"A CROSS SECTIONAL STUDY OF ASSOCIATION OF FUNDUS MANIFESTATIONS WITH THE SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS."

By

Dr. SHRAVAN MASURKAR

Dissertation submitted to the

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

In partial fulfillment of the requirements for the degree of

MASTERS OF SURGERY

In

OPHTHALMOLOGY

Under the guidance of

Dr. M.H.PATIL

M.S. (OPHTHALMOLOGY)

PROFESSOR OF OPHTHALMOLOGY

DEPARTMENT OF OPHTHALMOLOGY

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

AND RESEARCH CENTRE, BIJAPUR-586103

2015

B.L.D.E. UNIVERSITY'S

SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL &

RESEARCH CENTRE, BIJAPUR

DECLARATION BY THE CANDIDATE

I, **Dr. SHRAVAN MASURKAR,** hereby declare that this dissertation entitled **"A CROSS SECTIONAL STUDY OF ASSOCIATION OF FUNDUS MANIFESTATIONS WITH THE SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS."** is a bonafide and genuine research work carried out by me under the guidance of **Dr.M. H. Patil**, Professor, Department of Ophthalmology, B.L.D.E.U'sShri B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date:

Place: Bijapur

Dr. SHRAVAN MASURKAR
Post Graduate Student,
Department of Ophthalmology,
B.L.D.E.U's Shri B. M. Patil Medical College, Hospital & Research Centre,
Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL &

RESEARCH CENTRE, BIJAPUR

CERTIFICATE BY THE GUIDE

This to certify that the dissertation entitled **"A CROSS SECTIONAL STUDY OF ASSOCIATION OF FUNDUS MANIFESTATIONS WITH THE SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS."** is a bonafide research work done **Dr. SHRAVAN MASURKAR**, under my overall supervision and guidance, in partial fulfillment of the requirements for the degree of M. S. in Ophthalmology.

Date:

Place: Bijapur

Dr. M.H.PATIL

M.S. (OPHTHALMOLOGY)

Professor of Ophthalmology

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College, Hospital & Research Centre, Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL &

RESEARCH CENTRE, BIJAPUR

ENDORSEMENT BY THE HEAD OF DEPARTMENT

This to certify that the dissertation entitled **"A CROSS SECTIONAL STUDY OF ASSOCIATION OF FUNDUS MANIFESTATIONS WITH THE SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS."** is a bonafide research work done by **Dr. SHRAVAN MASURKAR.** under the guidance of **Dr. M.H.Patil**$_{M.S,}$ Professor, Department of Ophthalmology at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date:

Place: Bijapur

DR. VALLABHA.K$_{M.S,DOMS.}$
Professor and Head,
Department of Ophthalmology,
B.L.D.E.U's Shri B. M. Patil Medical College, Hospital & Research Centre,
Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL &

RESEARCH CENTRE, BIJAPUR

ENDORSEMENT BY THE PRINCIPAL

This to certify that the dissertation entitled **"A CROSS SECTIONAL STUDY OF ASSOCIATION OF FUNDUS MANIFESTATIONS WITH THE SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS"** is a bonafide research work done by **Dr. SHRAVAN MASURKAR.** under the guidance of **Dr. M. H. Patil**$_{M.S,.}$ Professor, Department of Ophthalmology at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date**:**

Place: Bijapur.

DR. M. S. BIRADAR **M.D.**
Principal,
B.L.D.E.U's Shri B. M. Patil Medical College, Hospital & Research Centre, Bijapur.

B.L.D.E. UNIVERSITY'S

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL

&RESEARCH CENTRE, BIJAPUR

COPYRIGHT

DECLARATION BY THE CANDIDATE

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

Date:

Place: Bijapur

Dr. SHRAVAN MASURKAR.

Post Graduate Student,

Department of Ophthalmology,

B.L.D.E.U's Shri B. M. Patil Medical College, Hospital & Research Centre, Bijapur.

ACKNOWLEDGEMENT

This piece of work has been accomplished with the grace of almighty . It gives me immense pleasure to express my heartfelt gratitude to my grandparents and parents. I dedicate this page to each and everyone who have helped me to explore the expanses of knowledge.

I express my profound gratitude and sincere thanks to my guide, ***Dr.M.H.Patil****M.S,Professor, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, for his constant and unfailing support, professional insight, valuable suggestions, motivation and exemplary guidance to carry out and complete this dissertation. I am deeply grateful to him for providing me necessary facilities and excellent supervision to complete this work.*

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

*My thanks to our Medical Superintentend****Dr.Shivakumar.Hiremath*** *for this support and inspiration.*

I am deeply indebted and grateful to my professors, ***Dr.VallabhaK*** *M.S.,DOMSand* ***Dr. Sunil. G. Biradar****M.S., Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, who with their valuable suggestions and constant guidance supported me throughout the preparation of this dissertation work.*

*My thanks to****Dr.Shadakshari.S.Math****M.S., AssociateProfessor and* ***Dr.Raghavendra.Ijeri****M.S., Assistant professor, Department of Ophthalmology, B.L.D.E.U's Shri B. M. Patil Medical College, Bijapur, for their valuable suggestions and encouragement which have definitely helped me improve my research work.*

I sincerely acknowledge, ***Dr.Ravi.Jadhav, Dr.Renuka. Kattimani, Dr.Varunkumar****,* ***Dr.Akshatha, Dr.Sushma, Dr.Radhika,*** *for their help and encouragement which has definitely helped to improve my research work.*

It is with great pleasure I express my sincere gratitude to my parents, uncle (Kishor R.Masurkar) and relatives for their constant encouragement, inspiration and sacrifices.

I also thank all my Post-graduate colleagues ***Dr. Darshan ,Dr.Shilpa*** *and friendsin the department for their co-operation during the preparation of this dissertation.*

I also thank all my Post-graduate junior colleagues ***Dr. Harsha, Dr. Neeta, Dr. Gautham*** *and friends in the department for their co-operation during the preparation of this dissertation.*

I thank ***Mrs. Vijaya Soraganvi****, Statistician for her masterly guidance and statistical analysis. I sincerely acknowledge the support and kindness shown towards me by all the staff of Central Library, Shri B. M. Patil Medical College, Bijapur, at all times.*

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

I am grateful to ***PREETI INTERNET ZONE****, Bijapur for their timely and fantastic printing work.*

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

Date- ***Dr. SHRAVANMASURAKAR***

Place:

LIST OF ABBREVIATIONS

χ^2	Chi square
AION	Anterior ischemic optic neuropathy
BRVO	Branch retinal vein occlusion
CRAO	Central retinal artery occlusion
CRVO	Central retinal vein occlusion
FBS	Fasting blood sugar
HDL	High density lipoprotein
IHD	Ischemic heart disease
LDL	Low density lipoprotein
TIA	Transient Ischemic Attack
VLDL	Very low density lipoprotein

LIST OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

ABSTRACT

BACKGROUND AND OBJECTIVES

Dyslipidemia in hypertensive patients may act as a predisposing risk factor, an aggravating or complicating factor.This study aims to assess the association between concentrations of various serum lipids, lipoproteins and retinal changes in patients with hypertension.

METHODS

The cross sectional study was conducted at B.L.D.E.U'S ShriB.M.Patil Medical College, Hospital and Research centre, Bijapurfrom 1st October 2012- 31st March 2014.A total of 245 patients were included in the study who were diagnosed to have primary essential hypertension. All patients were evaluated in detail regarding hypertensive status and detailed ophthalmic evaluation was done.

Patients were divided in to retinopathy group and no retinopathy group initially. Further retinopathy patients were divided depending on the severity of the retinopathy in to GRADE I, II, III & IV.

RESULTS AND OBSERVATIONS

Off the 245 patients, 106patients had normal fundus and 139 patients had retinopathy. Among these patients 91 were males and 48 were females. With M:F=1.9:1 with the age range of 25-85 years (Mean age of 57.16 years). And average duration of hypertension of 7.55.

The correlation of severity of hypertension with retinopathy was statistically significant ($p<0.0001$) and the relationship between retinopathy and duration was also statistically significant ($p<0.0001$).

The increased level of total cholesterol correlated well with increasing severity of retinopathy. LDL cholesterol and Triglyceride levels correlated positively with

increasing severity of retinopathy which was statistically significant.($p<0.0001$)

CONCLUSION

Our study demonstrated an association between serum lipid parameters and the prevalence of hypertensive retinopathy. Dyslipidemia is already considered as important risk factor for end organ failureand it can be considered as risk factor for severity of retinopathy. Preservation of vision may be an additional motivating factor for lowering serum lipid levels in persons with hypertension in whom they are elevated.

Key words: Dyslipidemia; Essential hypertension, Hypertensive Retinopathy

INTRODUCTION

Systemic hypertension (140/90 mm hg and above) is present in 25% urban (34 million) and 10% rural (31.5 million) subjects in India.[1, 2]

High blood pressure is related with many clinical complications such as stroke, cardiac failure, myocardial infarction, renal failure, peripheral vascular diseases.[3]Retinopathy is considered one of the indicators of target organ damage.

Hypertensive retinopathy is among the vascular complications of essential hypertension. It is known that the auto regulation of the retinal circulation fails as bloodpressure increases beyond a critical limit.

However, elevated blood pressure alone doesnot fully account for the extent of retinopathy. There are cases in which retinopathy wasresolved despite the persistence of high blood pressure. In addition to the effect of highblood pressure, other factors and humoral components probably take part in thepathogenesis of hypertensive retinopathy.[4]

Hyperlipidemia contributes to the process of arteriosclerosis, which develops even faster in the presence of hypertension or diabetes mellitus. Hyperlipidemia or dyslipidemia is known to be an important risk factor inhypertensive patients.[5,6,7]

Although atherosclerotic changes were described in retinal arteries half a centuryago (then called arteriosclerotic or arteriolosclerotic), there are no data on lipoproteinsand retinal artery atherosclerosis.[8]

Dyslipidemiais known to be a risk factor for retinopathy and other ocularabnormalities. When it is associated with diseases like diabetes, hypertension theoutcome is complicated. Its role in association with diabetic retinopathy and age relatedmaculopathy is well proven.[9,10]

Dyslipidemia in hypertensive patients may act as a predisposing risk factor, anaggravating or complicating factor.[7]

An understanding of various ocular manifestations,spectrum of findings and their association with components of lipid profile (LDL, HDL,VLDL, Total Cholesterol, and Triglycerides) may be helpful in risk stratification and intailoring of anti-hypertensive and lipid lowering treatment.

Ophthalmoscopic findings are helpful in evaluating the duration, severity, predictions or hypertension vasculopathy effects.[3]

Hence this study aims to assess the association between concentrations of various serum lipids, lipoproteins and retinal changes in patients with hypertension.

AIM AND OBJECTIVE OF THE STUDY

1. To evaluate fundus findings, and to correlate findings with the components of lipid profile (LDL, HDL, VLDL total cholesterol and triglycerides) in hypertensive patients.

REVIEW OF LITERATURE

Hypertension may be defined as “the level of which the benefits (minus the risks and costs) of action exceeds the risks and costs (minus the benefits) of inaction.[11]

As per the JNC VII report on prevention detection, evaluation and treatment of high B.P.[12x]

Category	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Normal	< 120	< 80
Pre hypertension	120-139	80-89
Stage 1	140-159	90-99
Stage 2	> 160	>100

Optimal BP with respect to cardiovascular risk is < 120 mm Hg systolic and < 80 mm Hg diastolic. Based on the average of two or more readings taken at each of two or more visits after an initial screening.

Essential hypertension [primary hypertension]

Essential hypertension is the name given to a group of persons whose arterial pressures are raised and in whom no specific disease can be found to account for the raised pressure. It is thus diagnosed by exclusion. Its clinical manifestations represent the consequences of raised arterial pressure on the cardiovascular system. The end stages of essential hypertension are a comparatively uniform malignant course dominated by Fibrinoid necrosis of arterioles, and much more benign course in which the other vascular lesions play a dominant role.

Clinical features of essential hypertension[11]

Age of onset

Since vascular disease is a consequence of advancing age, most patients with essential hypertension presenting with symptoms of benign phase belong to later agegroups.

Symptoms

The majority of patients with hypertension has no specific symptoms referable to their blood pressure elevation and will be identified only in the course of physical examination

Headache: is characteristic only of severe hypertension commonly localized to occipital region. Usually present when patient awakens in morning subsides spontaneously after several hours.

Other possible related complaints include

- Dizziness
- Palpitation
- Easy fatigability
- Impotence

Complaints referable to vascular disease include:

- Epistaxis
- Hematuria
- Blurring of vision due to retinal changes
- Dizziness due to TIA
- Angina and dyspnoea due to cardiac failure.

Clinical evaluation of essential hypertension

A strong family history of hypertension along with the intermittent finding of elevated pressure in the part favours the diagnosis of hypertension. Exclude the cause of secondary hypertension.

Elicit risk factors: Smoking, Diabetes, Renal disorders. Assess patients' life style: Diet, Physical activity, family status, and work.

Physical examination

Starts with general appearance: Round face and truncal obesity – Cushing syndrome.Is muscular development of upper extremities out of proportion to lower limit - Coarctation of Aorta.Compare the BP and pulse in both extremities and in supine and standing Measure patients height and weight. Fundus examination because this provides clues to the duration and prognosis of hypertension.Palpation and auscultation of carotid arteries for evidence of stenosis or occlusion.

Examination of cardia

For evidence of left ventricular hypertrophy and cardiac decompensation

For left ventricular lift

For 3rd and 4th heart sounds

Examination of lungs

For pulmonary rales

Extra cardiac murmurs and palpable collateral vessels as seen in Coarctation of aorta

Examination of abdomen

Auscultation for bruits originating in stenotic renal arteries. These have diastolic components, best heard just right or left of midline, above umbilicus Palpation for aneurysms and enlarged kidneys.

Examination of extremities for edema

Search for evidence of previous CVA

Laboratory investigations[13]

These are divided into those, which should be performed in all patients, with sustained hypertension (Basic studies) and those, which should be added if From the initial evaluation a secondary form of hypertension is suggested Arterial pressure is not controlled after initial therapy / (secondary studies)

Basic studies

a. **Always included**
    - Urine for protein, sugar, and blood, Microscopy.
    - Hematocrit
    - Serum creatinine, blood urea, serum electrolytes
    - Fasting blood glucose
    - Total cholesterol
    - ECG

b. Usually included
    - TSH
    - WBC counts
    - Fasting lipid profile
    - Serum uric acid
    - Chest X-ray
    - Echocardiogram

Special studies to screen for secondary hypertension:

Renovascular disease: ACE inhibitor radio nucleotide renal scan

Renal duplex Doppler flow studies

MR angiographies

Pheochromocytoma: 24 hour's urine for creatinine, metanephrines and catacholamies. Cushing's syndrome: Overnight dexamethasone suppression felt or 24 hours urine cortisol and creatinine Primary aldosteronism Plasma aldosterone and rennin activity ratio.

Complications of untreated hypertension [End organ damage in hypertension]

The adverse effects of hypertension principally involve the CNS, the retina, the heart, and kidneys.

CNS

Stroke

When this results from cerebral hemorrhage and cerebral ischaemia it is a common complication of hypertension and a major cause of death. Cerebral ischaemia is secondary to atherosclerosis. Cerebral hemorrhageis the result of both elevated arterial pressure and development of micro aneurysms called Charcot – Boucher aneurysm.

Hypertensive encepahlopathy[13]

This is a rare condition characterized by very high blood pressure andneurological symptoms including transient disturbances of speech, or vision, paraesthesia, disorientation, seizures and loss of consciousness. Papilloedema is common .Neurological deficit is reversible if hypertension is treated.

Heart

Most deaths due to hypertension results from, myocardial infarction / congestive heart failure.In short raised pressure lead to[14].Myocyte hypertrophy, interstitial changes and fibrosisReduction in flow in intracardiac vessels and endothelial dysfunction – smallvessel disease.Epicardial (large vessel) coronary disease Increased peripheral resistance and loss of compliance in arteries.

Kidney[13]

Atherosclerotic lesions of the afferent and efferent arterioles and the glomerular capillary tufts are the most common renal vascular lesions in hypertension and results in a decreased glomerular filtration rate and tubular dysfunction. Proteinuria and microscopic hematuria occur because of glomerular lesions and approximately 10% of the deaths caused by hypertension results from renal failure. Blood loss in hypertension occurs not only from renal lesions. Epistaxis hemoptysis and metrorrhagia also occur frequently in these patients.

HYPERTENSIVE RETINOPATHY

Hypertensive retinopathy is a condition characterized by a spectrum of retinal vascular signs in people with elevated blood pressure.[15]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.[16]

This clinical practice is supported byboth previous[17]and current[18]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 thebasis of the JNC criteria, the presence of retinopathy may be an indication for initiatingantihypertensive treatment, even in people with stage 1

hypertension (blood pressure, 140to 159/90 to 99 mm Hg) who have no other evidence of target-organ damage.

Pathophysiology

The retinal circulation undergoes a series of pathophysiological changes in response to elevated blood pressure.[19]In the initial, vasoconstrictive stage, there isvasospasm and an increase in retinal arteriolar tone owing to local auto regulatory mechanisms. This stage is seen clinically as a generalized narrowing of the retinalarterioles. Persistently elevated blood pressure leads to intimal thickening, hyperplasia ofthe media wall, and hyaline degeneration in the subsequent, sclerotic, stage. This stagecorresponds to more severe generalized and focal areas of arteriolar narrowing, changesin the arteriolar and venular junctions (i.e., arteriovenous nicking or nipping), andalterations in the arteriolar light reflex (i.e., widening and accentuation of the central lightreflex, or "copper wiring").

This is followed by an exudative stage, in which there is disruption of the blood–retina barrier, necrosis of the smooth muscles and endothelial cells, exudation of bloodand lipids, and retinal ischemia. These changes are manifested in the retina asmicroaneurysms, hemorrhages, hard exudates, and cotton-wool spots. Swelling of theoptic disk may occur at this time and usually indicates severely elevated blood pressure(i.e., malignant hypertension). Because better methods for the control of blood pressureare now available in the general population, malignant hypertension is rarely seen.

In contrast, other retinal vascular complications of hypertension, such as macro aneurysmsand branch-vein occlusions, are not uncommon in patients with chronically elevatedblood pressure. The stages of hypertensive retinopathy described, however, may not besequential. For example, signs of retinopathies that reflect the

exudative stage, such asretinal hemorrhage or microaneurysms, may be seen in eyes that do not have features ofthe sclerotic stage (e.g., arteriovenous nicking).The exudative signs are nonspecific,since they are seen in diabetes and other conditions.

CLINICAL ASPECTS

Retinal vascular response and other retinal changes seen in hypertension are variable and depend on several factors. The most important factors are the rate anddegree of hypertension and the baseline condition of the retinal vasculature.Concomitant diseases, such as diabetes or renal or connective tissue disordersalso play a role in the severity of findings.

- Clinical findings seen in the retinal vasculature in hypertensive retinopathy
- include the following:[20]
- Arteriolar narrowing (focal and diffuse)
- Arteriovenous crossing changes
- Arterial tortuosity
- Arterial sclerosis
- Increased branching angles

One of the earliest and classic signs of hypertensive retinopathy is arteriolar narrowing. An increase in vascular wall tone initiated acutely by autoregulatorymechanisms causes a decrease in caliber of the vessel, in what has been described as the vasoconstrictive phase.[19]A more focal or diffuse vasoconstrictive state may be observed,depending on the initial condition of the retinal vasculature . Vessels with areas ofsclerosis lack muscle tone and tend to dilate secondary to elevated intraluminal pressure.

Nonsclerotic vessels exhibit narrowing because of intact muscular walls and preservedautoregulatory response. Hayreh and colleagues,[21]in comparing prehypertensivefluorescein fundus angiograms to those with hypertensive retinopathy, found nonarrowing of arterioles. They concluded that the apparent narrowing was caused by an ophthalmoscopicartifact produced by retinal edema masking the arteriole walls from thesides along with the contrast of dilated venules, which made the arterioles appear more tapered.

Clinical findings in hypertensive retinopathy are:[22]

- Hemorrhages
- Retinal and macular edema
- Edema residues (hard exudates)
- Inner retinal ischemic spots (cotton-wool spots)
- Nerve fiber layer loss
- Focal intraretinalperiarteriolar transudates (FIPTs)

Hemorrhages

One common feature is hemorrhage. Hemorrhages within the more superficiallayers of the inner retina have a flame-shaped appearance because they track along axonsin the nerve fiber layer. Deeper hemorrhages in the retina have a dot or blot appearance,which varies with the configuration of the neural elements of layer to which they areconfined.

Hayreh and associates[20]concluded that retinal hemorrhages were only a minorfeature of hypertensive retinopathy. They found that nerve fiber layer hemorrhages in thedistribution of the radial peripapillary capillaries were more common than dot or blothemorrhages or subhyaloidhemorrhages. This probably reflects the arteriolar character ofthe radial peripapillary capillary bed. Hard exudates,

cotton-wool spots, and retinaledema are additional manifestations of the exudate phase of hypertensive retinopathy andindicate a more serious stage of the disease.

Hard exudates

Hard exudates or, more appropriately, edema residues are formed from extravasated plasma during the exudative phase. The residues are composed of lipids andcholesterol, giving them a characteristic waxy yellow or glistening appearance, and seemto settle in a bathtub ring-like configuration. They generally are found in the posteriorpole and assume patterns that reflect the source of the leakage (i.e., circinate rings) andthe neural elements of the layer in which they are found (i.e., macular star).[20]

Retinal and macular edema

The development of clinical edema of the retina and macula may stem from avariety of events at a cellular level and may have multiple causes. Loss of autoregulation, as in episodes of acute hypertension, may result in an increase in transmuralpressure in the capillaries leading to transudation of plasma into surrounding retinaproducingextracellular edema. Intracellular edema is the direct result of retinal ischemia.Breakdown of the retinal pigment epithelium (RPE) blood–retinal barrier secondary tohypertensive choroidopathy produces serous retinal detachments overlying regions of

choroidal ischemia. Diffusion of subretinal fluid into the retina eventually may createtissue edema.

Cotton-wool spots

Cotton-wool spots, or so-called soft exudates, are areas of acute inner retinal ischemia caused by occlusion of terminal arterioles. They have a fluffy white appearance,irregular borders, and most commonly are found in the posterior pole and

along thedistribution of the radial peripapillary capillary bed. They are localized within the nervefiber layer, often involving the underlying ganglion cells and inner nuclear while sparing the deeper retinal layers. Fluorescein angiography demonstrates nonperfusion of thecotton-wool spots and adjacent capillaries. Typically, they resolve ophthalmoscopically in 4 to 6 weeks, leaving a corresponding nerve fiber layer defect.

Focal intraretinalperiarteriolar transudates (FIPTs)

Cases of accelerated hypertension often are manifest by FIPTs. These appear asdull-white round, focal areas surrounding arterioles. The proposed mechanism of FIPTformation is the focal breakdown of the blood–retinal barrier, with the accumulation ofplasmatic deposits in the retina following an accelerated hypertension event.[22]

ARTERIOSCLEROTIC CHANGES

Sclerotic changes are observed within the vessel walls as constriction of the retinal vasculature persists. The arteriole wall normally is invisible, appearing only as anerythrocyte column with central light reflex by ophthalmoscopy. As the wall thickensfrom continuous vasoconstriction, the light reflex becomes more diffuse and partially

obscures the blood column, giving the once transparent arteriole a yellowed or copperwire appearance. Progression of the thickening and sclerotic changes eventually obscuresthe blood column completely, producing a silver wire appearance. Along with reflectivechanges, the thickening produces arteriovenous crossing changes. Retinal arterioles andvenules share a common adventitial sheath at their crossing points. As the arteriolar wallthickens, the venule appears tapered if posterior to the arteriole or elevated if over thearteriole.Other characteristic changes common but not unique to arteriosclerosis areincreased tortuosity secondary to fibrous

replacement of the vessel wall and increasedarteriolar branching angles, known as perpendicularization. Progression ofarteriosclerosis leads to endothelial damage and necrosis of the muscular component ofthe vessel walls.

This leads to a breakdown of the blood–retinal barrier, causing exudative leakage into the retina. The formation of fibrin and thrombosis within thevessel may cause closure of the lumen, resulting in ischemic changes within the retina.This has been described as the exudative phase. Concomitant choroidopathy maycontribute to further retinal changes seen in this phase of hypertensive retinopathy.[20]

- Complications related to hypertensive retinopathy include the following:
- Central or branch artery occlusion
- Central or branch vein occlusion
- Macroaneurysms
- Epiretinal membrane
- Neovascularization
- Vitreous hemorrhage
- Cystoid macular edema

Artery and vein occlusions and macroaneurysms are the most common results ofarteriosclerotic changes found in this condition. The development of neovascularizationwith or without vitreous hemorrhageis the result of subsequent retinal ischemia. Cystoidmacular edema also may result from ischemia, as well as from the breakdown of theretinal vascular and RPE blood barrier.

Epiretinal membranes may form in the cicatricialphase of any of these events. Consequent development of a full macular hole after severe hypertensive retinopathy with macular edema and vitreous hemorrhage also has been described. Other recent findings have shown abnormal electroretinogram and visuallyevoked potential studies 2 to 4 years after an accelerated hypertensive event likely relatedto retinal infarction and ischemic optic neuropathy, respectively.[20]

The overall rates of hypertensive retinopathy in the nondiabetic population rangesfrom 0.8% to 7.8%.[23]The study of populations is difficult and highly variable because ofdifferent evaluation methods, grading classifications of retinopathy, selection bias groups,and the association of other systemic diseases.

HYPERTENSIVE CHOROIDOPATHY

The choroid is very sensitive to blood pressure changes that only indirectly affectthe overlying RPE and neurosensory retina. Its pathophysiologic response to arterialblood pressure changes also is very different than that of the retinal vasculature. Thechoroid receives sympathetic innervation and is sensitive to circulating vasoconstrictivefactors such as angiotensin II, adrenaline, and vasopressin. These factors and neuralstimulation can initiate vasoconstriction of the choroid and choriocapillaris, leading tofocal ischemia. The overlying RPE and the outer blood–retinal barrier may be compromised as a result.

The clinical features of hypertensive choroidopathy include the following:[20]

- Elschnig's spots and Siegrist's spots (RPE and choroidal infarcts)
- Subretinal exudates
- Serous retinal detachments
- RPE depigmentation (chronic)
- Choroidal sclerosis

Clinically, direct changes to the choroidal vasculature are difficult to detect byophthalmoscopy. Many findings seen as retinal changes are a result of choroidalvasculature response to blood pressure change. Elschnig's spots are ischemic infarcts ofthe RPE that coincide to hypoperfusion of the underlying choroid . They appear as focalsubretinal lesions with yellowish halo. In an experimental model, Elschnig's spotsappeared within 24 hours of accelerated hypertension. Ischemic infarcts at the equatorhave a more linear appearance and are referred to as Siegrist's streaks.

The presence of Siegrist's streaks may indicate a more advanced vascular sclerosis. As ischemic RPEbecomes edematous, the blood–retinal barrier becomes disrupted, allowing leakage offluid from the choroid into the subretinal space and forming serous detachments.Resolution of such detachments can follow rapidly the restoration of blood pressurecontrol. Over time, the Elschnig's spots and areas of serous detachment develop centralareas of pigmentation with surrounding atrophy. Sclerotic choroidal vessels becomevisible through areas of atrophic RPE.

In malignant hypertension, one of the earliest changes seen is optic nerve head edema. Clinical Findings in Neuropathy

- Optic disc edema
- Optic disc pallor
- Optic disc ischemia

The clinical appearance is indistinguishable from other causes of optic nerve head swelling, such as elevated intracranial pressure. The mechanism of disc swelling remains controversial. Kishi and associates[23] demonstrated axonal hydropic swelling that wassecondary to ischemia. They later concluded that the ischemia produced was related to vasoconstriction of the peripapillarychoroidal and optic nerve head vessels.

The direct vasoconstrictive and occlusive properties seen in hypertensive choroidopathy lead to ischemic changes in the optic nerve head because it receives most of its blood supply from the peripapillarychoroidal vessels. Further, vascular endothelial substances can diffuse easily into the optic nerve head from the surrounding choroidal bed and cause vasoconstriction of the optic nerve head vessels.[23,24]

CLASSIFICATION OF HYPERTENSIVE RETINOPATHY[20]

Since Marcus Gunn's description in 1898 of the changes in retinal vessels in patients with arterial hypertension, various classification systems have been attempted toexplain observed changes and correlate them with the systemic disease. The first majorclassification scheme, by Keith and colleagues, was designed to relate survival to retinalvascular changes in the hypertensive population. Patients are classified according to theseverity of their fundus changes into four groups and morbidity is looked at over a 5-year period. They found that changes correlated directly with the degree of systemichypertension and, inversely, with the prognosis for survival. Later, Wagener andcoworkers developed more quantitative criteria for classifying hypertensive retinalvascular changes.

This system was based on the narrowing of arterioles and graded focalarterial constriction. In addition, they grouped vascular hypertension with associatedretinal changes within each group. Although more precise in design, reproducibility ofthe system was poor, and it was soon replaced by a more complete classification schemedescribed by Harold Scheie in 1953. He graded changes of hypertension and arteriolarsclerosis separately in five stages. He defined hypertensive changes as those related toarteriolar constriction and vascular changes from long-standing hypertension as arteriolarsclerosis.

Keith-Wagener-Barker Classification[15]

Group	Description
1	Mild to moderate narrowing and sclerosis of the arteries
2	Moderate to marked sclerosis of the retinal arterioles; exaggeration of the light reflex; arteriovenous compression changes or generalized or localized narrowing of the arterioles
3	Retinal arteriolar narrowing and focal constriction; retinal edema; cotton-wool spots; hemorrhage
4	Group 3 plus papilledema

Wagener-Clay-Gipner Modification of Generalized Arteriolar Narrowing[15]

Group	Description
1.	Reduction of caliber of arterioles to three fourths of average caliber
2.	Reduction of caliber of arterioles to half of average caliber
3.	Reduction of caliber of arterioles to one third of average caliber
4.	Arterioles thread-like or invisible

Scheie Classification[15]

Group	Hypertension	Arteriolar Sclerosis
0	No change	
1	Barely detectable arteriolar narrowing	Barely detectable light reflex changes
2	Obvious arteriolar narrowing with focal irregularities	Obvious increased light reflex changes
3	Grade 2 plus retinal hemorrhages or exudates	Copper wire arterioles
4	Grade 3 plus papilledema	Silver wire arteriole.

One of the problems that complicates all classification systems is the ophthalmoscopic variation in the extent of the acute hypertensive changes and those ofthe duration-related sclerotic changes observed in the same patient. In 1957, Leishmannpresented a seven-part classification that took into account the development of arteriolarsclerosis as part of the natural aging process and emphasized the modified ability ofretinal arterioles with involutional sclerosis to respond to hypertension. Subsequent to these discussions, clinicians have become sensitive to the need to account for thepresence or absence of arteriolar sclerosis in interpreting the fundi of hypertensivepatients. In 1966, the original study by Keith and associates was updated and modified toinclude the grading of generalized and focal arteriolar narrowing and arteriolar sclerosis.This modified system served as a helpful prognosticator of hypertensive disease.Arteriolar narrowing and focal constrictions in the absence of retinal hemorrhages or discedema seem to be the most sensitive indicators employed by the system. It currentlyremains the most commonly used classification for grouping hypertensive patientsophthalmoscopically[9].

Blood Pressure

In many studies it has been proved that there is a strong association between presence of hypertensive retinopathy and elevatedblood pressure. [25-29]

In elevated hypertension there is occurrence of specific retinal signs which is confirmed in few studies.[30,31]Other signs of retinopathy like focal arteriolar narrowing, retinal hemorhagesmicroaneurysms and cotton-wool spots were related to current blood pressure levels.[30,31]Hyperglycemia, inflammation and endothelial dysfunction may also be involved in pathogenesis of retinopathy.[32]

The Risk of Stroke

In a Population-based study by Wisconsin[33] and in Japan[34] , have shown that the risks of fatal and nonfatal stroke are higher in persons with signs of retinopathy. The Atherosclerosis Risk in Communities study, showed that some signs of retinopathy (e.g., retinal hemorrhages, microaneurysms, and cotton-wool spots) were associated with a risk of newly diagnosed clinical stroke that was two to four times as high as that for patients who did not have these signs, even when the analysis was controlled for the effects of long-term elevations in blood pressure, cigarette smoking, elevated lipid levels, and other risk factors for stroke.[35]

The Risk of Coronary Heart Disease

The are only few studies regarding the association of hypertensive retinopathy and the risk of coronary heart disease. In the National Health Examination Survey, persons with retinal arteriolar narrowing, as detected on ophthalmoscopy, were more likely to have preexisting coronary heart disease as those without these changes, after the analysis was controlled for the presence or absence of hypertension and diabetes and for serum cholesterol levels.[36]In a study of 560 men with hypertension and hyperlipidemia, the presence of hypertensive retinopathy predicted a

doubling of the risk of coronary heart disease, and the presence of either generalized or focal narrowing of the arterioles predicted almost a three times risk [37]

Associated factors

Less than half the retinal changes associated with hypertension cannot be explained by high blood pressure. The low sensitivity of retinal abnormalities associated with hypertension indicates that hypertensive retinopathy is not common in hypertensive people. In both the Beaver Dam eye study and the Blue Mountains eye study little difference was found in the presence of haemorrhages and exudates between normotensive and hypertensive people aged over 65.

Various other conditions have been associated with hypertensive retinopathy, such as

1. Ethnicity,[38]
2. Smoking,[41 – 43]
3. Intima-media thickness,[41,42]
4. Carotid plaque score,[42]
5. Carotid artery stiffness,
6. Serum cholesterol concentration,[41,36,43]
7. Diabetes,[36,43]
8. Body mass index.[36,43]

SERUM LIPIDS AND RETINOPATHY

Studies have shown that with increasing severity of hypertension, the prevalenceof elevated total cholesterol, LDL cholesterol and low HDL cholesterol was higher.[7]

According to the American Heart Association guidelines, blood pressure < 130/85 mmHg; total cholesterol < 200 mg/dl; triglycerides < 200 mg/dl; HDL > 40 mg/dl and LDL < 130 g/dl, are favourable risk factors. In addition, certain lipid ratios like total cholesterol / HDL cholesterol and the LDL cholesterol/HDL cholesterol ratio also correlate with cardiovas cular disease. The recommended ratios for the two are ≤ 3.5 [42]

HDL and LDL are two of the four main groups of plasma lipoproteins that areinvolved in lipid metabolism and the exchange of cholesterol, cholesterol ester andtriglycerides between tissues [43,44]

Some studies have shown an inversecorrelation between plasma HDL levels and risk of cardiovascular disease, implying thatfactors associated with HDL protect against atherosclerosis. Some of these factors appear to have anti-oxidant and anti-inflammatory effects which may obviate processes thatinitiate atherogenesis.[45,46]Epidemiological studies have also shown that elevatedconcentrations of total or LDL cholesterol in the blood are risk factors forcoronary disease.[47]

Most extra-hepatic tissues, although having a requirement forcholesterol, have low activity of the cholesterol biosynthetic pathway. Their cholesterol requirements are supplied by LDL, which is internalised by receptor-mediatedendocytosis.

A major function of HDL cholesterol is to enhance reverse cholesteroltransport by scavenging excess cholesterol from peripheral tissues

followed byesterification through lecithin:cholesterolacyltransferase and delivering it to the liver andsteroidogenic organs for subsequent synthesis of bile acids and lipoproteins and eventual elimination from the body. [48,49]This role of HDL has been shown to be responsible for itsatheroprotective properties.

HDL cholesterol also regulates the exchange of proteins andlipids between various lipoproteins. [50]In addition, HDL provides the protein componentsrequired to activate lipoprotein lipase which releases fatty acids that can be oxidised bythe β-oxidation pathway to release energy.[43,44]Most importantly, HDL can inhibitoxidation of LDL as well as the atherogenic effects of oxidised LDL by virtue of itsantioxidant property .[49]

In a prospective study on ocular manifestation of hyperlipoproteinemia, variousocular abnormalities like xanthelesma, arcussenilis and juvenilis, lipid keratopathy, irisxanthoma, lenticular opacities, drusen of macula, lipemiaretinalisetc weredocumented.[51]

In another study to evaluate lenticular opacities in patients with underlyingdyslipidiaemia, it was found that cortical opacification was most prevalent sign ofdyslipidaemia and occurred in relatively young age and cortical opacification should beregarded as an indicator for lipid profile evaluation.[52]

Hypertension with dyslipidaemiacan lead to complication andhence this study to evaluate the role of altered lipid profile on visual function inhypertensive patients.

MATERIALS AND METHODS

SOURCE OF DATA:

This study will be carried out on patients diagnosed to have essential hypertension attending / admitted to B.L.D.E.U'S Shri B.M.Patil Medical College, Hospital and Research centre, Bijapurfrom 1st October 2012- 31st March 2014.

METHOD OF COLLECTION OF DATA

Sample Size: The prevalence rate of hypertension is 5.9% in urban and 3.5 in rural among male population, 6.9% in urban and 3.5% in rural among female population.[13]

The total prevalence rate of hypertension is 19.8% approximated to 20%. Consideringprevalence rate of hypertension 20%, at 95% confidence level and at ±5 margin of error. The calculated sample size is 245.

$$n = \frac{(1.96)^2 \times p \times q}{d^2}$$

p =Prevalence

q = 100-p

d = margin of error.

Hence a minimum of 245 cases ofhypertension will be included in the study.

Statistical analysis:

Data will be analyzed by following methods

1. Mean ± SD
2. Statistical tests like't' and X^2 tests.

Inclusion Criteria

Patients diagnosed to have essential hypertension according to JNC 7 classification will be included in the study.

Exclusion Criteria

- Patients suffering from other diseases like Diabetes, HIV, syphilis, T.B, PIH, secondary hypertension,high myopes, and other retinal disease will be excluded from the study.
- Patients with ocular media haze in both eyes so as to interfere with a detailed examination of the fundus.

INVESTIGATIONS / INTERVENTIONS

- There are no animal experiments involved in this study.
- Blood glucose estimation, Complete blood profile
- Urine examination
- Fasting serum lipid profile.
- Other investigations like Blood urea, serum creatinine,Chest x-ray ECG ECHO, CT scan, abdominal USG will be done wherever required.

RESULTS

The present study **"A CROSS SECTIONAL STUDY OF ASSOCIATION OF FUNDUS MANIFESTATIONS WITH THE SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS. "**Conducted in BLDEA, Hospital and RC Bijapur in Shri B M Patil Medical College during the period of 1st October 2012- 31st March 2014. During this period after satisfying the selection criteria 245 patients were included in this study.

Off the 245 patients, 106(43.26%) patients had normal fundus and 139(56.73%) patients had retinopathy. The prevalence of retinopathy in this study is 56.73%. Among these patients 91 were males and 48 were females. With M: F= 1.9:1 with the age range of 25-85 years (Mean age of 57.16 years). And average duration of hypertension of 6.41 years.

TABLE 1. Prevalence of Hypertensive Retinopathy.

Hypertensive Retinopathy	No. of patients	
Normal	106 (43.26%)	
I	42	139 (56.73%)
II	61	
III	34	
IV	2	

Graph 1: Prevalance of Hypertensive Retinopathy

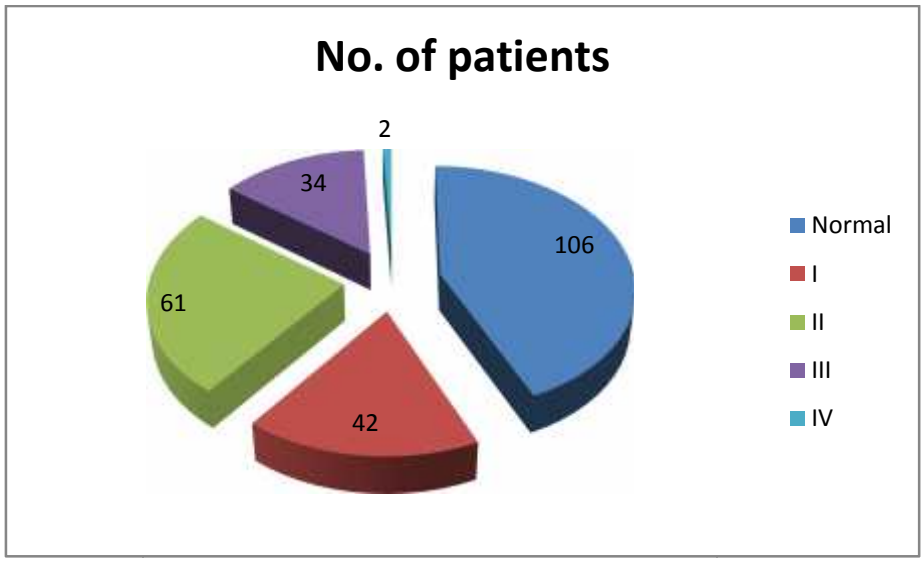


Table No. 2 : Retinopathy in Relation to Age Distribution.

Age	Normal	Grade I	Grade II	GradeIII	Grade IV	Total
<30	4	0	0	1	0	5
31-40	17	3	2	0	0	22
41-50	26	6	4	3	1	40
51-60	43	16	19	9	1	88
>61	16	17	36	21	0	90
Total	102	42	61	33	2	245

X^2=56.4; p<0.0001

Among 245 patients studied, 5(2.04%) patients belong to <30 years age category among this 1(20%) patient had retinopathy and 4(80%) had no retinopathy. 22(8.97%) patients belong to 31-40 years age category. Of these 05(22.72%) had retinopathy whereas 17(77.27%) did not have retinopathy.

The next category consisting of 40(16.32%) patients belong to (41-50) years of age group of which 26(65%) showed no retinopathy, 14(35%) showed retinopathy.

Another category consisting of 88(35.91%) patients belonging to 51-60 years of age group. 45(51.13%) patients had retinopathy, while 43(48.86%) patients, had no retinopathy.

In the last category that is above 60 years had 90(36.73%) patients, of which74(82%) had retinopathy and 16(17.77%) had no retinopathy. Over all there was statistically significant relation found with age distribution.

Graph 2.Retinopathy in Relation to Age Distribution

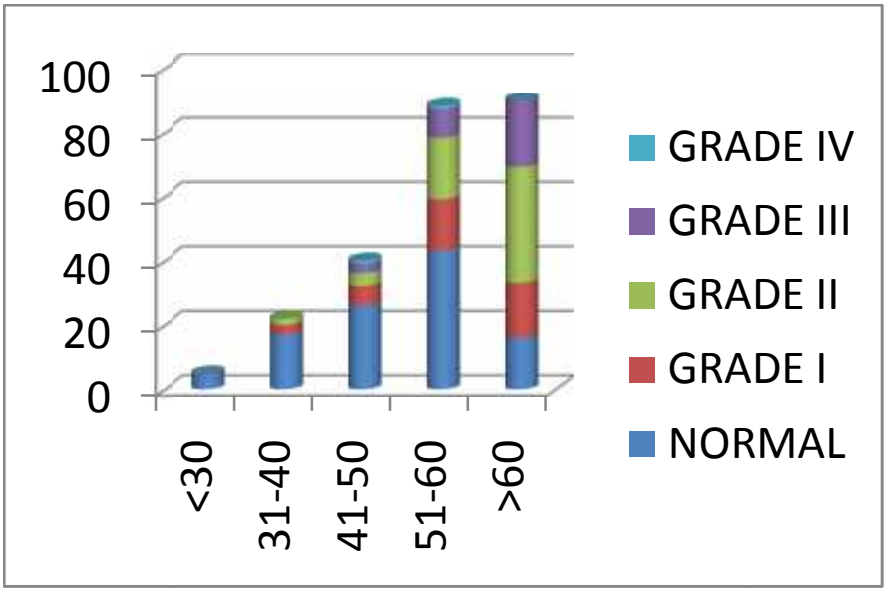


Table No. 3: Retinopathy in Relation to Sex Distribution

SEX	RETINOPATHY	NO RETINOPATHY	TOTAL
MALE	91	73	164
FEMALE	48	33	81
TOTAL	139	106	245

X^2=0.314 ; p=0.57

There were 164 (66.93%) males and 81 (33.06%) females in our study group. Out of which 91(65.46%) males and 48(34.53%) females had retinopathy. The remaining 73(68.86%) males and 33(31.13%) females had no retinopathy respectively. There was no statistical significance.

Graph 3.Retinopathy in relation to sex distribution.

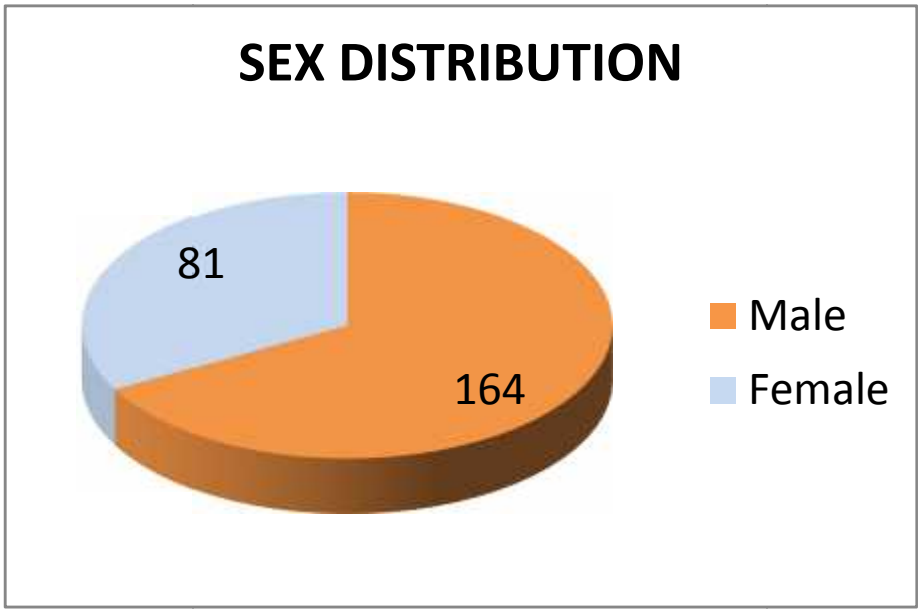


Table 4 : Relationship between Retinopathy and Duration of Hypertension

Grade of Retinopathy	Duration of Hypertension (Yrs)				Total
	0-5	6-10	11-15	>15	
Normal	92	14	00	00	106
I	19	20	03	00	42
II	19	25	13	04	61
III	06	11	09	07	34
IV	00	00	01	00	02
TOTAL	136	71	26	11	245

X^2 =56.5; P<0.001

Of 245 patients, 136 patients had hypertension since 0-5 years. Of which 44 had retinopathy of varying degrees, while 92 had no retinopathy. 71 patients were in 6-10 years, of which 14 patients had no retinopathy, while 57 patients had retinopathy.

Next group of 26 patients, had hypertension since 11-15 years, of which 26(100%) patients had retinopathy.

Among the last group of 11 patients who had hypertension since > 15 years, 11(100%) had retinopathy. Overall, the relationship between various grades of retinopathy and duration was statistically significant (p<0.0001).

Graph 4.Relationship between Retinopathy and Duration of Hypertension.

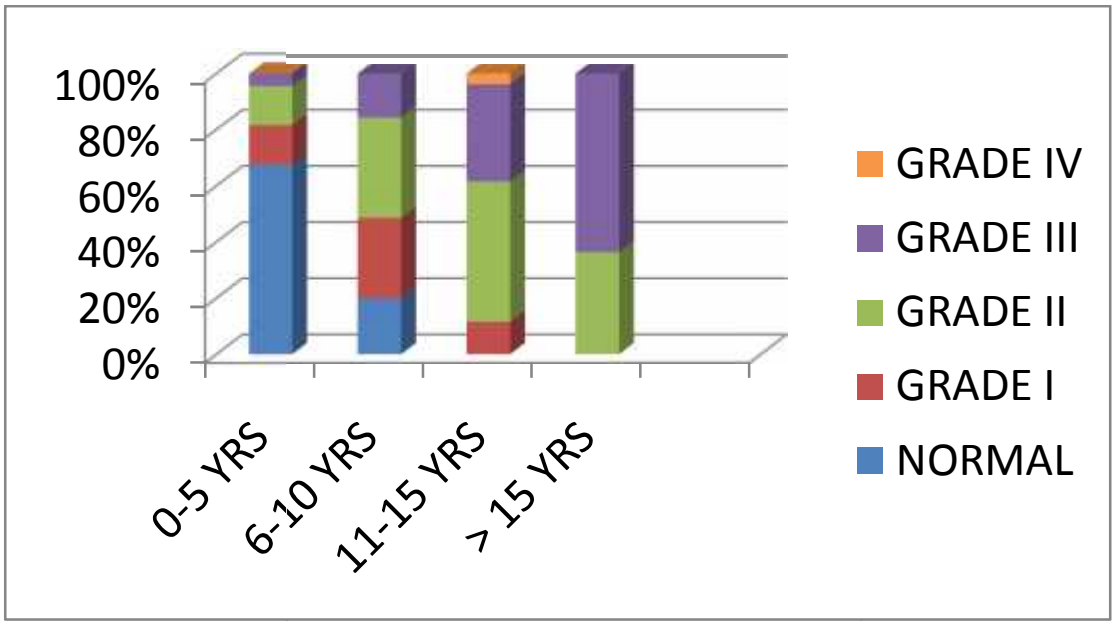


Table 5: Showing Relationship of Retinopathy with Serum Total Cholesterol

Grades of hypertension	Serum total cholesterol levels (mg/dl)			
	<200	200-239	>240	Total
Normal	88	12	06	106
I	34	07	01	42
II	37	15	09	61
III	16	09	09	34
IV	00	01	01	02
Total	175	44	26	245

X^2 =31.5; P<0.001

Out of 245 patients, 175(71.42%) had total serum cholesterol within normal limits (< 200 mg /dl). of which 87(49.71%) patients had retinopathy, while 88(50.28%) patients had no retinopathy.The next group of 44(17.95%) patients had total serum cholesterol between (200-239 mg/dl) which is considered to be borderline. Of which 32(72.72%) patients had retinopathy while 12(27.27%) had no retinopathy.

The last group of 26(10.61%) patients had serum total cholesterol levels of >240 which is considered to be abnormal. Of which 20(76.92%) patients had retinopathy, 6(23.07%) had no retinopathy. Overall the increase in total serum cholesterol levels correlated well with increasing severity of retinopathy ($p<0.0001$).

Graph 5.Showing Relationship of Retinopathy with Serum Total Cholesterol.

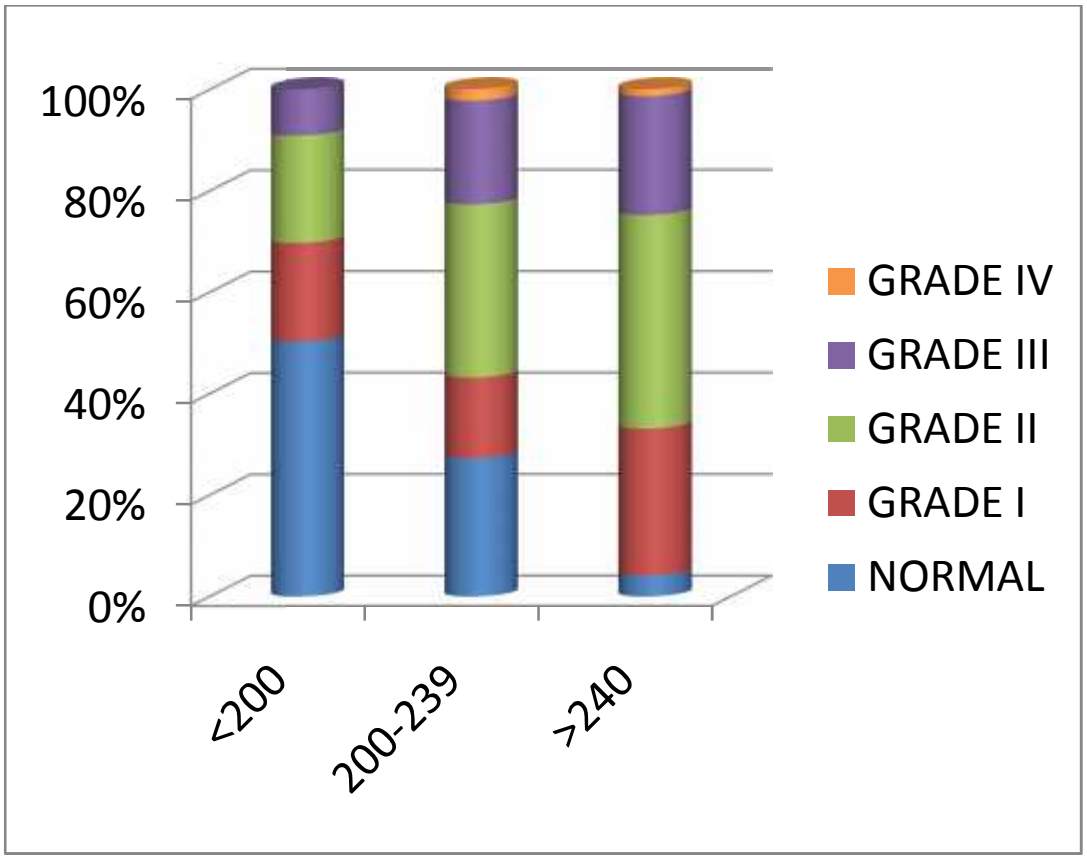


Table No. 6: Showing Relationship of Serum LDL Cholesterol with Retinopathy

Retinopathy	Serum LDL cholesterol (mg / dl) [56]			TOTAL
	< 130 (Normal)	130.159 (Borderline)	> 160 (Abnormal)	
Normal	86	10	10	106
I	38	02	02	42
II	33	12	16	61
III	16	04	14	34
IV	00	00	02	02
TOTAL	173	28	44	245

X^2 =47.7; P<0.001

Out of 245 patients studied. 173(70.61%) had serum LDL cholesterol levels of < 130 mg / dl (normal). Of which 86(49.71%) had no retinopathy while 87(50.28%) had retinopathy of varying grades.

The next group of 28(11.42%) had serum LDL cholesterol levels between (130-159) mg / dl (borderline) of which 10(35.71%) had no retinopathy, while 18(64.28%) had retinopathy.

The last group of 44 (17.95%) patients had serum LDL cholesterol levels of > 160 mg / dl (abnormal) of which 10(22.72%) patients had no retinopathy, while 34(77.27%) had retinopathy.

Overall, the increasing level of serum LDL – cholesterol showed statistically significant correlation with the grades of hypertensive retinopathy (p<0.0001).

Graph 6.Showing Relationship of Serum LDL Cholesterol with Retinopathy

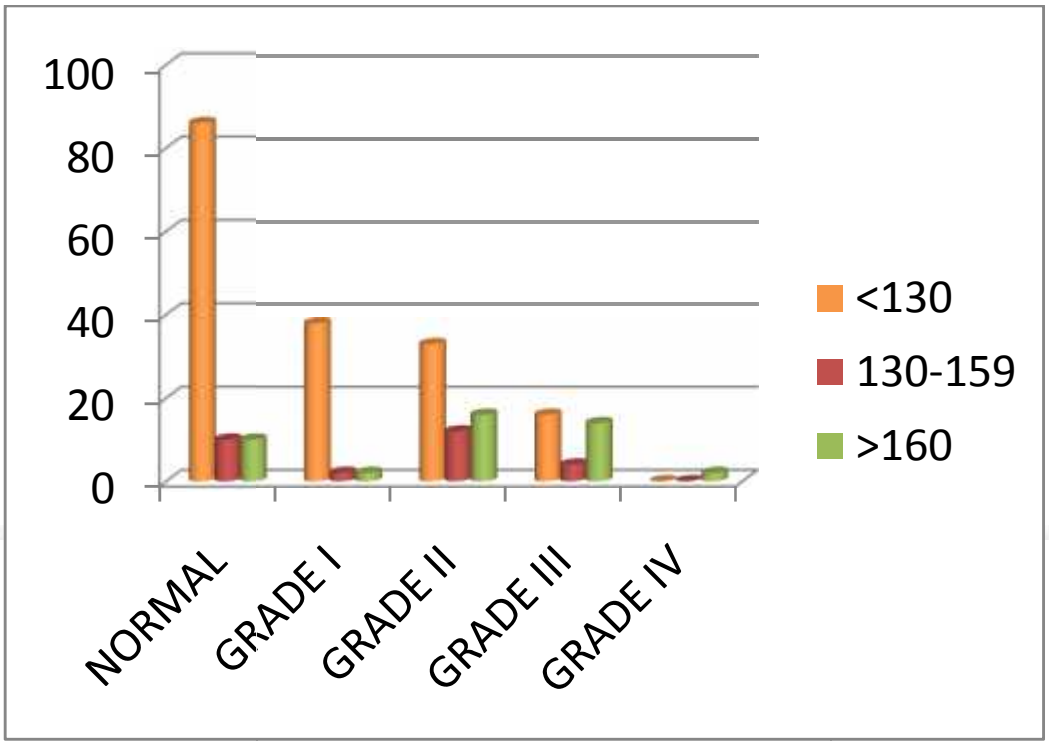


Table 7 : Showing Relationship between Serum HDL- Cholesterol with Retinopathy

Grades of Hypertension	Serum HDl cholesterol (mg/dl)			
	>60	36-60	<35	Total
Normal	01	56	49	106
I	01	26	15	42
II	00	39	22	61
III	00	24	10	34
IV	00	01	01	02
Total	02	146	97	245

X^2 =6.31; P=0.612

Out of 245 patients, 2 (0.81%) patient had serum HDL-cholesterol levels of >60mg / dl, among which 1 had retinopathy and 1 had no retinopathy.

The next group of 146(59.59%) had serum HDL – cholesterol levels between 36-60 ml/dl (borderline) of which 56(38.35%) patients had no retinopathy, whereas 90(61.64%) patients had retinopathy.

The last group of 97(39.59%) patients had serum HDL-C levels of < 35 gm/dl (abnormal) of which 49(50.51%) patients had no retinopathy, while 48(49.48%) had retinopathy.

Overall there was no statistically significant relation between the serum levels of HDL – cholesterol and the grades of retinopathy (p=0.612).

Graph 7.Showing Relationship between Serum HDL- Cholesterol with Retinopathy.

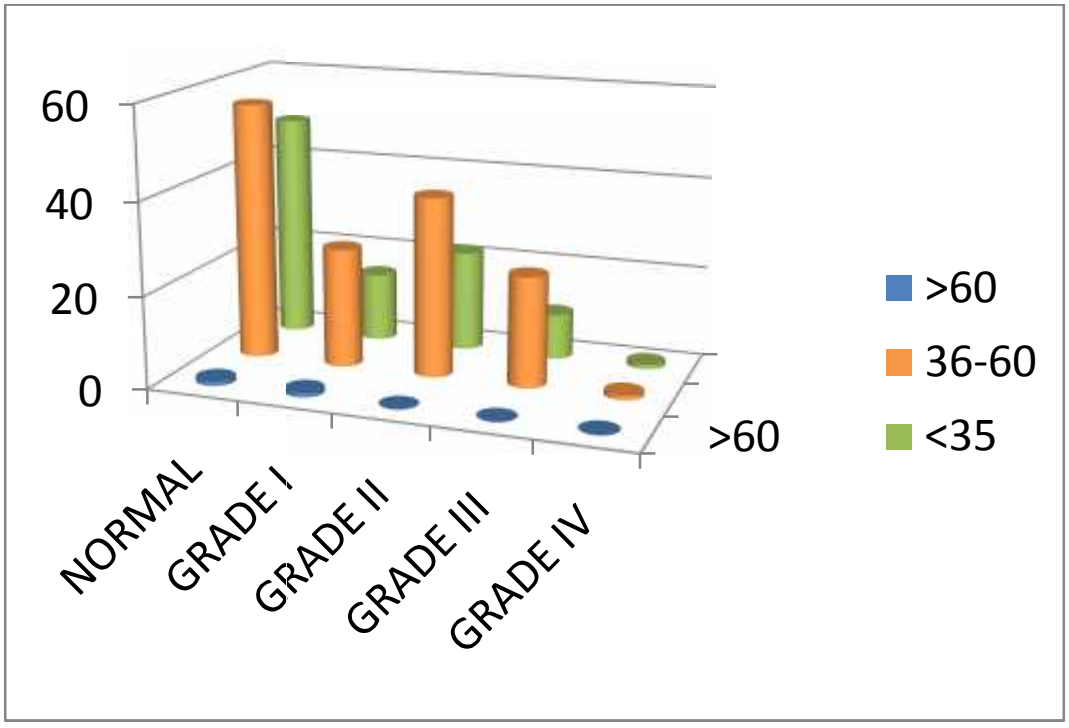


Table 8 : Showing Relationship of Serum Triglycerides with Retinopathy

Retinopathy	Serum TG levels(mg/dl)		
	<150	>150	Total
Normal	90	16	106
I	26	16	42
II	32	29	61
III	14	20	34
IV	02	00	02
Total	164	81	245

X^2 =32.9; P<0.001

Out of 245 patients, 164(66.93%) patients had serum triglycerides levels of < 150normal), of which 90(54.87%) patients had no retinopathy whereas 74(45.12%) patients had retinopathy.

In the remaining 81(33.06%) patients who had serum triglycerides levels of > 150 mg/dl 65(80.24%) patients had retinopathy, whereas 16(19.75%) patients did not.

Overall, serum triglycerides levels correlated positively with increasing severity of retinopathy which was statistically significant (p<0.0001).

Graph 8.Showing Relationship of Serum Triglycerides with Retinopathy

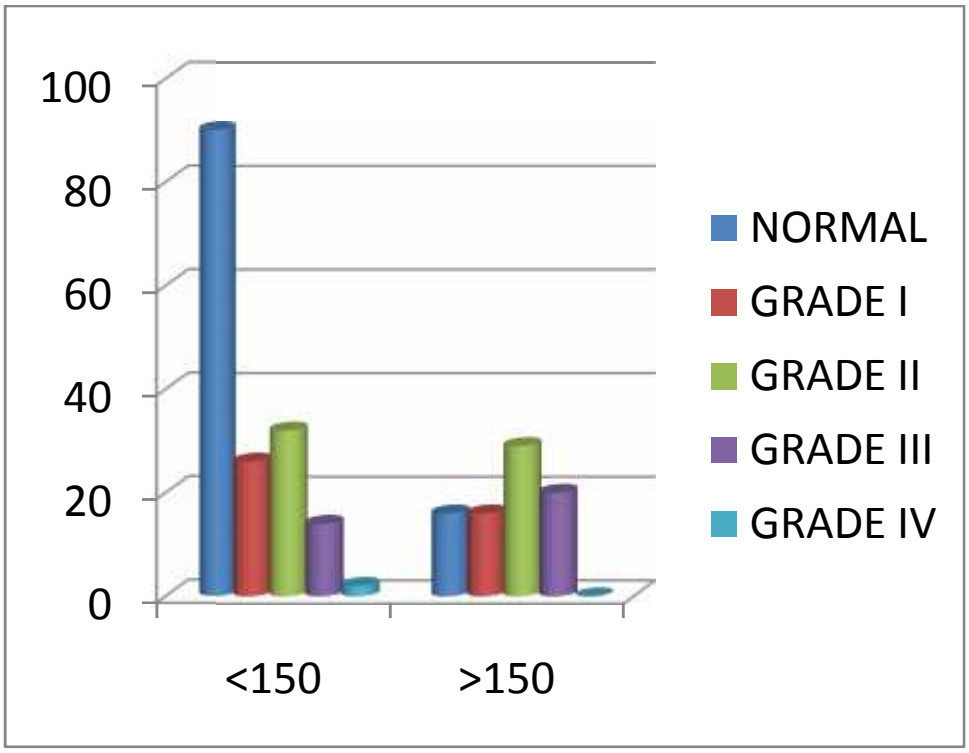


TABLE 9.SHOWING RELATION OF RAISED LDL AND TRIGLYCERIDE WITH RETINOPATHY.

Retinopathy	LDL>160 (TG<150)	TRIGLYCERIDE >150 (LDL<130)	LDL>160, TG>150	TOTAL
NORMAL	06	12	4	22
GRADE I	01	14	01	16
GRADE II	07	13	08	28
GRADE III	07	09	07	23
GRADE IV	02	00	00	02
TOTAL	23	48	20	91

X^2= 16.4; P= 0.037

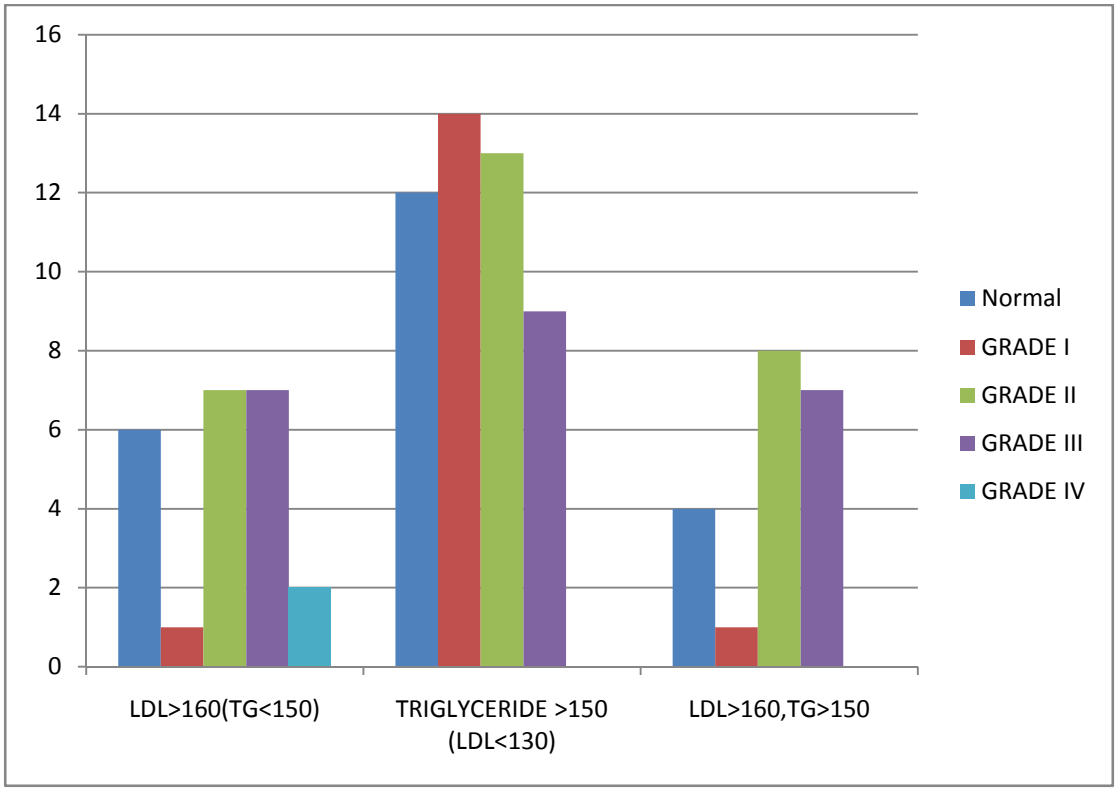
Out of 245 patients, 23(9.38%) patients had LDL>160 mg/dl with TG < 150 mg/dl. Among them 6(26.08%) patients had no retinopathy and 17(73.91%) had retinopathy.

In the next group of TG> 150mg/dl and LDL<130 mg/dl, there were 48(19.59%) patients. 12(25%) had no retinopathy, 36 (75%) had retinopathy.

In the last group of 20(8.16%) patients (LDL>160,TG>150) 04(20%) had no retinopathy, 16(80%) had retinopathy.

Over all there was statistical significance with individual raised levels of LDL, TG and both (P<0.05).

GRAPH 9. SHOWING RELATION OF RAISED LDL AND TRIGLYCERIDE WITH RETINOPATHY



DISCUSSION

A total of 245 patients were included in the study who were diagnosed to have primary essential hypertension. They were divided in to retinopathy group and no retinopathy group initially further retinopathy patients were divided depending on the severity of the retinopathy in to Grade I, II, III& IV.

The mean age of patients in present study population was 57.16 years ranging from 25-96 years. Out of 245 patients, 139 belonged to retinopathy group with mean age of 61.66±11.07 years, remaining 106 patients had normal fundus, with mean age of 51.77±11.51 years.($p<0.0001$)

Epidemiologic studies show that signs of hypertensive retinopathy are common in people 40 years of age or older. Prevalence rates ranged from 2 to 15 percent for various signs of retinopathy[25 - 27]

The higher rates of prevalence in these more recent studies are probably due to a higher sensitivity of photography, as compared with clinical ophthalmoscopy, for detecting certain signs of retinopathy.[4]

There were 164 males and 81 females in our study group, out of which 91(65.46%) males and 48(34.53%) females had retinopathy. 73(68.86%) males and 33(31.13%) females had no retinopathy respectively , there was no significant sex preponderance($p=0.57$)

Variations in the prevalence of specific signs of hypertensive retinopathy according to age and sex have not been consistently demonstrated.[25 - 27]There have been fewer studies of the incidence of hypertensive retinopathy. Two studies indicate that the incidence of various signs of retinopathy over a period of five to seven years ranges from 6 to 10 percent.[28,53]

Blood pressure

In the present study mean diastolic BP was 90.64±16.91 mm of Hg in retinopathy group and 82.18±12.57 mm Hg in No retinopathy group, which was statistically significant.

Mean systolic BP in retinopathy group 147.59±28.11 and in No retinopathy group it was 130.79±22.72 , shows statistical significant.

The mean duration of hypertension was 6.41 years was observed in total study population. Patients with retinopathy had mean duration of hypertension of 8.78 years, patients with No retinopathy had 3.17 years.

Numerous studies have confirmed the strong association between the presence of signs of hypertensive retinopathy and elevated blood pressure.[25 - 29] Two studies have further evaluated the effect of a history of elevated blood pressure on the occurrence of specific retinal signs.[30,31] In both studies, 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[30,31] and may therefore be more indicative of the severity of recent hypertension.

Furthermore, the observation of signs of retinopathy in people without a known history of hypertension suggests that these signs may be markers of a prehypertensive state. For example, generalized and focal narrowing of the retinal arterioles has been shown to predict the risk of hypertension in normotensive

persons.[51] other factors unrelated to hypertension[31] (e.g., hyperglycemia, inflammation, and endothelial dysfunction) may also be involved in the pathogenesis of retinopathy.

Serum lipids

In recent years there have been many studies demonstrating a correlation between increased arterial blood pressure and altered lipid profiles, and there has been an especially positive correlation between high cholesterol levels and blood pressure.[55]

More than 35 years ago, it was noticed that about two-thirds of patients with atherosclerotic retinal changes had atherosclerotic changes of other arteries as well and *vice versa.*[56 - 58]However, the studies did not determine serum lipids and apoproteins inthese patients. The arteries analyzed by ophthalmoscopy (branches of the central retina

artery) do have all the layers (endothelium–intima, basement membrane, media with smooth muscle cells, and adventitia) and resemble small arteries in other organs, including heart and brain.[59] Arterioles with the diameter smaller than 63 and 134 µm lack internal elastic lamina and continuous layer of smooth muscle cells.[60,61]

In our study the patients with retinopathy had mean serum lipid profiles as follows; Total cholesterol 201, LDL-cholesterol 125.47, HDL-cholesterol 37.79, and that of Serum Triglycerides being 169.71. And those with normal fundus had mean serum lipid levels as follows: Total cholesterol 172.09, LDL-cholesterol 103.15, HDL-cholesterol 36.69, and that of Serum Triglycerides being 116.11.

In general the association of serum total cholesterol levels was highly significant ($p<0.0001$). Similarly we found a significant association of serum LDL-Cholesterol and the severity of the retinopathy, ($p<0.0001$).

The mean serum HDL-Cholesterol values for retinopathy group were 38.68 & that for no retinopathy group was 39. There was no significant association of the serum HDL-Cholesterol & the retinopathy.

Although overall association of serum triglycerides was found to be significant with retinopathy ($p<0.0001$).

Change in lipid parameters is associated with changes in the retinal arteries correspond and more or associated with coronary artery atherosclerosis, suggesting once again that atherosclerosis is often a generalized process. Studies have shown that, when end organ vascular damage is an issue, multi-organ involvement is the rule rather than the exception[62] despite the indications that susceptibility to vascular damage of the vascular walls does not seem to be either synchronous or uniform in different areas.

CONCLUSION

Our study showed significant association between serum lipid parameters and hypertensive retinopathy.

Dyslipidaemia is already considered as an important risk factors for end organ failure. For severity of retinopathy dyslipidaemia can be considered a risk factor.

We can conclude that routine ophthalmological check-up of hypertensive patient is must. In patients with signs of hypertensive retinopathy further evaluation of lipid profile helps in early detection and treatment of risk factors. This may be helpful in preventing blindness as well as cardiovascular morbidity and mortality.

SUMMARY

Hypertension is an important worldwide public-health challenge because of its high frequency and concomitant risks of cardiovascular and kidney disease. This trend seems to be associated with socio economic and life style changes.

Studies have shown the brain, heart, eyes and the kidneys are most likely to be affected due to persistent elevation of arterial pressure and increased peripheral resistance. Systemic hypertension also affects the arteries, veins, choroid and the optic nerve in the eyes.

However the effects and many complications of hypertension may be delayed and can be prevented by prompt and effective treatment.

Our study denoted the relationship of dyslipdemia and fundus changes in hypertension. 245 patients having essential hypertension were screened for retinopathy changes. A major proportion amounting to 139(56.73%) patients had retinopathy whereas rest 106(43.26%) had no retinopathy .Subjects having retinopathy were mainly concentrated in the 6^{th} decade (88 patients, 35.91%) and over 60 years who were 90 patients(36.73%). Thus showing increasing prevalence of hypertensive retinopathy with increasing age.

No sex preponderance towards developing retinopathy was found in our study ($p=0.57$).

As regards to the duration of the disease; the prevalence of retinopathy definitely increased with increasing duration. All patients having hypertension for more than 10 years had retinopathy.

Our study showed that hypertensive retinopathy and its overall prevalence bears definite positive correlation with total cholesterol($p<0.0001$); LDL cholesterol ($p<0.0001$), serum triglycerides ($p<0.0001$).

BIBLIOGRAPHY

1. Bastola P, Pun CB, Koirala S, Shrestha UK. Fasting serum lipids and fundus changes in hypertensive patients. Nepal Journal of Medical sciences 2012; 1(2):103-7
2. Gupta R. Trends in hypertension epidemiology in India. J Hum Hypertens.2004; 18: 73-8.
3. .Besharati M R, Rastegar A, Shoja M R, Maybodi M E. Prevalence of retinopathy in hypertensive patients. Saudi Med J 2006; Vol. 27 (11): 1725-8.
4. MahmutIlkerYilmaz, AlperSonmez, SelimKilic, TurgayCelik, NecatiBingol, Murat Pinar, et al. The association of plasma adiponectin levels with hypertensive retinopathy. European Journal of Endocrinology 152 (2) : 233-240.
5. Law MR, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease. BMJ. 1994; 308:367-72
6. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories.Circulation.1998; 97:1837-47.
7. Thakur AK and Achari V. A study of lipid levels in uncomplicated hypertension. Indian heart Journal 2000; 52: 173-7.
8. Reiner E T, Reiner E, Sonicki Z. Atherosclerosis of Retinal Arteries in Men: Role of Serum Lipoproteins and Apoproteins. Croat Med J 2004; 45:333-7.

9. Chew EY, Klein ML, Ferris FL, Remaley NA, Murphy RP, Chantry K. et al. Association of elevated serum lipid levels with retinal hard exudates in diabetic retinopathy. Arch. Ophthalmol1996 ; 114 : 1079-1091.
10. Klein R, Clegg L, Cooper LS, Hubbard LD, Klein BEK, King WN. et al. Prevalence of age related maculopathy in the atherosclerosis risk in community study. Arch Ophthalmol, 1999 ; 117 : 1203-1209.
11. Norman M Kaplan. Systemic hypertension. Mechanism and diagnosis, chapter 28, BraunwaldZipes Libby Heart disease, 6th edition, Edt Eugene Braunwald, WB Saunder's Company 2001 : 942-946.
12. Aram V Chobanian, George L, Henry R, William C, Lee A, Joseph L. et al. Seventh Report of the Joint National Committee on prevention, detection. Evaluation and treatment of high blood pressure. JAMA 2003 ; 289(19) :
13. Gordan H. Williams, Hypertensive Vascular disease, chap 230, Harrisons Principles of Internal Medicine, vol 2, 16th Edition. Dennis L. EugenBraunwald, Anthony S, Stephen L, DANL. J Larry Jameson, McGraw Hill 2005 ; 1463 pp.
14. Berkin KE. Hypertension Essential Hypertension : The heart and hypertension heart 2001 ; 86 : 467-475.
15. Walsh JB. Hypertensive retinopathy: description, classification, and prognosis. Ophthalmology 1982;89:1127-1131.
16. World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. J Hypertens 1999;17:151-183.
17. The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Arch Intern Med 1997 ; 157 : 2413-2446. [Erratum, Arch Intern Med 1998;158:573.]

18. Chobanian AV, Bakris GL, Black HR. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 2003 ; 290:197.
19. Tso MOM, Jampol LM. Pathophysiology of hypertensive retinopathy. Ophthalmology 1982;89:1132-1145
20. Joseph BW, Richard BR, Daniel MB. Systemic Hypertension and the Eye, Duane's Ophthalmology 1999 CD-ROM Edition clinical volume 3 chapter 13 Lippincott Williams & Wilkins record 27788-
21. Hayreh SS, Servais G, Virdi PS: Retinal arteriolar changes in malignant arterial hypertension.Ophthalmologica1989 ; 198:178.
22. Hayreh SS, Servais G, Virdi PS: Fundus lesions in malignant hypertension. IV. Focal intraretinalperiarteriolar transudates. Ophthalmology 1986 ; 93:60.
23. Klein R, Klein BEK, Moss SE Wang Q : Hypertension and retinopathy, arteriolar narrowing, and arteriovenous nicking in a population. Arch Ophthalmol1994 ; 112:92.
24. Hayreh SS, Servais G, Virdi PS: Fundus lesions in malignant hypertension: V. Hypertensive optic neuropathy. Ophthalmology 1986 ; 93:74
25. Sossi N, Anderson DR: Blockage of axonal transport in optic nerve induced by elevation of intraocular pressure; Effect of arterial hypertension induced by angiotensin I. Arch Ophthalmol 1983 ; 101:94, 1983.
26. Yu T, Mitchell P, Berry G, Li W, Wang JJ. Retinopathy in older persons without diabetes and its relationship to hypertension. Arch Ophthalmol 1998;116:83-89

27. Wong TY, Klein R, Sharrett AR. The prevalence and risk factors of retinal microvascular abnormalities in older people: the Cardiovascular Health Study. Ophthalmology 2003 ; 110 : 658-666.
28. van Leiden HA, Dekker JM, Moll AC,. Risk factors for incident retinopathy in a diabetic and nondiabetic population: the Hoorn study. Arch Ophthalmol2003 ; 121 : 245-251.
29. Hubbard LD, Brothers RJ, King WN,. Methods for evaluation of retinal microvascular abnormalities associated with hypertension/sclerosis in the Atherosclerosis Risk in Communities Study. Ophthalmology 1999 ; 106 : 2269-2280.
30. Sharrett AR, Hubbard LD, Cooper LS,. Retinal arteriolar diameters and elevated blood pressure: the Atherosclerosis Risk in Communities Study. Am J Epidemiol1999 ; 150 : 263-270.
31. Wong TY, Hubbard LD, Klein R, Retinal microvascular abnormalities and blood pressure in older people: the Cardiovascular Health Study. Br J Ophthalmol2002 ; 86 : 1007-1013.
32. Wong TY, Klein R, Klein BEK, Tielsch JM, Hubbard LD, Nieto FJ. Retinal microvascularabnormalities, and their relationship with hypertension, cardiovascular disease, and mortality. SurvOphthalmol 2001;46:59-80.
33. Wong TY, Klein R, Nieto FJ,. Retinal microvascular abnormalities and 10-year cardiovascular mortality: a population-based case-control study. Ophthalmology 2003; 110:933-940.
34. Nakayama T, Date C, Yokoyama T, Yoshiike N, Yamaguchi M, Tanaka H. A 15.5-year follow-up study of stroke in a Japanese provincial city: the Shibata Study. Stroke 1997;28:45-52.

35. Wong TY, Klein R, Couper DJ, Retinal microvascular abnormalities and incident strokes: the Atherosclerosis Risk in Communities Study. Lancet 2001 ; 358:1134-1140.
36. Gillum RF. Retinal arteriolar findings and coronary heart disease. Am Heart J 1991 ; 122 : 262-263.
37. Duncan BB, Wong TY, Tyroler HA, Davis CE, Fuchs FD. Hypertensive retinopathy and incident coronary heart disease in high risk men. Br J Ophthalmol2002 ; 86 : 1002-1006.
38. Wong TY, Klein R, Duncan BB, Nieto FJ, Klein BE, Couper DJ, Racial differences in the prevalence of hypertensive retinopathy. Hypertension 2003;41: 1086-91.
39. Ikram MK, de Jong FJ, Vingerling JR, Witteman JC, Hofman A, Breteler MM, et al. Are retinal arteriolar or venular diameters associated with markers for cardiovascular disorders.The Rotterdam Study. Invest Ophthalmol Vis Sci2004 ; 45 : 2129-34.
40. Klein R, Sharrett AR, Klein BE, Chambless LE, Cooper LS, Hubbard LD, et al. Are retinal arteriolar abnormalities related to atherosclerosis: the atherosclerosis risk in communities study.ArterioscIerThrombVascBiol 2000;20: 1644-50.
41. Wong TY, Knudtson MD, Klein R, Klein BE, Hubbard LD. A prospective cohort study of retinal arteriolar narrowing and mortality. Am J Epidemiol 2004; 159: 819-25.
42. Glew RH, Williams M, Conn CA, Cadena SM, Crossey M, Okolo SN: Cardiovascular disease risk factors and diet of Fulani pastoralists of northern Nigeria. Am J Clin Nutr 2001, 74**:**730-736.

43. Gordon DJ, Rifkind BM: High-density lipoprotein: the clinical implications of recent studies. N Eng J Med 1989 ; 321(19):1311-1316.
44. McNamara DJ: Dietary fatty acids, lipoproteins, and cardiovascular disease. Adv Food Nutr Res 1999; 36:253.
45. Navab M, Berliner JA, Watson AD, Hama SY, Territo MC, Lusis AJ, Shih DM, Van Lenten BJ, Frank JS, Demer LL, Edwards PA, Fogelman AM: The Yin and Yang of oxidation in the development of the fatty streak. A review based on the 1994 George Lyman Duff Memorial Lecture. ArteriosclerThrombVascBiol 1996, 16:831-842.
46. Oram JF, Lawn RM: ABCA1: the gatekeeper for eliminating excess tissue cholesterol. J Lipid Res 2001, 42:1173-1179.
47. Law MR: Lowering heart disease risk with cholesterol reduction : evidence from observational studies and clinical trials. Eur Heart J Suppl 1999, 1:S3-S8.
48. Stein O, Stein Y: Atheroprotective mechanisms of HDL. Atherosclerosis 1999 ; 144 : 285303.
49. Das DK: Cardioprotection with high density lipoproteins. Fact or friction?Circ Res 2003 ; 92:258-260.
50. Jacques PF, Bostom AG, Williams RR, Ellison RC, Eckfeldt JH, Rosenberg IH, Selhub J, Rozen R: Relation between folate status, a common mutation in methylene tetrahydrofolatereductase and plasma homocysteine concentrations. Circulation 1996 ; 93:7-9.
51. Vinger PF and Sacs BA.Ocular manifestations of hyper lipoproteinaemia. Am J Ophthalmol, 1970 ; 70 : 563-573.

52. Meyer D, Maritz FJ, Liebenberg PH, Parkin DP, Burgess LJ. Cortical lens opacities in the young patients – an indication for a lipogram. Ophthalmology 2003 ; 110(3) : 578-583.
53. Klein R, Klein BEK, Moss SE. The relation of systemic hypertension to changes in the retinal vasculature: the Beaver Dam Eye Study. Trans Am OphthalmolSoc1997 ; 95 : 329-350
54. Wong TY, Klein R, Sharrett AR, et al. Retinal arteriolar diameters and risk for hypertension. Ann Intern Med 2004 ; 140 : 248-255.
55. Flesch M, Sachinidis A, Ko YD, Kraft K, Vetter H Plasma lipids and lipoproteins and essential hypertension. Clin Investig. 1994 ; 72(12):944-50.
56. Sautter H. “Early atherosclerotic syndrom ”of the eye [in German]. BerDtschGesOphtalmol. 1964;65:379-4.
57. Utermann D, Klempien EJ. On the relationships between the symptoms of arteriosclerosis on the fundus oculi and the degenerative diseases of the general vascular system.A biochemical and clinical-statistical study [in German].Doc Ophthalmol. 1968;24:201-74.
58. Utermann D. Arteriosclerosis of the eye. What the internist should know [in German]. Internist (Berl).1987; 28:357-65.
59. Hogan MJ, Feeney L. The ultra structure of the retinal blood vessels. I. The large vessels. J Ultrastruct Res. 1963;39:10-28.
60. Mausolf FA, editor. The eye and systemic disease. London: Mosby; 1980.
61. Hogan MJ, Feeney L. The ultrastructure of the retinal vessels. II. The small vessels. J Ultrastruct Res. 1963;49: 29-46.
62. Hertzer NR. Basic data concerning associated coronary disease in peripheral vascular patients. Ann Vasc Surg. 1987;1:616-20.

ANNEXURE-I

ETHICAL CLEARANCE

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-12 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title A cross sectional study of association of fundus manifestations with the serum lipid profile in hypertensive patients

Name of P.G. student Dr Shravan Maskar
Ophthalmology

Name of Guide/Co-investigator Dr M. H. Patil
Prof. ophthalmology

DR.TEJASWINI VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE-II

SAMPLE INFORMED CONSENT FORM:

TITLE OF PROJECT: "A CROSS SECTIONAL STUDY OF ASSOCIATION OF FUNDUS MANIFESTATIONS WITH THE SERUM LIPID PROFILE IN HYPERTENSIVE PATIENTS."

GUIDE : DR.M.H.PATIL

PRINCIPAL INVESTIGATOR : DR. SHRAVAN MASURKAR

PURPOSE OF RESEARCH

I have been informed that this study will analyse association between serum lipid parameters and hypertensive retinopathy.

PROCEDURE

I am aware that in addition to routine care received and I will be asked series of questions by the investigations.I have been asked to undergo the necessary investigation,which will help the investigator as a part of routine management.

RISKS AND DISCOMFORT

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to these feeling which are associated with the usual course of treatment.

BENEFITS

I understand that my participation in this study will help to analyze association between serum lipid parameters and hypertensive retinopathy.

CONFIDENTIALITY

I understand that medical information procedure by this study will become a part of my Hospital records and will be subject to the confidentiality and privacy regulation of the said hospital information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigators research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and videotapes or audio will be used only with my special written permission . I understand I may see the photographs and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION

I understand that i may ask more questions about the study at any time. **Dr Shravan Masurkar** is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OR PARTICIPATION

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present of future care at this hospital. I also understand that

Dr. Shravan Masurkar may terminate my participate in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

INJURY STATEMENT

I understand that in the unlikely event of injury to me resulting directly from my participation in this, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained (patients/relevant name)the purpose of the research, the procedures risks and benefits to the best of my ability in patients own language.

Investigator: Date:

I confirm that **Dr. Shravan Masurkar** has explained to me the research ,the procedure that I will undergo, and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore I agree to give my consent to participate as a subject in this research project.

Participant / Guardian: Date:

Witness to signature: Date:

ANNEXURE-III

CASE SHEETPROFORMA

Name: Date:

Age/Sex: O.P.No./I.P.NO

Occupation/Education/Marital status/Rural/Urban:

DOA: DOD:

Address:

Socio Economic Status:

History of presenting complaints:

General complaints:

Giddiness

Blurring of vision.

Headache

Ocular Symptomatology:

Past history :

History of similar complaints

History of ocular diseases

History of Hypertension/Diabetes/STD's/PTB

Treatment history:

Family History

1. Diabetes mellitus
2. Hypertension
3. Ocular disease

Personal History

1. Smoking Yes/No/Stopped Since
2. Alcohol Yes/No/Stopped Since
3. Tobacco Yes/No/Stopped Since

General Examination : Built Weight

Pallor Clubbing

Icterus Cyanosis

Lymphadenopathy

Vital Data : Pulse Temperature

BP RR

Systemic Examination: CVS RS

CNS GIT

Ocular Examination :

Right Eye		Left Eye
	Eye Brow	
	Eye lids and Adnexa	
	(Lacrimal sac)Skin, Lid margin, Eye lashes	
	Eye Balls	
	Position, Visual axis, Size of the eye ball	
	Ocular movements	
	Conjunctiva	
	Sclera	
	Cornea	
	Anterior Chamber	
	Iris	
	Pupil	
	Lens	
	Vision	
	Fundus	
	Intra ocular tension	
	Corneal sensations	
	Corneal staining	
	Direct ophthalmoscopy	

Indirect ophthalmoscopy

Anterior segment photograph

Fundus photograph

Investigations

Blood test

Hb	TC
DC	
ESR	RBS

Urine:

Sugar

Albumin

Microscopic

Lipid profile:

Total cholesterol

LDL

HDL

VLDL

Triglycerides

LDL: HDL

Other investigations

Diagnosis:

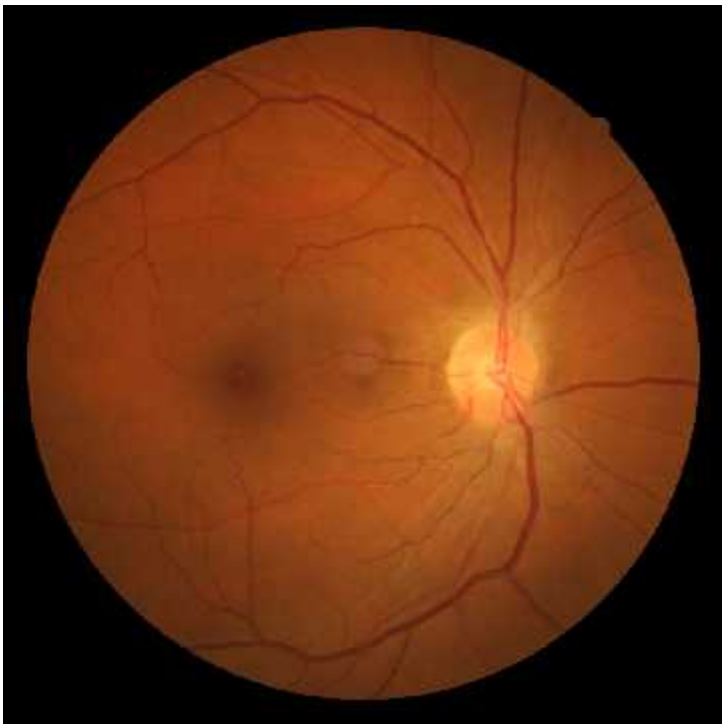
ANNEXURE-IV

KEYS TO MASTER CHART

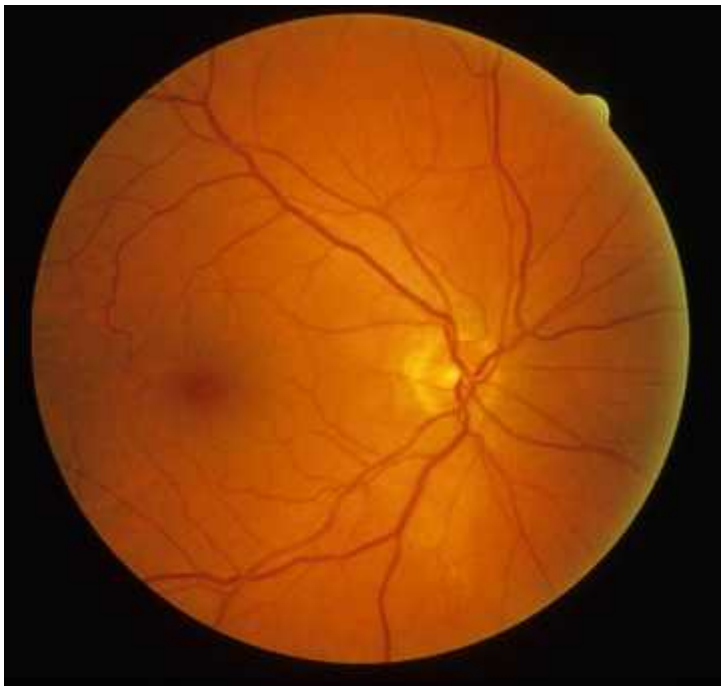
FBS	-	FASTING BLOOD SUGAR
HDL	-	HIGH DENSITY LIPOPROTEIN
LDL	-	LOW DENSITY LIPOPROTEIN
TG	-	TRIGLYCERIDE
BCVA	-	BEST CORRECTED VISUAL ACUITY
IOL	-	INTRA OCULAR LENS
CAT	-	CATARACT
PTER	–	PTERYGIUM
NS	-	NUCLEAR SCLEROSIS
PSC	–	POSTERIOR SUBCSPSULAR CATARACT
Y	-	YES
N	-	NO
OD	–	OCULUS DEXTER
OS	–	OCULUS SINISTER

ANNEXURE-V

GRADE I

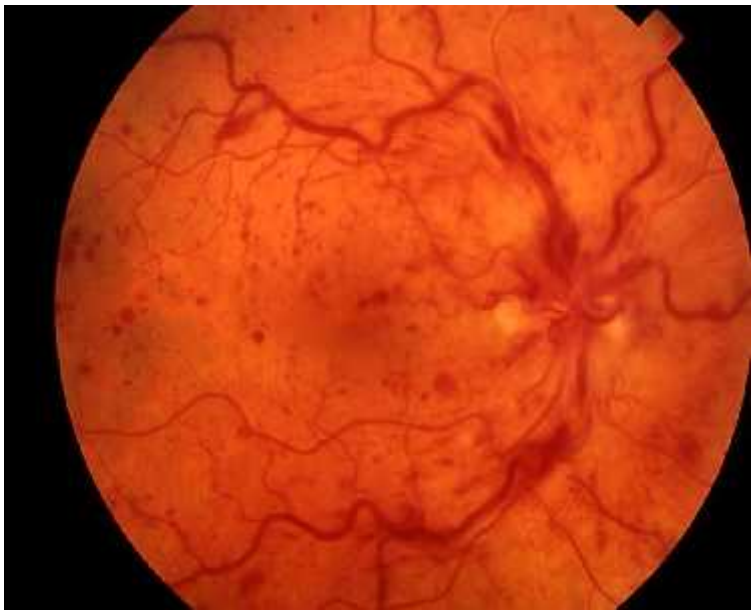


GRADE II



Grade III

GRADE IV



Sl. No.	Name	Age (Yrs)	Sex	IP No.	Duration (Yrs)	B.P. Sys mm Hg	B.P. Dia mm Hg	FBS mg/dl	Cholestrol				Dyslipidemia	BCVA		adnexa		conjunctiva		Cornea		AC		Pupil		Lens		Grades
									Total	HDL mg/dl	LDL	TG		OD	OS	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS	
1	Ganesh	44	M	34530	6	160	100	105	198	42	86	102	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
2	M L Hiremath	70	M	37067	10	180	90	95	196	36	168	176	Y	6/9,N6	6/60,N24	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	CAT,	II
3	Putalabai	60	F	37311	5	144	90	96	176	42	84	71	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
4	Basavaraj	49	M	36679	10	160	100	106	268	32	218	124	Y	6/24,N12	6/9,N8	N	N	N	N	N	N	N	N	RRR	RRR	N	N	IV
5	Ameenappa	57	M	40377	5	170	120	103	172	43	105	119	N	6/9,N6	6/9,N6	N	N	N	N	N	N	N	N	RRR	RRR	PSC	PSC	I
6	Shantawwa	55	F	40323	6	150	80	108	187	42	164	88	Y	6/9,N6	6/6P,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	NS	NS	III
7	Harish	55	M	36422	8	190	110	103	182	40	55	33	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
8	Gangaram	52	M	43477	10	190	120	96	190	42	92	86	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
9	yallappa	65	M	44012	5	150	80	101	189	42	86	150	N	6/12,N10	6/6P,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N+C	IOL	I
10	sangawwa	65	F	40532	5	150	90	112	179	39	94	88	N	6/9,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	IRR,NR	RRR	IOL	IOL	I
11	sangappa	65	M	45448	14	170	100	98	199	40	131	139	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
12	mannawwa	45	F	45377	4	150	90	106	182	46	147	133	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
13	kallappa	52	M	45265	4	160	96	108	217	36	146	175	Y	6/6,N6	6/6,N6	N	N	PTER	PTER	arcus	arcus	N	N	RRR	RRR	N	N	II
14	H D Karekal	65	M	45502	15	130	/92	95	232	42	173	166	Y	CF2MT	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	IOL	III
15	bhagirathi	48	F	46883	12	160	90	83	199	38	92	122	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
16	annaraya	69	M	47705	14	220	130	109	182	40	168	71	Y	6/60,N24	6/12,N8	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	III
17	Shashikala	42	F	44016	2	180	94	94	174	40	151	105	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
18	somolu	54	M	44921	5	150	88	92	183	42	84	120	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	NS	NS	II
19	abdul	42	M	307550	4	148	90	92	160	38	102	111	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
20	kareappa	58	M	309393	0	150	92	86	183	42	106	185	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	NS	NS	II
21	annapurna	68	F	315631	14	210	110	94	166	41	106	85	N	6/9,N6	6/12,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	NC	NC+PSC	I
22	balakrishna	70	M	311822	17	160	96	96	239	43	198	100	Y	6/9,N6	6/36,N18	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	CAT,	II
23	shantabai	65	F	316557	4	142	90	62	192	39	156	120	Y	6/9,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	N
24	parwathi	36	F	318171	4	140	90	105	163	40	93	161	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
25	RAMANAGOUDA	59	M	319559	9	220	140	106	270	38	212	134	Y	CF2MT	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	II
26	bhimanna	45	M	322064	6	190	100	101	184	36	170	132	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	N
27	fatima	60	F	323957	15	210	100	108	195	40	92	162	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	II
28	mallapa	60	M	325325	20	150	100	105	187	40	91	164	Y	6/12P,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	IOL	II
29	Sonawwa Latti	75	F	133462	8	162	104	108	173	38	99	78	N	6/12,N10	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	I
30	Ajaib Singh	75	M	140441	12	156	98	95	203	42	119	58	N	6/36,N18	6/12,N10	N	N	N	N	arcus	arcus	N	N	IRR,SR	RRR	CAT	IOL	I
31	Salman Shaik	36	M	125646	0	170	90	111	169	49	76	88	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
32	Laxmi Achar	50	F	130906	14	240	124	109	235	39	199	233	Y	6/6,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	III
33	Chennabasappa Badashet	43	M	125587	7	140	110	95	195	39	154	178	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
34	Leevalathi Angadi	36	F	130187	5	170	100	64	179	42	98	82	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
35	Sachin Naik	28	M	130277	0	154	90	107	186	45	112	75	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
36	Raju Kadai	44	M	126440	5	150	92	96	197	39	199	297	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	III
37	Laxman R. Dhotmal	67	M	126389	10	140	110	105	186	39	130	178	Y	6/6,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	CAT,	I
38	Mehboobi Hudli	55	F	126357	6	150	90	106	185	40	84	117	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
39	Ishwar Ligade	72	M	126509	8	140	92	111	169	42	109	130	N	6/9,N6	6/60,N36	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
40	Vasudev Patil	61	M	126118	10	130	96	108	209	39	114	175	Y	6/6,N6	6/9,N6	N	N	N	N	N	N	N	N	RRR	RRR	NS	CAT,	II
41	Sharifa N Tengimkeri	60	F	124352	11	150	86	102	191	42	96	113	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	NS	NS	II
42	Prakash Dalvai	64	M	121132	14	180	136	96	196	38	140	80	Y	6/60,N24	6/9,N6	N	N	N	N	N	N	N	N	RRR	RRR	PSC	PSC	II
43	Sheetala P Desai	50	F	120738	4	150	90	106	183	40	146	109	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	I
44	Shankar Rao	72	M	120888	13	160	100	98	236	42	180	88	Y	6/9,N6	6/6,N6	N	N	PTER	N	arcus	arcus	N	N	RRR	RRR	N	N	III
45	Haribabu	46	M	130936	6	170	100	106	248	43	87	202	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
46	Kashibai Mirji	65	F	143952	16	170	100	81	237	41	169	135	Y	CF1MT	6/9P,N6	N	N	N	N	OPAC	arcus	N	N	RRR	RRR	N	CAT,	II
47	Purushotam Patel	53	M	141021	20	190	100	108	286	37	258	98	Y	6/9,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	III
48	Sonabai Pattar	70	F	143256	8	140	90	104	192	46	120	120	N	6/9,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	II
49	Mahadevi Pattanashetty	65	F	144612	0	160	100	98	184	45	134	124	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
50	Laxmibai Kuradi	75	F	144767	4	140	80	92	176	48	102	144	N	6/12,N10	CF3MT	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	I
51	Dattareyamali	53	M	144930	5	160	100	108	184	33	157	203	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	III
52	Neelatai Jole	78	M	145408	6	130	90	99	213	39	65	137	N	6/9,N6	CF3MT	N	N	N	N	N	N	N	N	RRR	RRR	IOL	CAT,	II
53	Balabai Khot	83	F	146165	15	140	70	104	200	32	178	222	Y	CF4MT	PL,PR	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	CAT,	II
54	Ashok Arabhavi	65	F	144951	12	200	100	101	196	36	107	85	N	6/9,N6	6/24,N12	N	N	N	N	arcus	OPAC	N	N	RRR	RRR	PSC	PSC	II
55	Mumtaz Begum	40	M	142140	5	220	110	107	186	40	84	59	Y	6/6,N6	6/6,N6	N	N	N	PTER	N	N	N	N	RRR	RRR	N	N	I
56	Nandakumar	68	F	145095	11	130	90	106	297	28	238	156	Y	6/18P,N8	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	II
57	Vasant Kalburgi	51	M	145542	0	150	90	103	192	36	96	101	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
58	Somalingappa Karki	75	M	144396	18	200	110	103	265	44	115	31	Y	6/12P,N6	CF1MT	N	N	N	N	arcus	arcus	N	N	RRR	RAPD	CAT	CAT,	III

61	Yellappa Jadav	55	F	159863	9	170	100	98	239	42	136	307	Y	CF1MT	6/9,N6	N	N	N	N	arcus	arcus	N	N	RAPD	RRR	N	N	II
62	Subash Jorapur	56	M	159905	7	160	90	104	215	52	38	127	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
63	Kallappa Matur	52	M	160088	15	150	90	104	208	44	112	133	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
64	Bhupal Mamdapur	56	M	158793	5	160	90	99	186	55	89	78	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
65	Ashima Jain	40	M	158938	0	160	90	106	223	28	188	134	Y	PL,PR	6/6,N6	N	N	N	N	N	N	N	N	RAPD	RRR	N	N	II
66	Maruthi Gujnal	51	F	159133	6	160	100	96	201	45	91	111	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
67	Vittal Patil	44	M	157700	5	130	90	102	167	32	81	172	Y	6/6,N6	6/6,N6	N	N	PTER	PTER	N	N	N	N	RRR	RRR	N	N	I
68	Neela Gomadi	45	M	158177	4	150	90	103	160	32	70	92	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
69	Ninganagouda Patil	45	F	156823	4	140	90	104	179	43	110	128	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
70	Mangilal Jain	42	M	157046	5	140	90	92	158	30	96	108	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
71	Sambojirao More	65	M	109942	16	200	100	106	220	32	189	198	Y	6/12,N8	6/12,N9	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	III
72	machibai Jain	45	M	160261	5	190	120	108	162	24	56	110	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
73	Leelavathi Ratnoji	72	F	160773	7	150	90	105	194	68	110	80	N	6/18,N10	6/60,N24	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	I
74	Veena Kamath	55	F	162190	4	142	92	94	198	43	167	138	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
75	Lingappa Bhumadgi	55	F	160283	5	130	94	102	185	13	128	219	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
76	Arun Potdar	65	M	162935	0	140	90	102	160	38	78	78	N	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	N
77	Laxmibai Kuradi	58	M	162603	0	150	94	93	186	32	70	65	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
78	Sudhir	55	F	162848	4	150	100	102	185	32	135	136	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	N
79	Appaji Ramgouda	38	M	41752	0	140	92	103	177	33	110	170	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
80	Sharifa Tenginkeri	63	M	158140	4	150	90	92	162	38	80	78	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	N
81	Shivangouda Patil	60	F	162540	5	140	90	106	198	42	110	126	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	N
82	Sridevi	59	M	119961	4	140	92	92	163	32	78	87	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
83	Ashok keragol	45	F	120415	4	140	90	90	191	29	110	87	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
84	Arun Gurade	58	M	133408	8	140	96	90	201	24	188	145	N	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	III
85	Shankar Naik	40	M	131446	7	200	180	98	167	39	108	259	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
86	Parwathi Balekundri	70	M	121888	4	140	90	102	173	30	67	188	Y	6/9,N8	6/12,N8	N	N	N	N	arcus	arcus	N	N	RRR	IRR,SLUG	IOL	IOL	N
87	Kadappa Hanchinal	73	F	165005	15	160	70	102	187	28	192	86	N	CFCF	6/24,N12	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	III
88	Amarsingh Lamba	65	M	164335	9	170	80	96	214	37	65	59	N	6/6,N6	6/60,N18	N	N	PTER	PT	arcus	arcus	N	N	RRR	RRR	IOL	CAT,	II
89	Prakash Bhaskar Gerkal	42	M	163050	8	150	90	108	196	26	123	283	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	III
90	Laxmibai Patil	51	M	163162	13	160	100	92	230	36	203	87	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	IV
91	Baswaraj Patted	65	F	164973	5	120	90	105	168	54	89	67	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
92	Ramesh	62	M	165011	6	130	90	101	239	24	133	319	Y	6/9,N6	CF1/2MT	N	N	N	N	arcus	arcus	N	N	RRR	SLUG	N	N	III
93	Apasab Tavadare	56	M	165212	11	180	110	86	208	35	198	91	N	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	II
94	Babasaheb Sharbidri	60	M	145600	6	200	110	86	207	46	101	68	N	6/6,N6	6/6,N6	N	N	PTER	PTER	arcus	arcus	N	N	RRR	RRR	N	N	N
95	Giri Pikle	42	M	146477	4	150	90	92	189	42	107	150	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
96	Kalangouda B Patil	85	M	146490	30	150	90	106	226	36	126	436	Y	CF5MT	6/18P,N18	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	III
97	Mallawwa	62	M	165223	6	130	90	96	191	56	99	88	N	6/9,N6	NOPL	N	N	N		arcus	N	N	N	RRR	RRR	N	N	II
98	Mohammed Sharif	60	F	165215	5	130	92	92	186	25	101	120	N	6/9,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT,	N
99	Suryakanth	56	M	165219	4	140	82	96	172	38	158	256	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	III
100	sheela	54	M	165320	4	130	94	104	163	44	82	99	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
101	Hanamantaraya	60	F	9718	1	170	100	129	155	31	103	105	N	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
102	HIRU RATHOD	82	m	9721	7	150	120	144	175	53	101	102	N	CF 6	CF 6	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	II
103	SUMAN	42	M	10265	0	130	90	97	103	36	51	79	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
104	RAFEQ	65	M	26367	30	134	90	131	263	44	112	497	Y	CF2M	CF 6M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	IOL	III
105	MAHADEV	60	M	28183	2	180	100	100	212	45	150	83	N	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	N
106	kalavati	55	M	28185	5	120	92	75	135	30	87.6	87	N	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT,	N
107	SHARANAGOUDA	38	F	11242	1	116	70	97	189	77	105	218	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
108	SADASHIV	58	M	11243	5	130	80	135	333	37	239.3	257	Y	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	CAT,	III
109	SHAKARAPPA	58	M	12263	3	110	70	138	150	21	71	190	Y	6/24,N6	6/24,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
110	NEELKHANT	64	M	12367	10	120	94	217	286	46	207.6	190	Y	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	III
111	SHANKARGOUDA	52	M	12398	4	170	80	154	184	29	98	285	Y	6/24,N6	6/24,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	II
112	Hanamantaraya	28	M	12409	1	156	100	83	262	46	112.6	517	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	III
113	HUCHHAPPA	66	M	13466	10	130	80	127	197	44	96.8	287	Y	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	III
114	DHANABAI	50	M	8568	3	90	60	121	125	22	90	65	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
115	RAMAJI	65	F	8817	2	120	80	119	153	42	92	93	N	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	N
116	GANGAPPA	60	M	13508	3	120	70	131	146	37	81.4	138	N	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	N
117	SABU	55	M	8566	4	118	70	107	206	34	155.8	81	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	N
118	MANAPPA	51	M	6637	5	120	70	103	155	35	100.2	99	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
119	LAXMIBAI	45	M	6615	3	110	70	100	130	30	86.8	66	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
120	BASAVARAJ	40	F	6592	4	120	70	120	165	30	111.4	118	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
121	HANMANTH	65	M	6189	7	160	90	118	176	25	127.4	129	N	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	II
122	SANGAPPA	60	M	5110	2	100	60	100	220	30	165	99	Y	6/24,N6	6/24,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	CAT	II

124	KASHIBAI	60	F	13540	10	120	80	85	120	37	67.2	80	N	6/36,N6	6/36,N6	N	N	N	N	ARCUS	arcus	N	N	RRR	RRR	CAT	CAT	III
125	PULAPPA	72	F	13619	12	180	110	109	210	59	121.6	96	N	6/36,N6	6/36,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	II
126	SANGANNA	50	M	14784	4	90	60	93	260	27	196.6	182	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
127	CHANDRASHEKAR	54	M	14786	3	150	90	119	153	30	81	90	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
128	GOURAWWA	37	M	14876	3	120	70	97	180	42	80	145	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
129	SANGAMESH	75	F	14890	14	150	80	139	320	38	262	100	Y	6/36,N6	6/36,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	N	N	III
130	SHANKAR	42	M	14912	8	140	90	182	187	31	90	330	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
131	SARAWATI	37	M	15969	2	118	70	167	171	32	112	134	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
132	SANGANABASSAPA	65	F	15990	9	120	70	170	225	40	150	198	N	6/24,N6	6/24,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
133	SHARANAPPA	54	M	17711	7	130	90	215	164	40	106	58	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
134	SUMITRA	45	M	17712	2	140	90	109	210	59	121	96	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
135	Mallawwa	68	F	17714	9	120	84	180	132	32	58	209	Y	6/18,N6	6/24,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	I
136	SHANMUKKAPPA	70	F	17828	16	110	70	101	196	59	117	97	N	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
137	BHIMRAYA	65	M	17935	10	120	80	127	180	29	126	133	N	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
138	SHIAVABAI	60	M	18858	1	110	70	120	130	30	80	111	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	CAT	N
139	PAPPANNA	85	F	18914	15	100	70	158	135	38	83	67	N	6/36,N6	6/36,N6	N	N	N	N	arcus	arcus	N	N	-	RRR	IOL	IOL	III
140	ASHOK	45	M	19302	2	130	80	90	197	44	96	281	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
141	SHAKARAPPA	35	M	21544	1	120	80	104	96	19	56	82	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
142	IRANNA	55	M	21780	4	100	60	131	200	59	117	94	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
143	KAZI	56	M	24087	6	130	86	170	192	27	106	187	Y	CF3M	CF3M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
144	BHIMANGOUDA	44	M	24467	1	100	66	163	130	40	70	104	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
145	BASHASAB	55	M	24470	2	100	70	87	92	17	56	82	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
146	MUTANNA	60	M	24914	1	110	80	233	250	45	150	310	Y	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	II
147	MOHAN	50	M	25027	8	160	90	87	197	44	96	281	Y	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
148	NEELABAI	40	M	25427	2	136	90	124	260	27	196	182	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
149	CHIDAMBAR	48	F	25618	2	110	80	182	200	35	135	127	Y	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
150	PARMESH	80	M	25634	30	100	60	95	115	32	67	79	N	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	III
151	MUTTANNA	52	M	25705	3	120	90	110	190	42	123	123	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
152	SHARANAPPA	50	M	25027	1	180	90	190	241	36	166	170	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
153	DADAPATEL	60	M	25744	6	100	60	81	101	20	59	88	N	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
154	ASHOK	85	M	25650	30	150	90	121	141	34	88	93	N	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	III
155	SHUBASH	50	M	25771	2	110	70	141	129	32	85	59	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
156	IRAPPA	57	M	26265	5	110	80	224	236	36	156	187	Y	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	II
157	MAHADEV	55	M	26369	0	126	80	115	178	40	113	121	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
158	DUNDAPPA	55	M	28261	1	90	60	75	135	30	87	87	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
159	MALLAMMA	40	M	28511	1	130	90	83	183	42	123	86	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
160	SHANTABAI	65	F	28891	4	190	100	122	153	42	94	93	N	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	II
161	SURESH	60	F	29065	2	100	60	123	155	42	83	136	N	6/36,N6	6/36,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	IOL	N
162	RATNABAI	32	M	29167	0	114	84	96	137	38	73	130	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
163	NINGARAJ	65	F	29621	14	120	80	107	147	31	87	141	N	6/9,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
164	Kallappa	56	M	29659	5	110	70	96	137	38	73	130	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
165	VISHWANATH	48	M	29756	3	120	70	98	140	29	79.4	158	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
166	SHANKARGOUDA	54	M	29819	2	160	90	144	300	45	180	195	Y	6/9,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
167	VISHWANATH	65	M	29848	7	140	70	117	171	31	114	126	N	6/18,N6	6/9,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	IOL	N
168	KASHIBAI	60	M	29865	7	150	90	135	110	27	63	98	N	6/18,N6	6/18,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT	N
169	ASHOK	60	F	5848	10	130	80	120	130	26	89	71	N	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	I
170	SHIVAMMA	58	M	5856	4	127	80	147	216	39	139.2	457	Y	6/18,N6	6/18,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	III
171	SITAWWA	60	F	13604	6	120	80	112	192	30	138.8	124	Y	6/18,N6	6/18,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	II
172	AYUB	56	F	13785	3	110	70	117	196	59	117	97	N	6/9,N6	6/9,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	I
173	SIDDAPPA	57	M	14397	7	160	100	130	226	34	160	159	Y	6/9,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
174	SHIVAMMA	65	M	13684	10	140	100	85	200	34	120	254	Y	6/6,N6	6/6,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	III
175	SHIVARAM	65	F	13507	8	140	70	116	271	31	164	196	N	6/18,N6	6/18,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	II
176	shrikanth	62	M	13306	6	110	70	130	220	45	109	179	Y	6/36,N6	6/36,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	I
177	nandabasappa	24	M	12480	0	100	70	87	275	30	42	131	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
178	UMADEVI	55	M	5857	2	130	80	104	162	32	108	110	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
179	KALLAPA	58	F	6167	3	111	70	120	156	40	102	66	N	6/9,N6	6/9,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
180	RAM MORE	60	M	6248	5	100	78	180	198	33	136	145	N	CF5M	CF5M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	II
181	VALU PAWAN	65	M	6419	10	130	90	125	193	28	140	121	N	CF5M	CF5M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT	II
182	GUNAWANTHI	65	M	6677	7	110	70	101	150	40	94	76	N	CF4M	CF4M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	III
183	HAIRANBEE	48	F	6867	3	150	90	188	201	45	128	145	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
184	SOMARAYA	58	F	6970	3	100	70	183	150	30	74	231	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	I
185	SHANTABAI	60	M	7139	2	132	90	102	145	35	72.2	220	Y	CF5M	CF5M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	I

187	MADHURABAI	60	M	7233	2	142	110	91	215	34	130	254	Y	6/9,N6	6/9,N6	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	N
188	SITABAI	65	F	7351	3	142	80	139	230	42	168.4	123	Y	CF5M	CF5M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT	N
189	SONABAI	50	F	8038	4	90	64	194	229	41	167.4	103	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
190	RAJASAB	60	F	9278	6	170	110	141	145	31	93	103	N	6/9,N10	6/9,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	I
191	HOTRAPPA	65	M	9585	8	123	60	211	170	25	105	200	Y	CF5M	CF5M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	I
192	GOVIND	62	M	10357	5	130	90	189	264	40	168	176	Y	CF5M	CF5M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	II
193	ABDULLA	62	M	10695	4	150	90	156	165	38	77.4	248	Y	6/9,N10	6/9,N10	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	II
194	ARJUN	57	M	10799	3	130	80	100	166	29	96	204	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
195	Prakash	66	M	10907	6	110	80	123	155	45	94	78	N	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
196	KALLAPPA	26	M	10915	0	140	90	107	165	35	109.4	103	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
197	PAVADAPPA	55	M	11100	6	140	90	219	209	42	23	720	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	III
198	MAHALINGAWWA	57	M	11226	4	120	60	181	145	39	92	69	N	6/9,N10	6/9,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
199	IRRAPPA	62	F	13594	10	120	80	137	180	29	126.4	133	N	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	II
200	CHANDAMMA	43	M	12412	3	118	80	101	134	38	72.2	119	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
201	CHANDRAWWA YETMANI	80	F	10432	15	114	60	104	278	38	197.6	213	Y	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	III
202	LAXMIBAI TUMBAGI	70	F	10177	10	150	92	181	180	58	98.2	159	N	CF3M	CF3M	N	N	N	N	N	N	N	N	RRR	RRR	IOL	CAT	III
203	SHANKARAMMA	65	F	10143	5	160	90	152	231	52	146.6	162	Y	6/9,N10	6/9,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	II
204	TUBIRAM	60	F	7491	4	110	70	100	130	30	86	66	N	6/9,N10	6/9,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
205	RAMESH	60	M	7067	3	160	70	122	153	42.8	94	93	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
206	GURULING	58	M	7004	2	110	70	91	139	36	75	141	N	6/9,N6	6/9,N6	N	N	N	N	N	N	N	N	RRR	RRR	IOL	CAT	N
207	SANKAPPAGOUDA	38	M	20414	5	110	80	121	144	42	88.2	81	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
208	NIJALINGAPPA	62	M	20398	6	156	100	150	312	30	203	190	Y	CF4M	CF4M	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	II
209	MARUTI	60	M	19318	7	126	90	111	195	32	101	280	Y	CF5M	CF5M	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	CAT	CAT	I
210	MD.ALI	68	M	17780	3	120	80	133	153	36	71	225	Y	CF4M	CF4M	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	II
211	VASANTH	72	M	1751	10	140	120	93	200	29	120	144	N	CF3M	CF3M	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	I
212	DEVAKAWWA	69	M	1732	5	142	96	96	210	30	125	211	Y	CF4M	CF4M	N	N	N	N	N	N	N	N	RRR	RRR	IOL	IOL	I
213	SHREEDEVI	60	F	17289	6	110	70	189	250	40	114	176	Y	6/9,N6	6/9,N6	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	CAT	CAT	N
214	SIDDANNA	46	F	17172	2	140	60	104	182	42	108	101	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
215	CHANNAMALLAYA	38	M	16827	2	140	90	106	165	35	26.6	103	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
216	BAPU PATIL	40	M	16812	4	110	70	133	256	40	162.8	166	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
217	PHOOLSING RATHOD	68	M	16678	10	110	80	130	185	45	94	178	Y	CF5M	CF5M	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	II
218	KAMLAWWA	50	M	15380	3	110	70	100	194	40	106	101	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
219	SITABAI	80	F	14186	8	140	110	180	201	35	118	203	Y	CF5M	CF5M	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	N
220	MAHANTAMMA	75	F	14606	5	140	60	130	150	30	85	98	N	6/60,N10	6/60,N10	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	N
221	MUKTABAI	45	F	14589	2	130	90	139	150	31	86.2	91	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	N	N	N	N	N
222	MALLANGOUDA	76	F	14130	5	150	90	176	200	28	172	101	N	CF5M	CF5M	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	II
223	DUNDAPPA	60	M	13076	3	120	80	116	150	39	79	165	N	6/36,N10	6/36,N10	N	N	N	N	ARCUS	ARCUS	N	N	RRR	RRR	IOL	IOL	I
224	SHRIKANTH	60	M	14413	5	150	100	168	248	30	176	210	Y	6/36,N10	6/18,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
225	MAHADEVI	55	M	14533	3	90	60	148	209	39	158	59	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
226	BHARATI	60	F	14553	8	120	90	108	229	41	166	108	Y	6/36,N10	6/36,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
227	SONABAI	36	F	11252	0	110	80	106	200	31	151	128	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
228	MADUBABU	72	F	15883	8	120	70	150	163	32	84	159	Y	6/24,N10	6/24,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	I
229	NEELAWWA	50	M	10098	4	120	90	153	122	38	71	62	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
230	SAJABAI	96	F	9815	15	150	60	177	200	40	120	180	Y	CF3M	CF3M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	II
231	SURESH	65	F	9732	5	140	70	120	150	30	80	121	N	6/36,N10	6/36,N10	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	I
232	SUMANTH	30	M	9180	2	90	60	121	145	32	23	75	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
233	SHIVANAND	70	M	8968	8	120	90	96	225	30	165	150	N	6/36,N10	6/36,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	I
234	GURURAJ	80	M	8480	11	120	70	97	170	32	80.4	145	N	6/24,N10	6/24,N10	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	II
235	SHANTAPPA	65	M	8121	9	150	90	127	253	42	132	193	Y	6/36,N10	6/36,N10	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT	II
236	RANGAPPA	56	M	15902	4	130	90	111	149	32	85	87	N	6/24,N10	6/24,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
237	KRISHNA	75	M	15944	7	150	80	85	220	38	162	98	Y	CF3M	CF3M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	N
238	SHANKAR	51	M	15685	4	110	80	135	135	30	82	111	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
239	GANGABAI	37	M	15969	1	120	80	101	96	19	56	182	Y	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
240	SIDDANAGOUDA	80	F	17189	6	150	100	167	191	37	120	138	N	CF3M	CF3M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	N
241	ADAMANH	36	M	17346	2	140	90	125	200	36	116	148	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N
242	LAXMIBAI	65	M	17615	6	150	100	127	262	46	112	517	Y	CF3M	CF3M	N	N	N	N	arcus	arcus	N	N	RRR	RRR	CAT	CAT	III
243	BASSAMMA	70	F	18413	10	120	80	131	180	37	87	130	N	6/36,N10	6/36,N10	N	N	N	N	arcus	arcus	N	N	RRR	RRR	IOL	IOL	I
244	NINGAWWA	65	F	18578	6	140	80	140	149	35	90	135	N	6/36,N10	6/36,N10	N	N	N	N	N	N	N	N	RRR	RRR	CAT	CAT	N
245	RAMANAGOUDA	55	M	17564	4	150	90	123	150	32	66	150	N	6/6,N6	6/6,N6	N	N	N	N	N	N	N	N	RRR	RRR	N	N	N