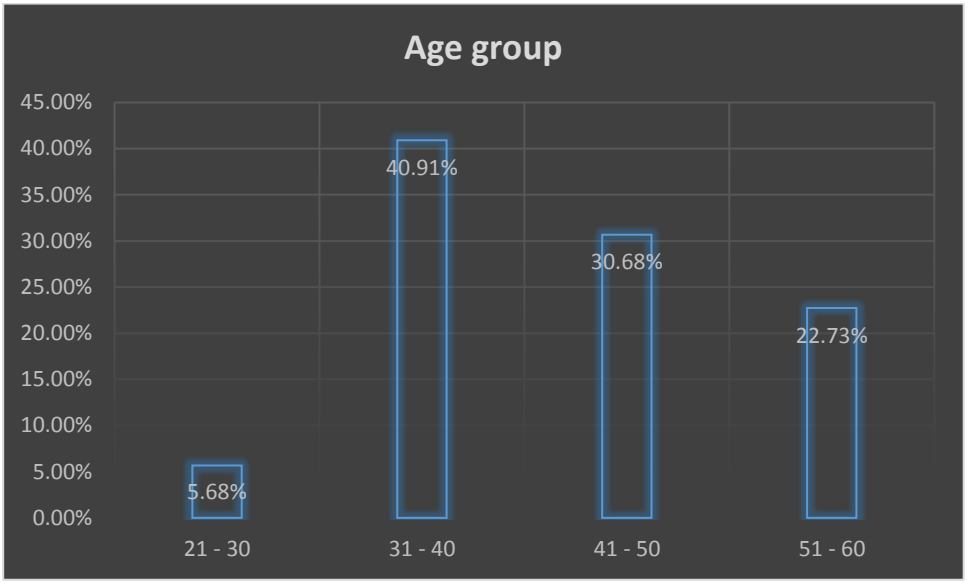

Fig 3: Distribution of subjects based on fundus changes Vs age group.

Table 2: Distribution of subjects based on sex .

Sex	Frequency	Percentage
Male	51	57.95
Female	37	42.05
Total	**88**	**100**

Table 2 : Sex Distribution: Of the total participants, 57.95% were male and 42.05% were female, indicating a slight male predominance in the study population. This may reflect either a higher prevalence of diagnosed hypertension among males or differences in health-seeking behaviour.

Table 3: Distribution of subjects based on Hypertensive Retinopathy Stages.

Hypertensive Retinopathy Stages	Frequency	Percentage
Stage 0	21	23.86
Stage 1	20	22.73
Stage 2	19	21.59
Stage 3	17	19.32
Stage 4	11	12.5
Total	**88**	**100**

Table 3:Hypertensive Retinopathy Staging: The staging of hypertensive retinopathy, based on standard classification, shows that 23.86% of patients had Stage 0 (no visible retinal changes), while Stage 1 and Stage 2 retinopathy were present in 22.73% and 21.59% of patients, respectively. Advanced stages—Stage 3 and Stage 4—were noted in 19.32% and 12.5% of patients, respectively. This progression underscores the chronic impact of systemic hypertension on retinal microvasculature and its potential as a marker for systemic vascular damage.

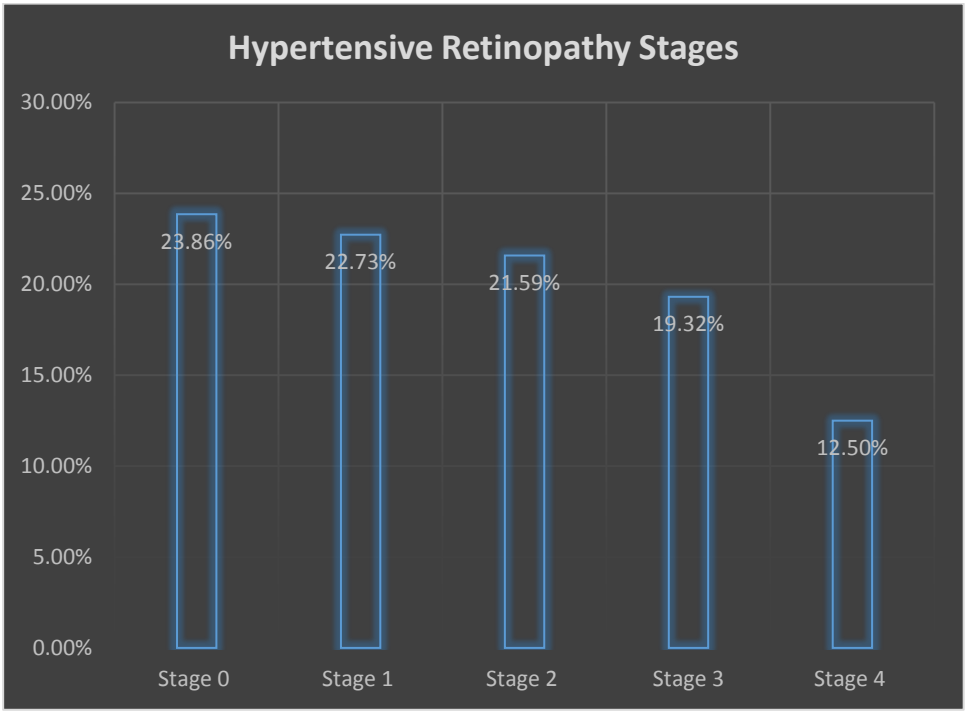

Fig 4: Distribution of subjects based on Hypertensive Retinopathy Stages

Table 4: Comparison of SBP and DBP between Hypertensive Retinopathy (HR) Stages.

Hypertensive Retinopathy Stages	SBP Mean ± Sd	DBP Mean ± Sd
Stage 0	126.87 ± 11.54	81.62 ± 4.67
Stage 1	134.64 ± 10.87	84.67 ± 5.37
Stage 2	142.37 ± 12.37	91.37 ± 6.72
Stage 3	154.37 ± 16.52	94.65 ± 7.24
Stage 4	158.84 ± 15.63	98.34 ± 6.54

Table 4:Association with Blood Pressure: A clear upward trend is observed in both systolic and diastolic blood pressure values across advancing stages of hypertensive retinopathy.

Patients with Stage 0 had a mean SBP of 126.87 ± 11.54 mmHg and DBP of 81.62 ± 4.67 mmHg. In contrast, those with Stage 4 exhibited significantly higher values, with a mean SBP of 158.84 ± 15.63 mmHg and DBP of 98.34 ± 6.54 mmHg. This progression reinforces the direct relationship between elevated blood pressure and severity of retinal vascular changes, highlighting the importance of early BP control to prevent end-organ damage.

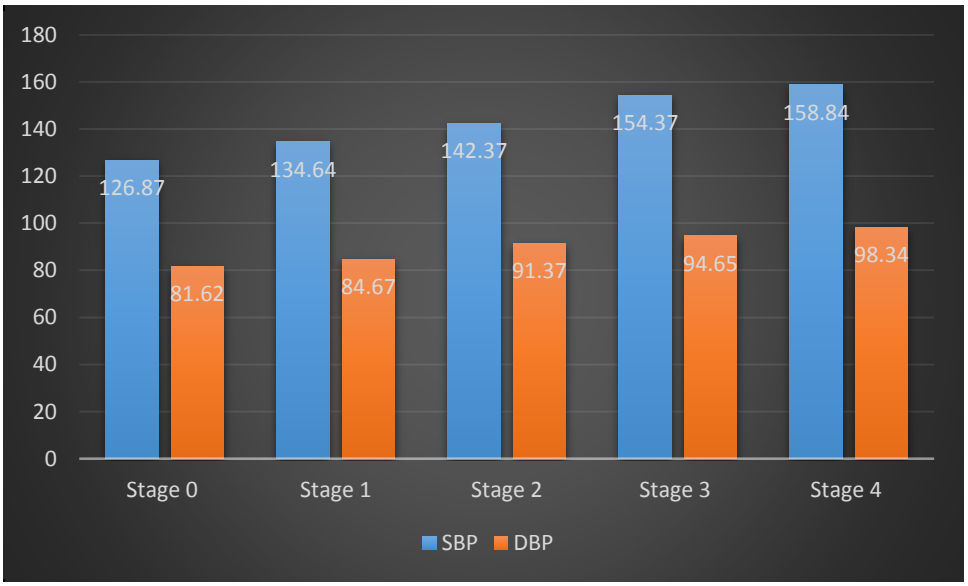

Fig 5: Comparison of SBP and DBP between Hypertensive Retinopathy (HR) Stages

Table 5: Correlation Between Hypertensive Retinopathy (HR) Stages and ECG Findings of Left Ventricular Hypertrophy (LVH).

Hypertensive Retinopathy Stages	Left Ventricular Hypertrophy (LVH) N (%)	Odds ratio with 95% CI
Stage 0 (21)	3 (14.29)	Reference
Stage 1 (20)	5 (25.00)	2 (0.41-9.78)
Stage 2 (19)	7 (36.84)	3.5 (0.75-16.28)
Stage 3 (17)	11 (64.71)	11 (2.28-53.18)
Stage 4 (11)	9 (81.82)	27 (3.8-191.67)

Table 5:Association with Left ventricular hypertrophy (LVH) : The prevalence of left ventricular hypertrophy increased significantly with advancing stages of hypertensive retinopathy: Stage 0: 14.29% (reference group), Stage 1: 25.00% (OR: 2; 95% CI: 0.41–9.78), Stage 2: 36.84% (OR: 3.5; 95% CI: 0.75–16.28), Stage 3: 64.71% (OR: 11; 95% CI: 2.28–53.18) and Stage 4: 81.82% (OR: 27; 95% CI: 3.8–191.67)

This pattern shows a strong and statistically significant association between the severity of hypertensive retinopathy and the likelihood of developing LVH. The increasing odds ratios

with narrow confidence intervals in advanced stages suggest a robust correlation, indicating that retinal microvascular changes which serve as a surrogate marker for cardiac structural remodelling in hypertensive patients.

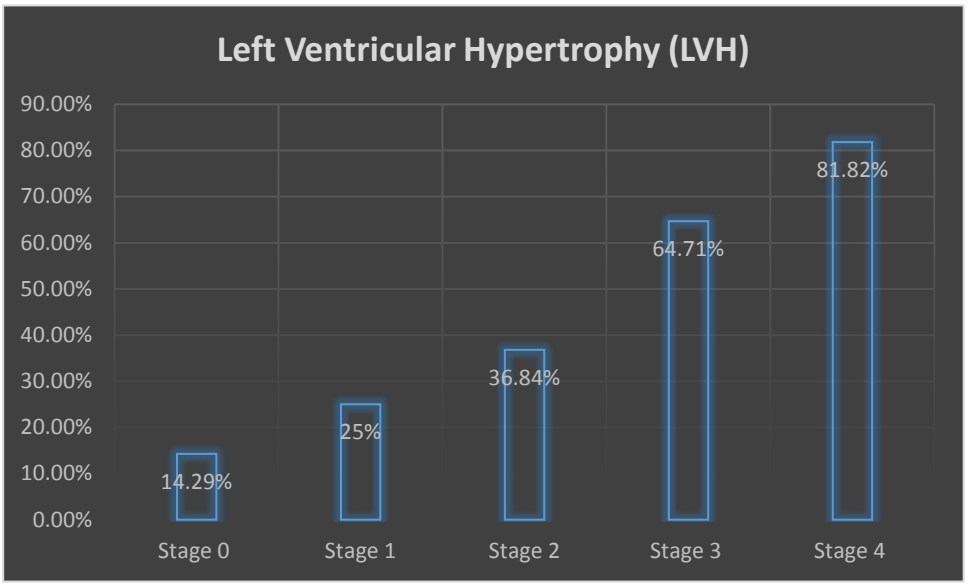

Fig 6: Correlation Between Hypertensive Retinopathy (HR) Stages and ECG Findings of Left Ventricular Hypertrophy (LVH)

Table 6: Comparison of left ventricular mass index in Echocardiographic Findings between Hypertensive Retinopathy (HR) Stages.

Hypertensive Retinopathy Stages	left ventricular mass index Mean ± Sd	P-values
Stage 0	84.32 ± 11.64	
Stage 1	90.18 ± 12.67	0.03
Stage 2	96.52 ± 14.67	0.001
Stage 3	107.37 ± 16.87	0.0001
Stage 4	112.67 ± 17.64	0.0001

Table 6:Left Ventricular Mass Index (LVMI): Mean LVMI values also showed a statistically significant increasing trend across retinopathy stages: Stage 0: 84.32 ± 11.64, Stage 1: 90.18 ± 12.67 (p = 0.03), Stage 2: 96.52 ± 14.67 (p = 0.001), Stage 3: 107.37 ± 16.87 (p = 0.0001) and Stage 4: 112.67 ± 17.64 (p = 0.0001)

The progressive rise in LVMI with increasing severity of hypertensive retinopathy underscores the parallel advancement of structural cardiac changes. The p-values indicate statistically significant differences compared to Stage 0, supporting the clinical relevance of retinal findings as predictive indicators of myocardial remodelling.

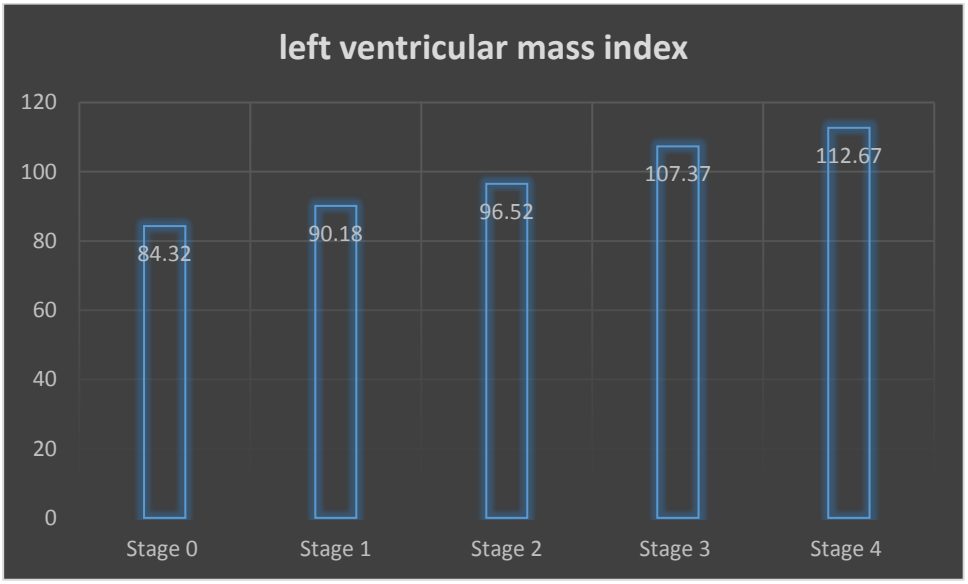

Fig 7: Comparison of left ventricular mass index in Echocardiographic Findings between Hypertensive Retinopathy (HR) Stages

Table 7: Comparison of Relative wall thickness in Echocardiographic Findings between Hypertensive Retinopathy (HR) Stages.

Hypertensive Retinopathy Stages	Relative wall thickness	
	Mean ± Sd	P-values
Stage 0	0.35 ± **0**.04	
Stage 1	0.41 ± 0.06	0.07
Stage 2	0.44 ± 0.05	0.04
Stage 3	0.47 ± 0.05	0.001
Stage 4	0.52 ± 0.06	0.0001

Table 7:Relative Wall Thickness (RWT): RWT also increased with retinopathy severity, reflecting progressive concentric hypertrophy: Stage 0: 0.35 ± 0.04, Stage 1: 0.41 ± 0.06 (p = 0.07), Stage 2: 0.44 ± 0.05 (p = 0.04), Stage 3: 0.47 ± 0.05 (p = 0.001) and Stage 4: 0.52 ± 0.06 (p = 0.0001). While the increase from Stage 0 to Stage 1 was not statistically significant (p = 0.07), all subsequent stages showed significant differences, particularly in advanced retinopathy.

These findings demonstrate a clear and statistically significant association between the severity of hypertensive retinopathy and parameters of cardiac remodelling, including elevated LVMI and RWT. Retinal microvascular changes appear to mirror myocardial

structural alterations, emphasizing the value of routine fundus examinations in risk stratifying hypertensive patients for subclinical cardiac involvement.

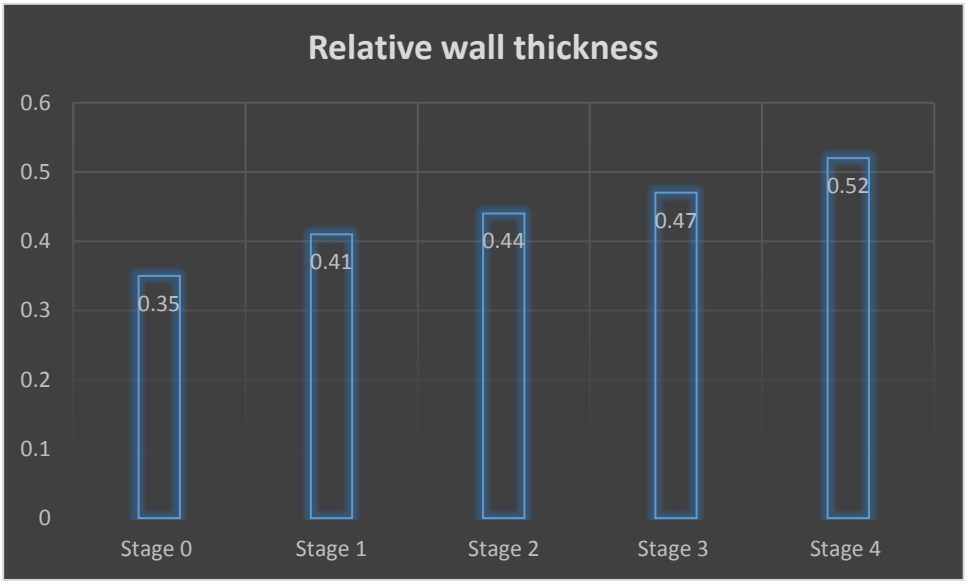

Fig 8: Comparison of Relative wall thickness in Echocardiographic Findings between Hypertensive Retinopathy (HR) Stages

Table 8: Correlation Between Hypertensive Retinopathy (HR) Stages and Echocardiographic Findings of Concentric remodelling.

Hypertensive Retinopathy Stages	Concentric remodeling N (%)	Odds ratio with 95% CI
Stage 0 (21)	3 (14.28)	Reference
Stage 1 (20)	4 (20)	1.5 (0.29-7.74)
Stage 2 (19)	9 (47.37)	5.4 (1.18-24.65)
Stage 3 (17)	13 (76.47)	19.5 (3.71-102.38)
Stage 4 (11)	10 (90.91)	60 (5.49-655.82.67)

Table 8:The prevalence of concentric remodelling rose markedly from 14.28% in Stage 0 to 90.91% in Stage 4. Odds Ratios indicated a strong and statistically significant association: Stage 2: OR 5.4 (95% CI: 1.18–24.65), Stage 3: OR 19.5 (95% CI: 3.71–102.38) and Stage 4: OR 60 (95% CI: 5.49–655.82). These findings show that advanced hypertensive retinopathy is a powerful predictor of concentric remodelling, reflecting increased cardiovascular risk.

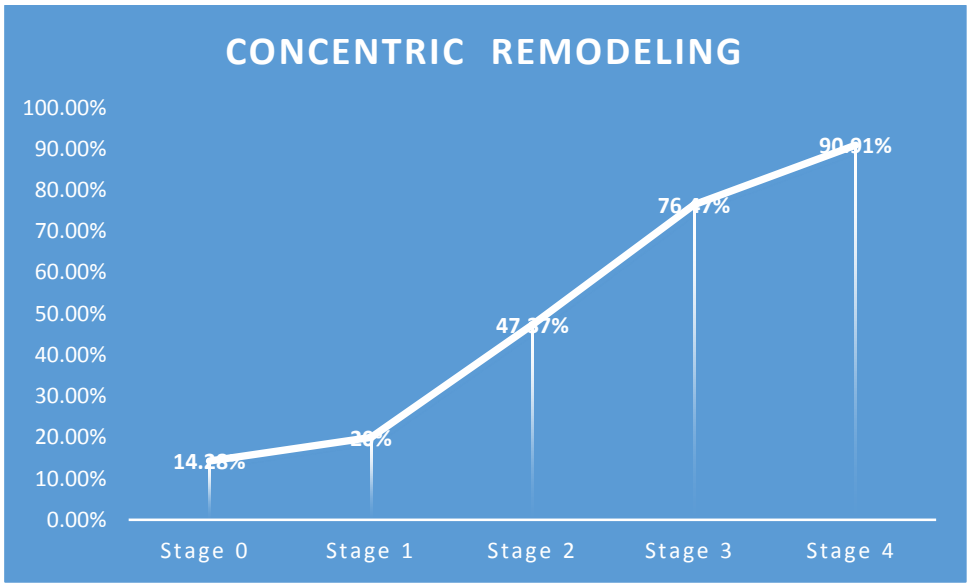

Fig 9: Relationship Between Hypertensive Retinopathy (HR) Stages and Echocardiographic Findings of Concentric remodelling

Table 9: Relationship Between Hypertensive Retinopathy (HR) Stages and Echocardiographic Findings of Eccentric hypertrophy.

Hypertensive Retinopathy Stages	Eccentric hypertrophy N (%)	Odds ratio with 95% CI
Stage 0 (21)	2 (9.52)	Reference
Stage 1 (20)	2 (10)	1.06 (0.13-8.31)
Stage 2 (19)	5 (26.32)	3.39 (0.57-20.1)
Stage 3 (17)	5 (29.41)	3.96 (0.66-23.76)
Stage 4 (11)	4 (36.36)	5.43(0.81-36.51)

Table 9:A gradual increase in eccentric hypertrophy was observed, though with less statistical strength: Stage 0: 9.52% and Stage 4: 36.36% Odds ratios increased, but confidence intervals remained wide and non-significant: Stage 4: OR 5.43 (95% CI: 0.81–36.51)

This suggests a trend toward increased eccentric remodelling with advancing retinopathy, but further investigation with a larger sample may be needed for statistical confirmation.

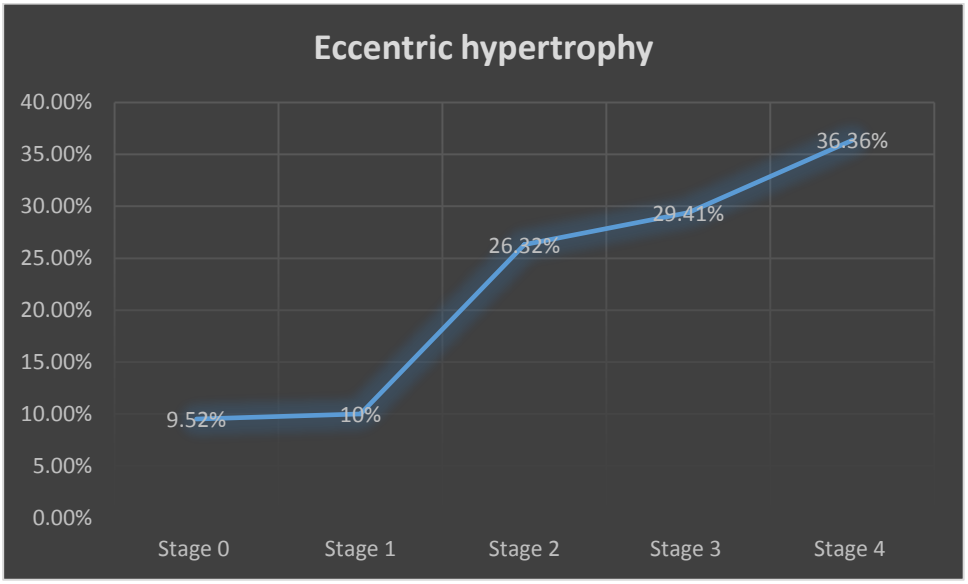

Fig 10: Correlation Between Hypertensive Retinopathy (HR) Stages and Echocardiographic Findings of Eccentric hypertrophy

Table 10: Comparison of Ejection fraction in Echocardiographic Findings between Hypertensive Retinopathy (HR) Stages.

Hypertensive Retinopathy Stages	Ejection fraction	
	Mean ± Sd	P-values
Stage 0	52.32 ± 4.62	
Stage 1	47.37 ± 5.61	0.01
Stage 2	42.82 ± 3.99	0.0001
Stage 3	36.71 ± 6.71	0.0001
Stage 4	28.42 ± 5.37	0.0001

Table10:Ejection fraction significantly decreased with retinopathy stage, reflecting worsening cardiac dysfunction: Stage 0: 52.32 ± 4.62, Stage 1: 47.37 ± 5.61 (p = 0.01), Stage 2: 42.82 ± 3.99 (p = 0.0001), Stage 3: 36.71 ± 6.71 (p = 0.0001) and Stage 4: 28.42 ± 5.37 (p = 0.0001). This progressive decline in EF suggests that as retinal damage worsens, the heart's functional capacity also deteriorates, further supporting the concept of parallel microvascular and myocardial changes in hypertensive individuals.

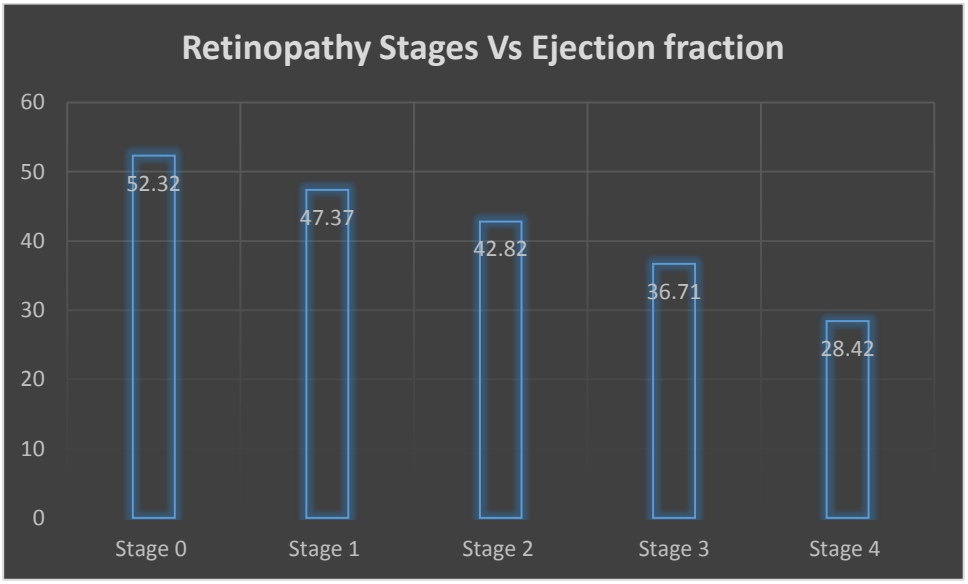

Fig 11: Comparison of Ejection fraction in Echocardiographic Findings between Hypertensive Retinopathy (HR) Stages

DISCUSSION

DISCUSSION

The study population showed a predominant age group of 31to 40 years (40.91%), followed by 41to 50 years (30.68%) and 51to 60 years (22.73%), with a smaller proportion of individuals in the 21 to 30 years range (5.68%). The findings highlight that hypertension and its complications, including hypertensive retinopathy and cardiac remodelling, are significant concerns even in relatively young adults. The high proportion of individuals in the 31–40 age group underscores the early onset of hypertension and its systemic effects, particularly in regions where hypertension management may be suboptimal. This demographic distribution suggests that early intervention is critical to prevent the progression of hypertensive retinopathy and associated cardiac changes, especially in younger adults who may not yet show overt signs of cardiovascular disease. Moreover, younger patients are often at risk of developing chronic hypertension, which can lead to long-term consequences like LVH and heart failure, even before symptoms become apparent.

Several studies suggest that cardiovascular remodelling and hypertensive retinopathy can begin to manifest in the 3rd to 5th decades of life, especially in untreated or poorly controlled hypertensive individuals. According to research by Keith[55] et al. and subsequent epidemiological studies, early signs of hypertensive organ damage (retinopathy, LVH) may occur in this age group due to early-onset or undiagnosed hypertension.

The study sample was predominantly male (57.95%), with females accounting for 42.05% of the population. This sex distribution is consistent with general trends observed in hypertensive populations, where males are often at a higher risk for developing hypertension and its complications at earlier ages compared to females. The higher percentage of males in this cohort may reflect the greater burden of early-onset hypertension in men.

While sex differences in cardiovascular health are well-documented, it's worth noting that hypertensive complications, including retinopathy and cardiac remodelling, affect both men and women, albeit at different rates and with varying clinical manifestations. Research suggests that men are more prone to developing LVH, a key marker of cardiac remodelling, at an earlier stage of hypertension compared to women. However, women may experience more significant cardiovascular risks post-menopause due to changes in hormonal regulation.

The male predominance in this study supports the importance of addressing hypertension management early, particularly in males, to reduce the risk of both retinal and cardiac complications. The differences observed between sexes may warrant further research to explore how sex hormones, lifestyle factors, and genetic predispositions contribute to the development of hypertensive retinopathy and associated cardiac remodeling.[46]

The distribution of hypertensive retinopathy stages in this study reflects the severity of retinal changes observed in individuals with systemic hypertension. The largest proportion of patients were classified in Stage 0 (23.86%), followed by Stage 1 (22.73%) and Stage 2 (21.59%). As the severity of hypertension increased, the prevalence of more advanced stages of retinopathy also rose, with Stage 3 and Stage 4 accounting for 19.32% and 12.5%, respectively.

This distribution suggests that early-stage hypertensive retinopathy (Stage 0 and Stage 1) is common among hypertensive patients, indicating that retinal changes can occur early in the disease process, even before more severe cardiovascular damage manifests. The declining percentage of Stage 4 cases could reflect the difficulty in achieving long-term hypertension control in those with advanced retinal changes, which often correlate with sustained or poorly controlled hypertension.

Association with Cardiac Remodelling:

The study also underscores the close relationship between hypertensive retinopathy severity and cardiac remodelling. As the stage of hypertensive retinopathy increases, there is a significant association with left ventricular hypertrophy (LVH), left ventricular mass index (LVMI), relative wall thickness (RWT), and ejection fraction (EF). This suggests that more severe retinal changes are not merely markers of eye health but also indicate underlying myocardial alterations, including structural changes in the heart that can contribute to heart failure and other cardiovascular diseases.[14]

The presence of hypertensive retinopathy, especially in the early stages (Stage 0 and Stage 1), could be used as a screening tool to identify hypertensive patients at higher risk for cardiac remodelling. Detecting these retinal changes early could prompt timely intervention to optimize blood pressure control, potentially preventing the progression to more severe stages of both retinopathy and cardiac complications. The progression from Stage 0 to Stage 4 represents a continuum of vascular damage that might parallel worsening cardiac dysfunction. In clinical practice, the presence of Stage 4 hypertensive retinopathy, with its high association with concentric remodelling and significant decline in ejection fraction, could be used as a marker for more aggressive management of hypertension and its complications.

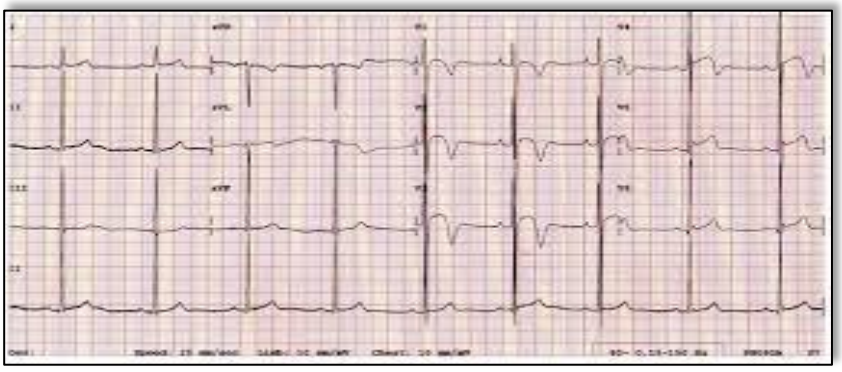

Figure12:ECG showing LVH

Blood Pressure Trends Across Hypertensive Retinopathy Stages:

The mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) consistently increased with the severity of hypertensive retinopathy stages. At Stage 0, the average SBP was 126.87 ± 11.54 mmHg and DBP was 81.62 ± 4.67 mmHg, which are consistent with borderline hypertensive values. As retinopathy progressed through Stage 1 to Stage 4, both SBP and DBP increased significantly, with the highest values observed in Stage 4 (SBP: 158.84 ± 15.63 mmHg, DBP: 98.34 ± 6.54 mmHg).

This increasing trend in blood pressure across retinopathy stages suggests a strong correlation between the degree of hypertension and the severity of retinal changes. Elevated SBP and DBP contribute to the development of hypertensive retinopathy by causing damage to the small retinal vasculature, which can progress with sustained or poorly controlled hypertension. This relationship reinforces the importance of early detection and aggressive blood pressure management to prevent both retinal damage and the associated cardiovascular risks.

Stage 0, with the lowest SBP and DBP, still demonstrates some retinal damage, implying that elevated blood pressure (even in the moderate range) may lead to early retinal changes. As the severity of hypertension increases in Stage 1 to Stage 4, both retinal damage and the risk for adverse cardiac remodelling, such as left ventricular hypertrophy (LVH) and decreased ejection fraction (EF), also become more pronounced.[47]

The progression of blood pressure levels directly correlates with worsening cardiac remodelling, particularly LVH and concentric remodelling. As hypertension becomes more severe, the heart's workload increases, leading to structural changes in the myocardium, such as increased left ventricular mass (LVM) and increased relative wall thickness (RWT). This

supports the notion that the degree of blood pressure elevation is a critical factor in determining both retinal and cardiac damage in hypertensive individuals. In particular, Stage 4 hypertensive retinopathy exhibited the highest SBP and DBP values, which were strongly associated with the most severe markers of cardiac remodelling. This highlights the importance of managing blood pressure not only to prevent retinopathy but also to minimize the risk of left ventricular dysfunction and heart failure.[48]

The data strongly suggests that blood pressure control plays a central role in preventing the progression of both hypertensive retinopathy and cardiac remodelling. For clinical practice, maintaining blood pressure below threshold levels is crucial to mitigate the detrimental effects on both the retina and heart. The association between elevated blood pressure and retinopathy severity further supports the utility of retinal examinations as part of a comprehensive cardiovascular risk assessment in hypertensive patients. Detecting early-stage retinopathy could prompt more rigorous blood pressure monitoring and treatment strategies aimed at preventing further organ damage.

The study reveals a clear association between hypertensive retinopathy stages and the presence of left ventricular hypertrophy (LVH), with the odds of LVH increasing dramatically as retinopathy severity worsens. In Stage 0, only 14.29% of patients had LVH, and this was used as the reference group. In Stage 1, the odds ratio for LVH was 2 (0.41–9.78), indicating a relatively low but notable increased risk compared to Stage 0. The odds ratio for LVH increases significantly in Stage 2 (3.5; 0.75–16.28), reflecting a more substantial risk. In Stage 3, 64.71% of patients had LVH, with an odds ratio of 11 (2.28–53.18), indicating a strong association between more advanced retinopathy and LVH. The highest risk for LVH was observed in Stage 4, where 81.82% of patients had LVH, and the odds ratio surged to 27 (3.8–191.67), showing a dramatic increase in risk with advanced hypertensive retinopathy.

The progression of LVH with increasing hypertensive retinopathy severity underscores the close relationship between vascular changes in the retina and structural changes in the heart. As the retina's small vessels become damaged by elevated blood pressure, the heart's left ventricle undergoes structural changes, such as hypertrophy, to compensate for the increased afterload caused by hypertension. Stage 3 and Stage 4 retinopathy, with their high odds of LVH, highlight a population at increased risk for more serious cardiovascular complications, including heart failure and arrhythmias. The presence of LVH is known to be a predictor of adverse cardiac outcomes, including systolic and diastolic dysfunction, and the results from this study suggest that hypertensive retinopathy may be an early indicator of such risks.

The association between LVH and hypertensive retinopathy may be explained by shared pathophysiological mechanisms, such as increased systemic vascular resistance and endothelial dysfunction. Both retinal vessels and the coronary microvasculature are vulnerable to the effects of sustained high blood pressure, leading to structural changes in the heart and retina over time. The worsening LVH in patients with more severe retinopathy could be indicative of progressive cardiovascular damage driven by uncontrolled or poorly controlled hypertension. The odds ratio increase in LVH across retinopathy stages emphasizes the need for close monitoring of cardiac function in patients with advanced retinopathy.[47]

The findings suggest that hypertensive retinopathy can be a useful tool for identifying individuals at high risk for LVH and subsequent cardiac remodelling. Clinicians should be aware of the potential for cardiac involvement in hypertensive patients with advanced retinopathy and consider early interventions such as stricter blood pressure control to prevent further cardiac remodelling and associated complications. As Stage 4 hypertensive retinopathy is associated with the highest risk for LVH, these patients should be prioritized

for more aggressive cardiovascular risk management to minimize the long-term impact on both their heart and retina.

The increasing LVMI with advancing hypertensive retinopathy stages strongly suggests that cardiac remodelling (specifically left ventricular hypertrophy) is closely linked to the degree of retinal damage caused by sustained hypertension. As the severity of retinopathy worsens, so does the left ventricular mass, indicating that the more extensive the vascular damage in the retina, the greater the structural changes in the heart. Stage 0, with the lowest LVMI, may still exhibit mild hypertensive changes but without significant cardiac hypertrophy. However, as the retinopathy progresses through Stages 1 to 4, the increasing LVMI indicates worsening left ventricular hypertrophy (LVH), which is a key marker of cardiac remodelling due to hypertension. This finding highlights the cumulative effect of chronic hypertension on both the retinal vasculature and cardiac muscle. The progressive increase in LVMI suggests that hypertensive patients with more severe retinopathy have a higher degree of cardiac involvement and are at greater risk for developing heart failure or arrhythmias in the long term.[14]

The progressive increase in LVMI is concerning because LVH is known to be a precursor to heart failure, particularly heart failure with preserved ejection fraction (HFpEF), which is common in hypertensive patients. Patients with Stage 4 hypertensive retinopathy, who have the highest LVMI, may require close monitoring for cardiac dysfunction and should be considered for aggressive blood pressure management to prevent further myocardial damage. Left ventricular hypertrophy is not only an indicator of heart damage but also a predictor of future cardiovascular events. The increasing LVMI across hypertensive retinopathy stages underscores the importance of early detection and intervention in hypertensive patients to prevent cardiac remodelling and improve long-term outcomes.

Blood Pressure Control and LVMI:

The data suggest that the severity of hypertension, as reflected by both increasing LVMI and retinopathy progression, is a critical factor in driving cardiac remodelling. Effective blood pressure control could prevent or slow the progression of both hypertensive retinopathy and left ventricular hypertrophy, improving overall cardiovascular health. Stage 4 patients, with the highest LVMI, may benefit from more intensive treatment strategies to control systolic and diastolic blood pressure, to reduce both retinal and cardiac damage. This highlights the importance of comprehensive cardiovascular care, including both ocular and cardiac assessments, in managing hypertensive patients.

The increase in LVMI with advancing hypertensive retinopathy stages emphasizes the interconnectedness of vascular damage across organs in hypertensive patients. Both retinal changes and cardiac remodelling reflect the systemic effects of uncontrolled hypertension, and understanding these associations can guide more effective monitoring and management of hypertensive individuals to prevent further cardiovascular complications[14].

Relative Wall Thickness (RWT) and Its Association with Retinopathy Stages:

The data demonstrates a significant increase in relative wall thickness (RWT) as the severity of hypertensive retinopathy progresses. The mean RWT values across the stages of hypertensive retinopathy are as follows: Stage 0: RWT = 0.35 ± 0.04, Stage 1: RWT = 0.41 ± 0.06, P = 0.07 (non-significant) Stage 2: RWT = 0.44 ± 0.05, P = 0.04 (significant), Stage 3: RWT = 0.47 ± 0.05, P = 0.001 (highly significant) and Stage 4: RWT = 0.52 ± 0.06, P = 0.0001 (highly significant).

Stage 0 shows the lowest RWT (0.35 ± 0.04), which is within normal limits. As the retinopathy progresses through Stage 1 to Stage 4, the RWT values increase significantly. The

P-value for Stage 1 (0.07) indicates that while there is a mild increase in RWT in Stage 1 compared to Stage 0, this difference is not statistically significant. However, from Stage 2 onward, there is a statistically significant increase in RWT, indicating that hypertensive retinopathy is increasingly associated with cardiac remodelling, specifically left ventricular hypertrophy and concentric remodelling.

Relative wall thickness (RWT) is an important marker of left ventricular remodelling, particularly for identifying concentric hypertrophy—a condition where the left ventricle's walls thicken in response to increased afterload caused by hypertension. The increasing RWT across retinopathy stages suggests that as the severity of retinal changes increases, the degree of cardiac remodelling also worsens. Stage 4 retinopathy, with the highest RWT value (0.52 ± 0.06), indicates the most severe form of concentric remodelling and is strongly associated with the most significant cardiac changes in patients with hypertension. These patients are at higher risk for adverse cardiovascular outcomes, including heart failure and arrhythmias.[49]

The progressive increase in RWT with worsening hypertensive retinopathy highlights the importance of early detection of hypertensive retinopathy as it could be an indicator of underlying cardiac dysfunction. This is particularly important in hypertensive patients, where cardiac remodelling may occur unnoticed unless routinely monitored. The significant rise in RWT from Stage 2 to Stage 4 suggests that once retinopathy reaches advanced stages, the risk for concentric hypertrophy and related cardiac issues becomes significantly higher. Clinicians should closely monitor cardiac function, particularly ventricular wall thickness and heart failure markers, in patients with advanced hypertensive retinopathy.[49]

Regular retinal examinations may serve as a non-invasive tool to assess the severity of hypertension-related vascular damage, potentially allowing for earlier intervention in patients at risk of cardiac remodelling. The association of increased RWT with worsening

hypertensive retinopathy underscores the importance of comprehensive management, including blood pressure control, to prevent cardiac complications. Given the strong association between RWT and cardiac outcomes, controlling blood pressure early in the course of hypertension could help prevent the development of concentric hypertrophy and left ventricular dysfunction. Early interventions can improve both retinal health and cardiac function.

The data underscores a significant association between relative wall thickness and the severity of hypertensive retinopathy, emphasizing the need for early cardiovascular risk stratification and management in hypertensive individuals. As RWT increases with advancing retinopathy stages, it signals worsening cardiac remodelling, which can eventually lead to heart failure and other complications. Clinicians should incorporate retinal assessment into the routine management of hypertensive patients to identify those at higher risk for cardiac remodelling and intervene promptly.[49]

Concentric Remodelling and Its Association with Retinopathy Stages:

The data clearly highlights a strong association between hypertensive retinopathy stages and the presence of concentric remodelling. As the severity of retinopathy progresses, the likelihood of developing concentric remodelling increases significantly, as indicated by the odds ratios and percentages across the stages of hypertensive retinopathy: Stage 0 (21 patients): Only 14.28% had concentric remodelling. This group serves as the reference with an odds ratio of 1. Stage 1 (20 patients): 20% of patients exhibited concentric remodelling, with an odds ratio of 1.5 (0.29–7.74), suggesting a slightly increased risk, though this was not statistically significant. Stage 2 (19 patients): 47.37% had concentric remodelling, with an odds ratio of 5.4 (1.18–24.65), indicating a moderate increase in risk. Stage 3 (17 patients): 76.47% showed concentric remodelling, with a very high odds ratio of 19.5 (3.71–102.38),

indicating a strong association between severe retinopathy and concentric remodelling. Stage 4 (11 patients): 90.91% had concentric remodelling, with an odds ratio of 60 (5.49–655.82), reflecting a dramatic increase in risk in patients with the most advanced hypertensive retinopathy.

Concentric remodelling refers to the thickening of the left ventricular walls in response to increased systemic vascular resistance, commonly seen in patients with chronic hypertension. This remodelling process is a key part of the adaptive response to pressure overload but can eventually lead to heart failure and other cardiovascular complications. The increase in concentric remodelling across the hypertensive retinopathy stages emphasizes that as vascular damage in the retina progresses, cardiac remodelling also worsens, highlighting the systemic effects of uncontrolled hypertension on both the heart and the retina. This finding suggests that patients with advanced hypertensive retinopathy may be at high risk for developing severe cardiac remodelling, leading to heart failure, arrhythmias, and other adverse outcomes.[49]

The association between concentric remodelling and hypertensive retinopathy can be explained by the common pathophysiological mechanisms underlying both conditions. Hypertension leads to increased afterload, which causes the left ventricle to undergo adaptive thickening of the walls. Similarly, the retina, being a microvascular structure, also suffers damage from chronic high blood pressure, resulting in retinal changes that correspond with the degree of vascular damage. In advanced stages of hypertensive retinopathy (Stages 3 and 4), the risk of severe concentric remodelling is markedly elevated, which is likely due to the chronicity and severity of the hypertension causing significant strain on both the cardiovascular system and the retinal vessels. The high odds ratio for concentric remodelling in Stage 3 (19.5) and Stage 4 (60) hypertensive retinopathy suggests that these patients

require close monitoring for cardiac involvement. As concentric remodelling is a marker of increased cardiovascular risk, identifying patients with severe retinopathy could help stratify risk and enable early intervention to prevent further cardiac deterioration. Blood pressure control is critical in preventing both retinal damage and cardiac remodelling. As the study indicates, patients with more severe retinopathy (especially Stage 3 and 4) are at much higher risk for concentric remodelling, necessitating more aggressive management of hypertension to minimize long-term cardiovascular complications.[49]

Eccentric Hypertrophy and Its Association with Retinopathy Stages:

The analysis reveals a moderate association between eccentric hypertrophy and hypertensive retinopathy stages, with a gradual increase in the prevalence of eccentric hypertrophy as the severity of retinopathy increases: Stage 0 (21 patients): Only 9.52% exhibited eccentric hypertrophy, which serves as the reference group. Stage 1 (20 patients): 10% of patients had eccentric hypertrophy, with an odds ratio of 1.06 (0.13–8.31), indicating a mild, non-significant increase in the risk. Stage 2 (19 patients): 26.32% of patients presented with eccentric hypertrophy, with an odds ratio of 3.39 (0.57–20.1), suggesting a moderate association, though the confidence interval is wide. Stage 3 (17 patients): 29.41% exhibited eccentric hypertrophy, with an odds ratio of 3.96 (0.66–23.76), indicating a stronger association, although the wide confidence interval indicates some uncertainty in the estimate. Stage 4 (11 patients): 36.36% had eccentric hypertrophy, with an odds ratio of 5.43 (0.81–36.51), suggesting an increased risk of developing eccentric hypertrophy in patients with advanced hypertensive retinopathy, though the wide confidence interval suggests inconsistent data.

Eccentric hypertrophy refers to the enlargement of the heart's chambers due to increased volume load, typically seen in conditions where there is chronic volume overload, such as in

valvular heart disease or left ventricular dilation. In the context of systemic hypertension, eccentric hypertrophy is less common than concentric hypertrophy, but it still represents an important aspect of cardiac remodelling. The slightly higher prevalence of eccentric hypertrophy in patients with higher-stage retinopathy (from Stage 2 onward) reflects a shift in cardiac remodelling as hypertension progresses. This suggests that hypertensive retinopathy not only signals vascular damage but may also contribute to an altered cardiac response—possibly due to changes in left ventricular compliance or increased filling pressures.[49]

The odds ratios indicate that as the severity of hypertensive retinopathy increases, the risk of eccentric hypertrophy rises, although the association is less pronounced compared to concentric remodelling. Eccentric hypertrophy is typically seen in conditions where there is volume overload, but in the case of hypertension, this finding might suggest diastolic dysfunction or left ventricular dilation as part of the overall cardiac remodelling process in response to prolonged high blood pressure.

While the association between eccentric hypertrophy and hypertensive retinopathy is less strong than that of concentric remodelling, the data highlights that eccentric hypertrophy becomes increasingly prevalent with worsening retinopathy. This suggests that the cardiac remodelling process in patients with advanced hypertensive retinopathy may include both concentric and eccentric changes, indicating volume overload and left ventricular dilation in addition to pressure overload. Further studies are needed to fully understand the underlying mechanisms and to determine the most effective strategies for managing cardiac remodelling in hypertensive patients with severe retinopathy.[49]

Ejection Fraction and Its Association with Retinopathy Stages:

The analysis shows a significant decline in ejection fraction (EF) as the severity of hypertensive retinopathy increases, with P-values indicating statistically significant

differences between the stages: Stage 0 (21 patients): The mean EF is 52.32 ± 4.62%, which is within the normal range for ejection fraction, indicating good left ventricular function in the absence of significant hypertensive retinopathy. Stage 1 (20 patients): The mean EF decreases to 47.37 ± 5.61%, with a P-value of 0.01, showing a significant reduction in EF compared to Stage 0, but still above the threshold for heart failure. Stage 2 (19 patients): The mean EF further declines to 42.82 ± 3.99%, with a P-value of 0.0001, suggesting that as retinopathy progresses, there is a more pronounced reduction in left ventricular systolic function. Stage 3 (17 patients): The mean EF decreases to 36.71 ± 6.71%, with a P-value of 0.0001, reflecting a significant deterioration in cardiac function, with borderline to moderate heart failure now observed. Stage 4 (11 patients): The mean EF drops to 28.42 ± 5.37%, with a P-value of 0.0001, indicating a severe reduction in left ventricular function, suggesting advanced heart failure in patients with the most severe hypertensive retinopathy.[14]

Ejection fraction is a critical marker of left ventricular function, and its decline is often associated with heart failure, whether systolic or diastolic. The study reveals that as hypertensive retinopathy progresses from Stage 0 to Stage 4, the ejection fraction decreases significantly, indicating worsening cardiac dysfunction. In Stage 0, the normal EF suggests that there is little to no cardiac impairment in patients with minimal retinopathy. However, as patients progress to Stage 1 and beyond, the gradual reduction in EF signals that hypertensive damage to the vasculature and myocardium is becoming clinically significant. This finding emphasizes the link between hypertensive vascular changes in the retina and cardiac dysfunction.

The decline in ejection fraction correlates with the degree of cardiac remodelling seen in hypertensive patients. As hypertension persists, the heart undergoes remodelling, both in terms of hypertrophy and dilation, which eventually compromises its ability to contract

effectively, leading to a decrease in cardiac output. Hypertensive retinopathy itself is a marker of systemic vascular damage, and the associated decline in EF suggests that patients with severe retinal changes have a higher likelihood of developing heart failure. The increase in vascular resistance (as seen in the retina and the heart) can lead to left ventricular hypertrophy, dilatation, and eventually systolic dysfunction. The significant drop in EF in Stage 3 and Stage 4 retinopathy (to 36.71% and 28.42%, respectively) signals advanced cardiac dysfunction. These patients are at a higher risk of heart failure and other cardiovascular complications. Early detection and management of hypertensive retinopathy could aid in risk stratification. Since ejection fraction is a key determinant in the management of heart failure, the reduction in EF across the stages of retinopathy calls for more aggressive management of hypertension and cardiac function. Treatment strategies could include early blood pressure control, anti-hypertensive therapy, and possibly heart failure management for patients with more advanced retinopathy. This study highlights a strong association between the severity of hypertensive retinopathy and left ventricular systolic dysfunction, as measured by ejection fraction. As retinopathy progresses from Stage 0 to Stage 4, there is a gradual and significant decrease in ejection fraction, suggesting that cardiac dysfunction is closely linked to the severity of hypertensive damage. These findings underscore the importance of comprehensive vascular and cardiac assessment in patients with hypertension and retinopathy, and the need for early intervention to prevent the progression to heart failure.

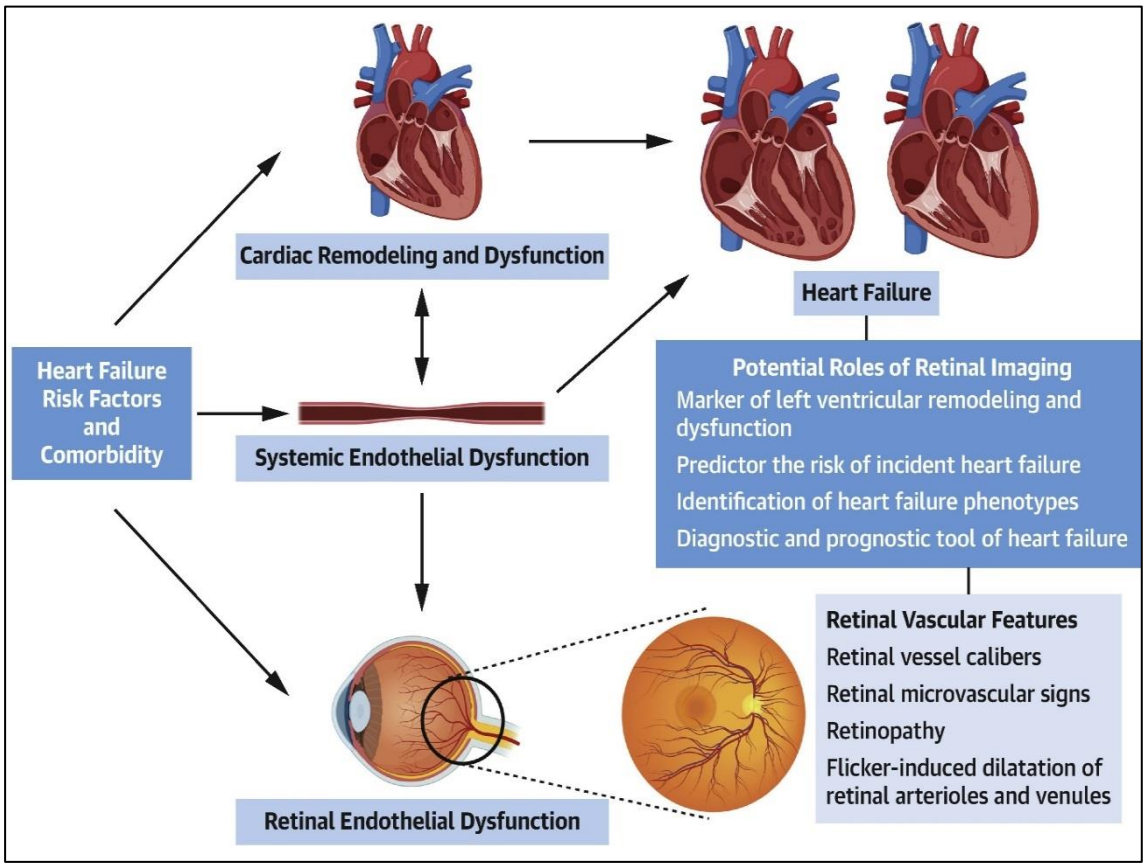

Figure 12 – Association of cardiac remodelling and retinal endothelial dysfunction

CONCLUSION

CONCLUSION

In conclusion, the association between cardiac remodelling and the severity of hypertensive retinopathy underscores the importance of comprehensive cardiovascular assessment in patients with systemic hypertension. While early stages of hypertensive retinopathy (grades 1 and 2) may present with subtle changes in the retinal vasculature, they are still linked to early signs of cardiac remodelling, such as left ventricular hypertrophy (LVH) and impaired myocardial function. These findings highlight the potential of retinal examination as a non-invasive tool for early detection of cardiac involvement in hypertensive patients. However, the presence of advanced hypertensive retinopathy (grades 3 and 4) is more strongly correlated with significant cardiac remodelling, including left atrial enlargement, reduced left ventricular ejection fraction, and increased incidence of heart failure. These associations suggest that hypertensive retinopathy, particularly in its advanced stages, may serve as an indicator of severe target organ damage and a predictor of adverse cardiovascular outcomes. Therefore, regular monitoring of retinal changes in hypertensive patients is recommended to facilitate early detection and timely intervention, potentially improving long-term cardiovascular health.

SUMMARY

SUMMARY

The study analyzed the relationship between hypertensive retinopathy and cardiovascular changes in 88 participants. Most subjects were aged 31–40 years (40.91%), followed by 41–50 years (30.68%), 51–60 years (22.73%), and 21–30 years (5.68%), with a slight male predominance (57.95%). Retinopathy was classified into five stages: 23.86% had Stage 0 (no changes), while the distribution in Stages 1 to 4 was 22.73%, 21.59%, 19.32%, and 12.5%, respectively. Blood pressure (both systolic and diastolic) showed a clear upward trend across retinopathy stages, indicating a direct relationship between BP elevation and retinal changes. Left ventricular hypertrophy (LVH) was increasingly prevalent with retinopathy severity, ranging from 14.29% in Stage 0 to 81.82% in Stage 4. The odds ratio for LVH was significantly higher in advanced stages, with Stage 4 showing an OR of 27 (95% CI: 3.8–191.67). Left ventricular mass index (LVMI) and relative wall thickness (RWT) also rose progressively with advancing retinopathy. Concentric remodelling became more common, from 14.28% in Stage 0 to 90.91% in Stage 4, while eccentric hypertrophy showed a gradual but less statistically robust increase. Ejection fraction declined significantly, from 52.32 ± 4.62 in Stage 0 to 28.42 ± 5.37 in Stage 4, reflecting worsening cardiac function. The findings demonstrate a strong link between hypertensive retinopathy severity and cardiac alterations, emphasizing the importance of routine retinal and cardiovascular assessments in hypertensive patients.

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ANNEXURE

ANNEXURE I

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) Grade I Grade II Grade III Grade IV		

Lab Investigations

RBS

Lipid profile

Blood urea

Serum Creatinine

Urine

. ECG

11. 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.

PATIENT INFORMATION SHEET

TITLE: "ASSOCIATION OF HYPERTENSIVE RETINOPATHY AND CARDIAC REMODELLING IN SYSTEMIC HYPERTENSION"

This information is to help you understand the purpose of the study titled "**A**ssociation of Hypertensive Retinopathy and Cardiac Remodeling in Systemic Hypertension". 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.

High blood pressure 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 hypertensive 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, retinal examination, Electrocardiography & 2D Echocardography.

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. ATHMIKA. R, MBBS

Junior Resident

Department of Ophthalmology,

SDUMC, Kolar – 563101

Contact no: 9080095834

Mail ID: athmikarj@gmail.com

ಶೀರ್ಷಿಕೆ: " ವ್ಯವಸ್ಥಿತ ಅಧಿಕ ರಕ್ತದೊತ್ತಡದಲ್ಲಿ ಹೈಪರ್ಟೆನ್ಸಿವ್ ರೆಟಿನೋಪತಿ ಮತ್ತು ಕಾರ್ಡಿಯಾಕ್ ರಿಮಾಡೆಲಿಂಗ್‌ನ ಅಸೋಸಿಯೇಷನ್ "

"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 ಆಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ ಅಂಡ್ ರಿಸರ್ಚ್, ತಮಕ, ಕೋಲಾರ - 563101 ನಲ್ಲಿ ಸಂಪರ್ಕಿಸಿ.

<u>ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ</u>

ಡಾ. **ಆತ್ಮಿಕಾ. ಆರ್**

<u>ಎಸ್ ಡಿ ಯು ಎಮ್ ಸಿ.</u>

ಟಮಕ, ಕೋಲಾರ

ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 9080095834

athmikarj@gmail.com

ANNEXURE III

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

INFORMED CONSENT FORM

Case no:

IP no:

TITLE: "ASSOCIATION OF HYPERTENSIVE RETINOPATHY AND CARDIAC REMODELLING IN SYSTEMIC HYPERTENSION"

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:			

<u>ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನಾ ಸಂಸ್ಥೆ, ಟಮಕ, ಕೋಲಾರ - 563101.</u>

<u>ತಿಳಿವಳಿಕೆ ಸಮ್ಮತಿ ನಮೂನೆ</u>

ಶೀರ್ಷಿಕೆ: " ವ್ಯವಸ್ಥಿತ ಅಧಿಕ ರಕ್ತದೊತ್ತಡದಲ್ಲಿ ಹೈಪರ್ಟೆನ್ಸಿವ್ ರೆಟಿನೋಪತಿ ಮತ್ತು ಕಾರ್ಡಿಯಾಕ್ ರಿಮಾಡೆಲಿಂಗ್ ನ ಅಸೋಸಿಯೇಷನ್ "

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:			

ANNEXURE IV

PHOTOGRAPHS

1. Slit lamp examination

2.Fundus examination by IDO

MASTER CHART

sl no	Age	Sex	Retinopathy Stages	SBP	DBP	ECG-LVH	2DECHO-LVMI	2DECHO-CON RE	2DECHO-ECC RE	EF%
1	41	M	3	142	95	N	119	Y	N	30
2	44	M	1	137	79	N	89	N	N	44
3	33	M	0	147	85	N	99	N	N	49
4	47	F	2	140	87	N	111	N	N	47
5	45	M	3	157	92	Y	94	Y	N	42
6	52	F	1	144	88	N	103	N	N	53
7	31	M	0	131	86	N	85	N	N	57
8	43	F	2	132	95	N	89	N	N	41
9	50	M	3	168	97	N	109	Y	N	38
10	53	F	1	124	80	N	81	N	N	47
11	35	M	4	154	92	Y	124	Y	N	31
12	47	F	2	148	92	Y	96	Y	N	44
13	34	F	0	138	78	N	76	Y	N	55
14	25	M	1	137	90	Y	94	Y	N	49
15	36	F	3	146	99	Y	104	Y	Y	33
16	46	M	3	160	86	Y	122	Y	N	40
17	45	F	1	141	84	N	94	N	N	51
18	54	M	2	154	96	N	102	N	N	42
19	34	F	0	153	92	N	91	N	N	52
20	42	M	4	168	100	Y	116	Y	N	27
21	35	F	0	148	75	Y	70	N	N	46
22	36	M	1	122	85	N	78	N	N	45
23	46	M	3	169	94	Y	115	N	N	35
24	55	M	2	143	88	N	87	Y	Y	40
25	32	M	0	116	82	N	82	Y	N	51
26	35	F	1	136	81	N	100	Y	N	48
27	52	M	3	159	101	N	104	Y	N	37
28	27	F	0	138	81	N	88	N	N	53
29	34	M	2	151	92	Y	100	Y	N	47
30	49	F	1	130	89	N	93	N	Y	42
31	52	M	4	162	98	Y	107	Y	Y	33
32	35	F	0	125	89	N	95	N	N	59
33	31	M	0	126	88	N	77	N	N	54
34	37	F	2	129	87	N	98	N	N	46
35	51	F	1	129	83	Y	105	N	N	46
36	36	M	0	132	82	N	84	N	N	48
37	34	F	3	155	92	Y	96	Y	Y	34
38	42	M	4	150	95	Y	97	Y	N	28
39	53	F	0	129	83	Y	92	Y	Y	50
40	36	M	1	142	78	N	106	Y	N	47
41	42	F	2	143	96	Y	93	Y	Y	44
42	34	M	0	144	77	N	79	N	N	52
43	57	F	3	151	91	N	124	N	N	33
44	36	M	1	127	84	N	96	N	N	51
45	28	M	0	136	72	N	86	N	N	56
46	36	M	3	148	95	Y	95	Y	N	37
47	58	M	2	138	94	N	103	N	N	41
48	46	M	1	133	87	N	91	N	N	52
49	34	M	4	147	101	N	129	Y	N	24
50	32	F	3	168	97	N	109	Y	Y	38
51	56	M	0	128	80	N	65	N	N	53
52	38	F	1	139	90	Y	87	N	N	43
53	35	M	2	137	95	N	110	Y	Y	42
54	42	F	0	132	82	N	96	N	N	47

55	57	M	4	163	104	Y	110	N	Y	29
56	36	F	1	129	81	N	90	N	N	52
57	45	M	0	131	87	N	87	N	N	45
58	35	F	3	154	90	Y	106	Y	N	41
59	54	F	1	134	91	N	93	N	N	44
60	34	M	0	144	87	Y	83	N	Y	50
61	47	F	2	151	99	Y	108	N	N	40
62	53	M	4	157	92	Y	105	Y	N	30
63	36	F	0	125	80	N	72	N	N	58
64	46	M	1	128	85	Y	92	Y	Y	45
65	24	F	3	163	95	N	118	N	N	35
66	37	M	2	143	91	N	91	Y	N	41
67	57	F	1	138	83	N	85	N	N	46
68	35	M	0	130	80	N	90	N	N	52
69	49	M	4	166	94	N	122	Y	Y	27
70	56	F	2	155	90	Y	95	N	Y	45
71	46	M	0	117	77	N	81	N	N	55
72	52	M	1	141	82	N	94	N	N	48
73	36	M	3	155	96	Y	103	Y	Y	34
74	54	F	0	97	75	N	78	N	N	51
75	46	M	4	162	99	Y	98	Y	N	32
76	47	M	1	128	81	Y	98	N	N	47
77	34	F	2	149	94	N	106	Y	N	43
78	55	M	3	170	99	Y	104	N	N	32
79	50	M	2	144	98	Y	104	N	N	43
80	47	F	1	146	85	N	77	N	N	42
81	26	F	4	160	97	Y	110	Y	N	29
82	36	M	2	153	91	N	87	N	N	44
83	51	F	3	138	91	Y	112	Y	N	43
84	43	M	2	131	87	Y	105	Y	N	41
85	42	M	4	153	104	Y	117	Y	Y	27
86	36	F	2	135	92	N	112	N	N	42
87	47	M	3	162	91	Y	110	Y	Y	35
88	35	M	2	150	93	N	100	Y	Y	46