

**A STUDY ON  
PREVALENCE OF METABOLIC SYNDROME AMONG  
HYPERTENSIVE ADULTS (20-45 YEARS) AND THEIR  
COMPLIANCE TO TREATMENT IN A TERTIARY CARE  
HOSPITAL, CHENNAI – A HOSPITAL BASED MIXED METHOD  
STUDY**

*Dissertation submitted to*

**THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY  
CHENNAI, TAMIL NADU.**

*In partial fulfillment of the regulations  
for the award of the degree of*

**M.D. GENERAL MEDICINE**

**BRANCH -I**

*Register number -200120101029*



**DEPARTMENT OF GENERAL MEDICINE  
GOVERNMENT STANLEY MEDICAL COLLEGE  
CHENNAI  
MAY 2023**

## CERTIFICATE BY THE INSTITUTION

This is to certify that this dissertation entitled "A STUDY ON PREVALENCE OF METABOLIC SYNDROME AMONG HYPERTENSIVE ADULTS (20-45 YEARS) AND THEIR COMPLIANCE TO TREATMENT IN A TERTIARY CARE HOSPITAL, CHENNAI – A HOSPITAL BASED MIXED METHOD STUDY" is a bonafide work done by Dr.A.R.SREE BALAJI, post graduate student, Department of General Medicine, Stanley Medical College & Hospital, Chennai - 600001, Registration number: 200120101029 in partial fulfillment of the requirement for award of degree of M.D General Medicine (Branch-I), carried out by him under direct supervision and guidance, during the academic year 2020 – 2023.

*S.M. Sujatha*  
**Dr. S.M. SUJATHA, M.D.,**  
GUIDE: 57062  
Professor of Medicine  
Stanley Medical College  
**Dr. S.M. SUJATHA M.D.**  
Unit Chief,  
Department of General Medicine,  
Stanley Medical College & Hospital,  
Chennai - 600001.

*S. Parimalasundari*  
**Prof. Dr. S. PARIMALASUNDARI M.D.,**  
Professor and head,  
Department of Medicine,  
Govt. Stanley Medical College & Hospital,  
Chennai - 600 001.  
Head of the Department,  
Department of General Medicine,  
Stanley Medical College & Hospital,  
Chennai - 600001.

*P. Balaji*  
**Prof. Dr. P. BALAJI M.S., FRCS., Ph.D., FCLS.,**  
DEAN  
Dean  
STANLEY MEDICAL COLLEGE  
CHENNAI 600 001  
Government Stanley Medical College and Hospital,  
Chennai.

## CERTIFICATE BY THE GUIDE



This is to certify that this dissertation work titled “A STUDY ON PREVALENCE OF METABOLIC SYNDROME AMONG HYPERTENSIVE ADULTS (20-45 YEARS) AND THEIR COMPLIANCE TO TREATMENT IN A TERTIARY CARE HOSPITAL, CHENNAI – A HOSPITAL BASED MIXED METHOD STUDY” is done by the post graduate student Dr.A.R.SREE BALAJI with Registration Number 200120101029 for the award of M.D. DEGREE in the branch of GENERAL MEDICINE (BRANCH-I), for the academic year 2020 - 2023. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains, from introduction to conclusion pages and result, shows 7% (SEVEN PERCENTAGE) of plagiarism in the dissertation.

*S.M. Sujatha*  
Dr. S.M. SUJATHA, M.D.,  
Guide & Supervisor signature  
Reg. No: 57062  
Professor of Medicine  
Stanley Medical College  
Prof. Dr. S.M. SUJATHA M.D.,  
Unit Chief,  
Department of General Medicine,  
Stanley Medical College & Hospital,  
Chennai.

## Document Information

<b>Analyzed document</b>	SREEBALAJI THESIS pdf 1.pdf (D152843677)
<b>Submitted</b>	12/9/2022 7:57:00 PM
<b>Submitted by</b>	SREE BALAJI A R
<b>Submitter email</b>	sreebalaji3369@gmail.com
<b>Similarity</b>	7%
<b>Analysis address</b>	sreebalaji3369.tnmg@analysis.arkund.com

## Sources included in the report

<b>W</b>	URL: <a href="http://104.36.64.144/news/shownews.php?id=36">http://104.36.64.144/news/shownews.php?id=36</a> Fetched: 3/8/2022 5:05:44 AM	 <b>23</b>
<b>W</b>	URL: <a href="https://journals.lww.com/jhypertension/Fulltext/2018/10000/2018_ESC_ESH_Guidelines_for_the_man...">https://journals.lww.com/jhypertension/Fulltext/2018/10000/2018_ESC_ESH_Guidelines_for_the_man...</a> Fetched: 10/31/2020 1:27:38 AM	 <b>2</b>
<b>W</b>	URL: <a href="https://academic.oup.com/eurheartj/article/39/33/3021/5079119">https://academic.oup.com/eurheartj/article/39/33/3021/5079119</a> Fetched: 10/31/2019 11:22:05 PM	 <b>15</b>

## Entire Document

11 Introduction

12 INTRODUCTION Hypertension is the dreaded silent killer in world. In this industrialised world stress is invariably present in all parts of world across ages. Combined with obesity and insulin resistance, hypertension accelerates the endotheliopathy leading to various micro and macro vascular complications. This prepones the incidence of various morbidities, thus reducing the productivity of the affected and eventually leading to mortality. This drastically affects the economy and morale of the family. Thus early detection of hypertension and the constellation of factors contributing towards the metabolic syndrome is important in preventing life threatening vascular complications. It seen that even though these conditions are diagnosed at the health care facility level the compliance to treatment is poor. Our study aims at screening of hypertensive patients for metabolic syndrome and identifying the reasons for non compliane in their treatment. Once identified, the reasons for non compliance and the solutions to rectify them can be applied in our day to day practice, thus improving the effective delivery of health interventions to the last mile.

13 Aim & Objectives

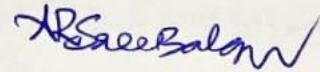
14 AIM AND OBJECTIVES AIM : Aim of the study is to assess the prevalence of metabolic syndrome among hypertensive adults (20-45 years) and their compliance to treatment in a tertiary care hospital, Chennai PRIMARY OBJECTIVE : To study the prevalence of metabolic syndrome in hypertensive adults of age between 20 and 45 years SECONDARY OBJECTIVE: 1. To study the compliance to drugs and lifestyle modifications in hypertensive patients with and without metabolic syndrome 2. To explore the reasons for poor compliance to treatment (drugs and lifestyle modifications ) among patients with metabolic syndrome

15 Review of Literature

16 16 REVIEW OF LITERATURE DEFINITION AND DETERMINANTS Blood pressure is defined as the "Lateral pressure exerted by the column of blood on the vessel wall measured in mmHg Blood Pressure = Cardiac Output X Peripheral Vascular Resistance(PVR) = Stroke Volume X Heart Rate(HR) X PVR =(End Diastolic Volume–End Systolic Volume)X HR X PVR Therefore, blood pressure is determined by 1. Preload (EDV) – volume of the blood in ventricle at the end of diastole 2. Afterload (PVR) – resistance against which the ventricle has to pump blood 3. Myocardial contractility 4. Venous return TECHNIQUES TO MEASURE BLOOD PRESSURE Various non invasive methods to measure blood pressure are 1.Palpatory method 2.Auscultatory method 3.Oscillometric method The 'SPRINT' trial has demonstrated that oscillometric method of measuring blood pressure using Omron 907 oscillometric BP apparatus is more accurate and reproducible among all the non invasive methods. It is more reflective of the

## DECLARATION

I solemnly declare that the dissertation titled “**A STUDY ON PREVALENCE OF METABOLIC SYNDROME AMONG HYPERTENSIVE ADULTS (20-45) YEARS AND THEIR COMPLIANCE TO TREATMENT IN A TERTIARY CARE HOSPITAL, CHENNAI – A HOSPITAL BASED MIXED METHOD STUDY**” is a bonafide work done by me at Government Stanley Hospital, Chennai between April 2021 and March 2022 under the guidance and supervision of **Prof. Dr. S.M.SUJATHA, M.D.** I also declare that this bonafide work or a part of this work was not submitted by me or any other for any reward, degree or diploma to any other university or board either in India or abroad. This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree in General Medicine (Branch – I).



Dr.A.R.SREE BALAJI

M.D General Medicine Post Graduate

Government Stanley Medical College

Register Number :200120101029

Chennai – 600001.

Date: 15/12/2022

Place: Chennai

## **SPECIAL ACKNOWLEDGEMENT**

I gratefully acknowledge and thank

Prof. Dr. P.BALAJI M.S.,FRCS.,Ph.D.,FCLS.,

Dean

Government Stanley Medical College and Hospital, Chennai

for granting me permission to utilize the resources of this institution for my study

## ACKNOWLEDGEMENT

My sincere thanks to **Prof. Dr. S. PARIMALASUNDARI M.D**, Professor and Head of Department of General Medicine, Stanley Medical College and Hospital, for her vital guidance and support for the successful completion of my dissertation.

I am extremely grateful to **Prof. Dr.S.CHANDRASEKAR, M.D.**, Professor of Department of General Medicine, Government Stanley Medical College and Hospital for his guidance and motivation throughout my postgraduate period.

I am extremely grateful to **Prof. Dr.S.M.SUJATHA, M.D.**, Professor and our Unit Chief, Department of General Medicine, Government Stanley Medical College and Hospital for her full-fledged support and ward classes throughout my postgraduate period. Her valuable suggestions and guidance were pivotal during my study period.

I am extremely grateful and fondly remember **Prof.Dr.MOHAMMED KALIFA, M.D.**, Professor, Department of General Medicine, Government Stanley Medical College and Hospital, whose association with our unit was a memory to cherish, although it was a brief one. The Almighty keeps the Good near to himself.

My special thanks to my beloved unit Assistant professors **Dr.R.SIVARAMAN,M.D., Dr.A.R.BALAMURUGAN,M.D., and Dr.R.NAVEEN KUMAR, M.D.**, for their valuable support throughout the study. It is their moral support which helped us to tide over the turbulent times.

My sincere gratitude to the patients and their attenders for the cooperation aiding in the successful conduct of my study.

A special thanks to all my friends for their help.

## ETHICAL COMMITTEE APPROVAL



### GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL, CHENNAI -01 INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK : "A STUDY ON PREVALENCE OF METABOLIC SYNDROME AMONG HYPERTENSIVE ADULTS (20-45 YEARS) AND THEIR COMPLIANCE TO TREATMENT IN A TERTIARY CARE HOSPITAL, CHENNAI A HOSPITAL BASED MIXED METHOD STUDY"

PRINCIPAL INVESTIGATOR : DR. A.R. SREE BALAJI,  
DESIGNATION : PG IN GENERAL MEDICINE,  
DEPARTMENT : DEPARTMENT OF GENERAL MEDICINE

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.03.2021 at the Council Hall, Stanley Medical College, Chennai-1 at 11 am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

  
MEMBER SECRETARY,  
IEC, SMC, CHENNAI.



## ABBREVIATIONS

<b>ABI</b>	<b>Ankle–brachial index</b>
<b>ABPM</b>	<b>Ambulatory blood pressure monitoring</b>
<b>ACE</b>	<b>Angiotensin-converting enzyme</b>
<b>ACEi</b>	<b>Angiotensin-converting enzyme inhibitor</b>
<b>ACR</b>	<b>Albumin:creatinine ratio</b>
<b>CKD</b>	<b>Chronic kidney disease</b>
<b>CK-MB</b>	<b>Creatinine kinase-muscle/brain</b>
<b>COPD</b>	<b>Chronic obstructive pulmonary disease</b>
<b>DBP</b>	<b>Diastolic blood pressure</b>
<b>eGFR</b>	<b>Estimated glomerular filtration rate</b>
<b>ESC</b>	<b>European Society of Cardiology</b>
<b>ESH</b>	<b>European Society of Hypertension</b>
<b>HbA1c</b>	<b>Haemoglobin A1c</b>
<b>HBPM</b>	<b>Home blood pressure monitoring</b>
<b>HDL-C</b>	<b>HDL cholesterol</b>
<b>HELLP</b>	<b>Haemolysis, elevated liver enzymes and low platelets</b>
<b>HFpEF</b>	<b>Heart failure with preserved ejection fraction</b>
<b>HFrEF</b>	<b>Heart failure with reduced ejection fraction</b>
<b>HMOD</b>	<b>Hypertension mediated organ damage</b>
<b>LVH</b>	<b>Left ventricular hypertrophy</b>

<b>MAP</b>	<b>Mean arterial pressure</b>
<b>MI</b>	<b>Myocardial infarction</b>
<b>MRI</b>	<b>Magnetic resonance imaging</b>
<b>NT-proBNP</b>	<b>N-terminal pro-B natriuretic peptide</b>
<b>o.d.</b>	<b>Omni die (every day)</b>
<b>PAD</b>	<b>Peripheral artery disease</b>
<b>PRA</b>	<b>Plasma renin activity</b>
<b>PWV</b>	<b>Pulse wave velocity</b>
<b>RAS</b>	<b>Renin–angiotensin system</b>
<b>RCT</b>	<b>Randomized controlled trial</b>
<b>SPC</b>	<b>Single-pill combination</b>
<b>SPRINT</b>	<b>Systolic Blood Pressure Intervention Trial</b>
<b>TIA</b>	<b>Transient ischaemic attack</b>
<b>VEGF</b>	<b>Vascular endothelial growth factor</b>

**PREVALENCE OF METABOLIC SYNDROME AMONG  
HYPERTENSIVE ADULTS (20-45 YEARS) AND THEIR  
COMPLIANCE TO TREATMENT IN A TERTIARY CARE  
HOSPITAL, CHENNAI – A HOSPITAL BASED MIXED METHOD  
STUDY**

**ABSTRACT**

**BACKGROUND:**

Metabolic syndrome is the term given to address a collection of risk factors. These risk factors are considered to increase risk for diabetes mellitus, coronary artery disease and cerebrovascular accident. Low patient compliance has been one of the greatest challenges for success in the treatment as isolate entities. In practice, the treatment of metabolic syndrome implies the sum of difficulties usually found in monitoring each one of its components. This study was conducted to find the prevalence of metabolic syndrome in young hypertensive individuals, their compliance to treatment and reasons for poor compliance.

**METHODS:**

The study is a sequential explanatory type of mixed methods study. Quantitative component will be followed by the qualitative research method. This particular type was chosen as the study findings of the quantitative method will be explained by the in-depth interviews administered to the part of purposively selected study participants.

- i) Quantitative component of it will be a cross sectional study that addresses the first and second objectives of the study.
- ii) Descriptive qualitative study using in-depth interview will be done to study the third objective.

## RESULTS:

62.8% of the study participants are found to have metabolic syndrome with more female preponderance. 33% of the study participants were newly diagnosed with impaired glucose tolerance or diabetes mellitus. Patient related factors were predominant reasons for non compliance. Involving a family member in treatment plan along with combination pill therapy greatly improves the compliance.

## CONCLUSION:

Contrary to our belief, prevalence of metabolic syndrome in hypertensive adult population is very high. This condition go highly unnoticed until a major thrombotic event occurs. A multimodal approach involving the planning at government level, healthcare provider level along with patient education and involvement of the family in treating these chronic illness proves to be a great success in greatly improving the health and financial aspects of the client's life.

## KEYWORDS:

Metabolic Syndrome, Hypertension.

## TABLE OF CONTENTS

<b>SERIAL NUMBER</b>	<b>TOPIC</b>	<b>PAGE NUMBER</b>
1	Introduction	12
2	Aims & Objectives	14
3	Review of Literature	16
4	Methodology	67
5	Observation and Results	74
6	Discussion	108
7	Conclusion	112
8	Limitations	114
9	Bibliography	116
	<b>ANNEXURES</b>	
	Informed Consent	134
	Proforma	137
	Master chart	145

# *Introduction*

## **INTRODUCTION**

Hypertension is the dreaded silent killer in world. In this industrialised world stress is invariably present in all parts of world across ages. Combined with obesity and insulin resistance, hypertension accelerates the endotheliopathy leading to various micro and macro vascular complications. This prepones the incidence of various morbidities, thus reducing the productivity of the affected and eventually leading to mortality. This drastically affects the economy and morale of the family. Thus early detection of hypertension and the constellation of factors contributing towards the metabolic syndrome is important in preventing life threatening vascular complications. It seen that even though these conditions are diagnosed at the health care facility level the compliance to treatment is poor.

Our study aims at screening of hypertensive patients for metabolic syndrome and identifying the reasons for non compliane in their treatment. Once identified, the reasons for non compliance and the solutions to rectify them can be applied in our day to day practice, thus improving the effective delivery of health interventions to the last mile.

# *Aim & Objectives*



## **AIM AND OBJECTIVES**

### **AIM :**

Aim of the study is to assess the prevalence of metabolic syndrome among hypertensive adults (20-45 years) and their compliance to treatment in a tertiary care hospital, Chennai

### **PRIMARY OBJECTIVE :**

To study the prevalence of metabolic syndrome in hypertensive adults of age between 20 and 45 years

### **SECONDARY OBJECTIVE:**

1. To study the compliance to drugs and lifestyle modifications in hypertensive patients with and without metabolic syndrome
2. To explore the reasons for poor compliance to treatment (drugs and lifestyle modifications ) among patients with metabolic syndrome

# *Review of Literature*

## **REVIEW OF LITERATURE**

### **DEFINITION AND DETERMINANTS**

Blood pressure is defined as the “Lateral pressure exerted by the column of blood on the vessel wall measured in mmHg

$$\begin{aligned}\text{Blood Pressure} &= \text{Cardiac Output} \times \text{Peripheral Vascular Resistance(PVR)} \\ &= \text{Stroke Volume} \times \text{Heart Rate(HR)} \times \text{PVR} \\ &= (\text{End Diastolic Volume} - \text{End Systolic Volume}) \times \text{HR} \times \text{PVR}\end{aligned}$$

Therefore, blood pressure is determined by

1. Preload (EDV) – volume of the blood in ventricle at the end of diastole
2. Afterload (PVR) – resistance against which the ventricle has to pump blood
3. Myocardial contractility
4. Venous return

### **TECHNIQUES TO MEASURE BLOOD PRESSURE**

Various non invasive methods to measure blood pressure are

1. Palpatory method
2. Auscultatory method
3. Oscillometric method

The ‘SPRINT’ trial has demonstrated that oscillometric method of measuring blood pressure using Omron 907 oscillometric BP apparatus is more accurate and reproducible among all the non invasive methods. It is more reflective of the

Mean Blood Pressure than the systolic or diastolic blood pressure.

The PALAPTORY method avoids the pitfall of missing the auscultatory gap. But the disadvantages are 1. Only Systolic BP can be measure

2. The SBP measured by this method approximately 4-6mmHg lower than the actual blood pressure. It assumes that as the cuff is deflated, the initial flow of blood itself is sufficient to generate pulse wave to be detected by the fingers. But in real world scenario, it takes further rush of blood volume into the arteries to generate pulse wave and definite pulsations may not be felt by the fingers until the cuff pressure has been reduced by >4-6mmHg from the actual SBP

The AUSCULTATORY method of BP measuring was initially described by Korotkoff in 1905. It is based on the fact that there are no sounds heard over the normal brachial artery if we apply stethoscope on it. If the apparatus cuff pressure is raised above the systolic pressure and gradually deflated, a series of sounds are heard on the artery distal to the cuff and BP can be measured based on the interpretation of the sounds. These sounds are due to turbulent blood flow in the artery. These sounds are known the Korotkoff sounds and are described to have 5 phases.

Phase 1 : Clear sharp tapping sound heard as the blood initially rushes through the previously occluded artery. At cuff pressure immedietly below the systolic blood pressure, flow through the artery occurs only at the peak of the systole. This intermittent turbulence produces the clear sharp tapping sound.

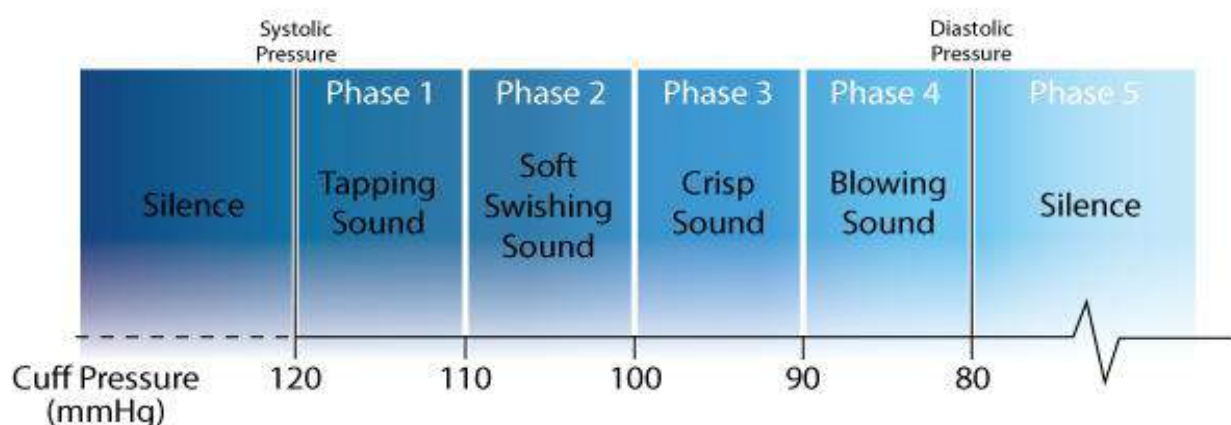
Phase 2 : Murmurish sound heard and remain the same for next 10-15mmHg

Phase 3 : It starts with a clear knocking or banging sound that continues for the next 12 to 14 mmHg

Phase 4: The transition from phase 3 to phase 4 is usually very sudden. The sounds remain muffled, faint and indistinct. This phase lasts for about 4-6mmHg.

As long as the cuff pressure is above the diastolic pressure, the flow inside the artery is turbulent making sounds.

Phase 5: This phase begins when the Korotkoff sounds begin to disappear completely. The silence will continue upto 0 level. Usually this phase marks the diastolic pressure. In children, adolescents, adults after exercise and in others with hyperdynamic circulatory states phase 4 corresponds to the diastolic blood pressure.



**AUSCULTATORY GAP:** In some patients with hypertension there may be a pause in Korotkoff sounds. As the cuff pressure is lowered initially a few faint sounds are heard and then disappear soon. After a pause i.e., a brief period of silence these sounds start to reappear. This period of silence may last for 40-60mmHg. This may lead to a false low systolic bp or a false high diastolic bp. To avoid this misinterpretation of false low systolic blood pressure, initially palpatory method should be performed to estimate the systolic blood pressure followed by auscultation method. To avoid the misinterpretation of false high diastolic blood pressure, we should continue hearing for sounds until the cuff pressure is lowered till zero mark.

In OSCILLOTORY method, systolic blood pressure was measured palpatory method while the diastolic pressure was measured from the oscillations of the mercury column. After inflating the cuff above systolic pressure, it should be deflated progressively. The oscillations will appear at one point, reach the maximum and eventually start to disappear. The midpoint of maximum oscillations or the lower level of these oscillations can be considered as diastolic blood pressure.

In the two cuff oscillometric method, one cuff is placed on the upper arm and the another one is placed slightly inflated below. As the pressure in upper cuff is raised and then lowered, pulsations can be recorded from the lower cuff.

## GRADING OF HYPERTENSION<sup>1</sup>

<b>Category</b>	<b>SBP (mmHg)</b>		<b>DBP (mmHg)</b>
Office BP <sup>a</sup>	≥140	and/or	≥90
Ambulatory BP			
Daytime (or awake) mean	≥135	and/or	≥85
Night-time (or asleep) mean	≥120	and/or	≥70
24 h mean	≥130	and/or	≥80
Home BP mean	≥135	and/or	≥85

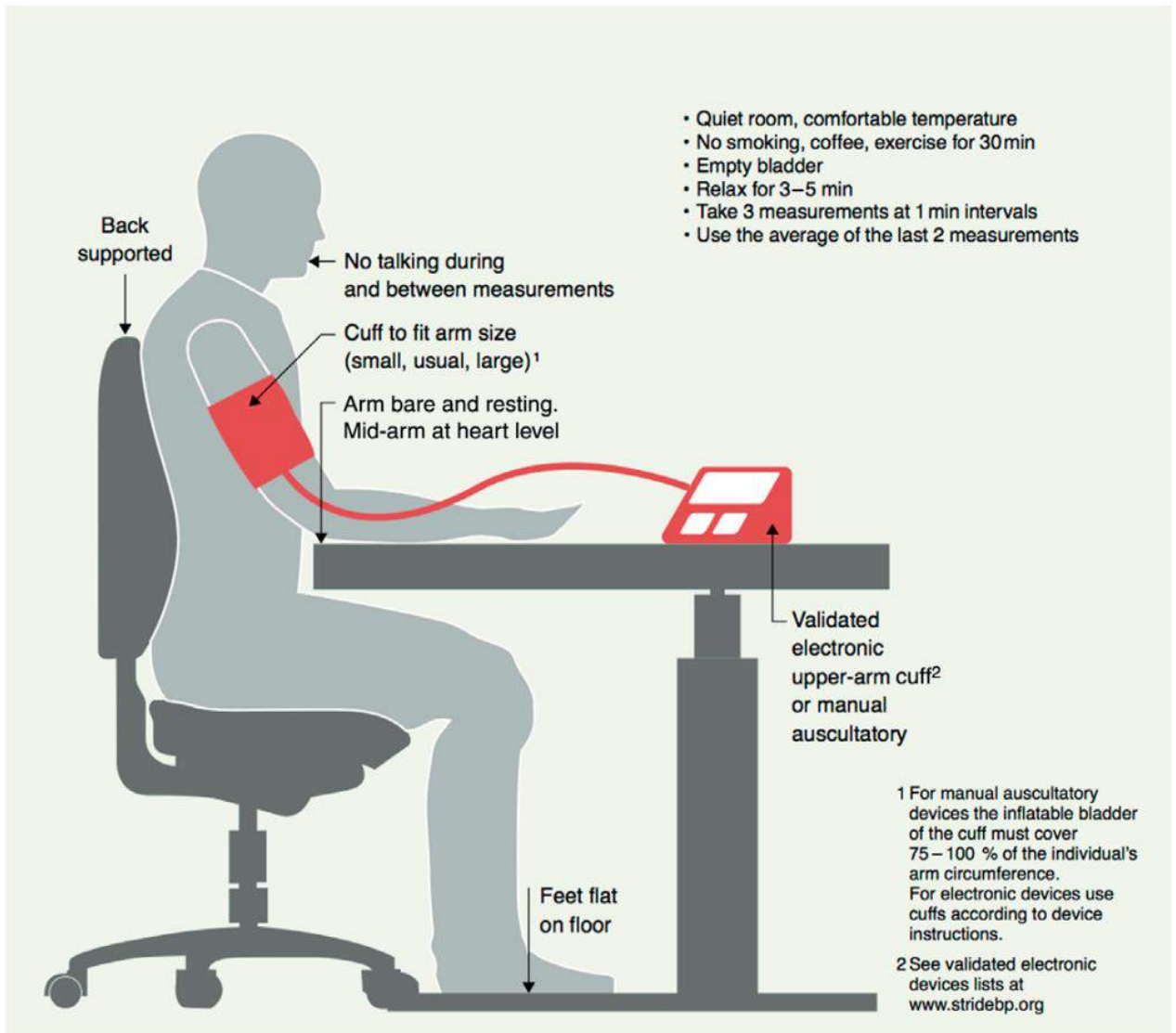
### **VARIOUS METHODS OF BLOOD PRESSURE MEASUREMENT:**

The various methods of blood pressure measurement are

- a) Conventional office blood pressure measurement
- b) Unattended office blood pressure measurement
- c) Out of office blood pressure measurement
- d) Home blood pressure monitoring
- e) Ambulatory blood pressure monitoring

Conventional office blood pressure measurement:

It is the measuring of blood pressure by a health care worker in his office and it is the most followed procedure worldwide.



Precautions to take in Office BP measurement:

- i. Room should be quiet with comfortable temperature
- ii. Smoking, Caffeine intake and Exercise should be avoided 30minutes before blood pressure measurement.
- iii. Bladder should be emptied



- iv. Patient should be seated on a chair with back support, legs uncrossed and feet flat on floor. Arms to rest on table with midarm at the heart level.
- v. Relax for 3-5 minutes before BP measurement.
- vi. Inflatable rubber bladder should cover 75-100% of the arm circumference.
- vii. In each visit three BP measurements should be made 1-2 minutes apart. If the first two measurements differ by  $>10\text{mmHg}$ , additional BP measurements are to be made. BP is taken as the average of the last two measure BP values.
- viii. For patients with unstable BP measurements, like those in Arrhythmia, multiple recordings may be needed. Also for them Manual BP measurement is preferred. Always Heart rate and Pulse should me measured to exclude any arrhythmias.
- ix. When using Auscultatory method, Phase 1 and Phase 5 of Korotkoff,s sounds are considered as Systolic and Diastolic BP respectively.
- x. BP is to be measured in both the arms in first visit. The arm with the higher value is recorded and is considered for subsequent measurements as reference.<sup>2</sup>

### **WHITECOAT HYPERTENSION:**

It is a condition in which Blood pressure is found elevated in the office but with ABPM or HBPM it is within normal limits. White coat hypertension is prevalent in around 30-40% of people. It is commonly found in elderly age, women, nonsmokers. It is

seen in all grades of hypertension including Resistant hypertension. It is more prevalent among Grade 1 hypertension. HMOD is less prevalent among white coat hypertensives<sup>4,5</sup>. Compared with normotensives these people have increased adrenergic activity<sup>3</sup>, increased risk of metabolic syndrome, frequent asymptomatic cardiovascular damage, increased risk of diabetes onset and increased risk of isolated systolic hypertension. This white coat hypertension is not innocent as it appears.

### **MASKED HYPERTENSION:**

Masked hypertension refers to the condition in which Office BP measurement is normal but ABPM or HBPM is higher i.e., a condition in which the Hypertension is masked on Office blood pressure measurement. It is prevalent in around 15% of the population<sup>6</sup>. The prevalence is greater in younger people, smokers, male sex, alcohol consumption, anxiety, job stress, higher physical activity, job stress, obesity, diabetes, family history of hypertension and CKD. These people tend to present with high normal BP range.

### **UNATTENDED OFFICE BLOOD PRESSURE MEASUREMENT:**

Usage of automated BP apparatus has resulted in more reproducible Blood pressure recordings and they have the advantage that BP can be measured without the participation of health care workers. In the unattended office blood pressure measurement, the patients are seated alone and unobserved. The recorded BP is found to reduce the “white coat” effect.<sup>7,8</sup>

## **HOME BLOOD PRESSURE MONITORING:**

Home Blood pressure monitoring employs the semiautomatic BP apparatus for BP measurement in home. Precautions mentioned for office BP measurement are to be taken for Home BP measurement also. BP measurement is to be done at morning and evening, atleast for 3 days, preferably for 6 to 7 consecutive days before hospital visit. Average values of the above recordings is taken.<sup>9</sup> Patient's self monitoring has dramatic improvement in drug compliance. Telemonitoring and smartphone applications combined with patient education and counselling have improved the treatment quality.

## **AMBULATORY BLOOD PRESSURE MONITORING:**

Ambulatory blood pressure monitoring provides average of BP values over a defined period which is usually 24 hours. The device is worn by the client on his arm over 24 hours and the device is programmed to record BP at 15 – 30 min intervals. Also the client's activity and sleep time are recorded manually. A minimum of 70% usable BP recordings are required for the valid ambulatory blood pressure measurement. ABPM values are lower than office BP values and they are a better predictor of Hypertension mediated organ damage than that of office BP.<sup>10-18</sup>

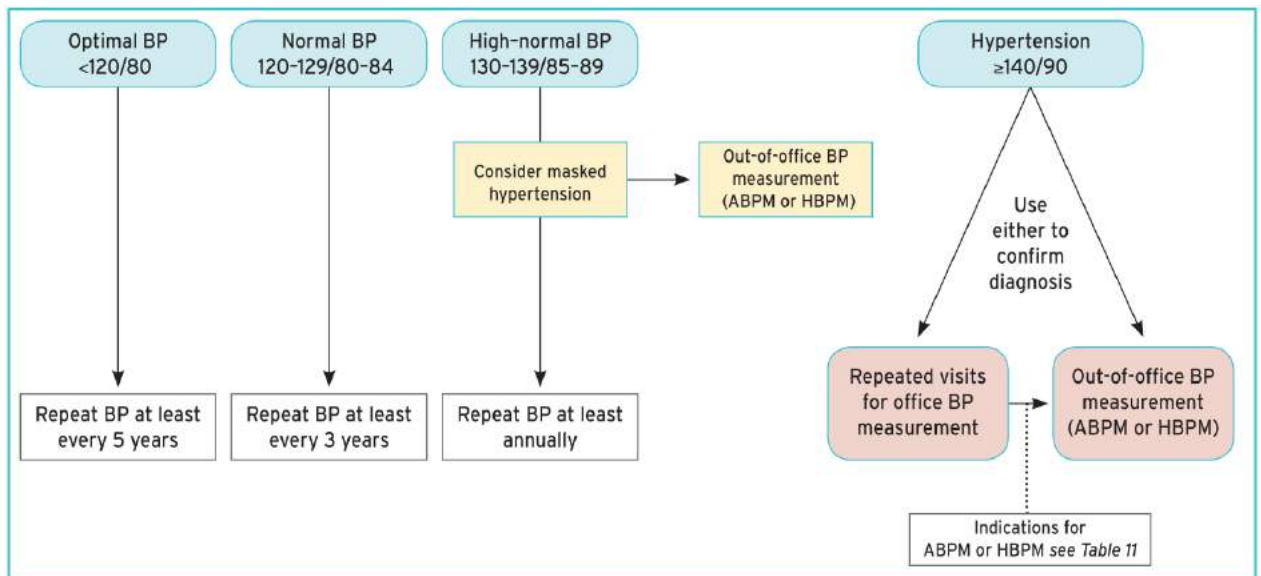
This ABPM has an unique advantage of measuring blood pressure in sleep. Usually 10% of blood pressure dips during the sleep. Reduced or absent nocturnal dipping is seen in sleep disturbances, obesity, OSAS, high salt intake, autonomic dysfunction, orthostatic hypotension, diabetic neuropathy, CKD and old age<sup>19</sup>. Nocturnal BP is better predictor of the outcome than the day time BP. Also it is found that in patients with extremes of nocturnal dipping there is increased risk of morbidity.

With ABPM additional indices with prognostic values such as 24hr BP variability, ambulatory arterial stiffness index and morning BP surge are obtained. The extent of their

validity is to be studied in detail.

ABPM	HBPM
<p><b>Advantages</b></p> <ul style="list-style-type: none"> <li>● Can identify white-coat and masked hypertension</li> <li>● Stronger prognostic evidence</li> <li>● Night-time readings</li> <li>● Measurement in real-life settings</li> <li>● Additional prognostic BP phenotypes</li> <li>● Abundant information from a single measurement session, including short-term BP variability</li> </ul>	<p><b>Advantages</b></p> <ul style="list-style-type: none"> <li>● Can identify white-coat and masked hypertension</li> <li>● Cheap and widely available</li> <li>● Measurement in a home setting, which may be more relaxed than the doctor's office</li> <li>● Patient engagement in BP measurement</li> <li>● Easily repeated and used over longer periods to assess day-to-day BP variability</li> </ul>
<p><b>Disadvantages</b></p> <ul style="list-style-type: none"> <li>● Expensive and sometimes limited availability</li> <li>● Can be uncomfortable</li> </ul>	<p><b>Disadvantages</b></p> <ul style="list-style-type: none"> <li>● Only static BP is available</li> <li>● Potential for measurement error</li> <li>● No nocturnal readings<sup>a</sup></li> </ul>

### SCREENING FOR HYPERTENSION:



### CONFIRMING THE DIAGNOSIS OF HYPERTENSION:

Unless the blood pressure is significantly elevated to Grade 3 or there is undeniable proof of HMOD (such as LVH, vascular damage, hypertensive retinopathy with exudates and haemorrhages, etc.), the diagnosis of hypertension should not be made simply on a

single set of blood pressure measurements from a single office visit. Repeat blood pressure readings at subsequent office visits have long been used by all other clinicians in nearly all patients to confirm a BP increase that persists as well as to classify their hypertension status in clinical practice and RCTs. The number of visits and the duration between visits vary depending on how severe the hypertension is and these two factors are inversely associated. Thus, depending on the degree of BP elevation and whether it is grade 2 or above, less frequent visits and shorter intervals between visits (i.e. a few days or weeks) are necessary if there is proof of HMOD or CVD<sup>20</sup>. In contrast, repeat measurements may be taken in a few months in patients with BP rise in the grade 1 range, particularly if the patient is low risk and there is no HMOD. Simultaneously during this time of BP monitoring, standard screening tests, CV risk assessment and other procedures are often carried out.

## **INDICATIONS FOR HOME BLOOD PRESSURE MONITORING AND AMBULATORY BLOOD PRESSURE MONITORING:<sup>1</sup>**

<p>Conditions in which white-coat hypertension is more common, e.g.:</p> <ul style="list-style-type: none"> <li>● Grade I hypertension on office BP measurement</li> <li>● Marked office BP elevation without HMOD</li> </ul>
<p>Conditions in which masked hypertension is more common, e.g.:</p> <ul style="list-style-type: none"> <li>● High-normal office BP</li> <li>● Normal office BP in individuals with HMOD or at high total CV risk</li> </ul>
<p>Postural and post-prandial hypotension in untreated and treated patients</p>
<p>Evaluation of resistant hypertension            Evaluation of BP control, especially in treated higher-risk patients            Exaggerated BP response to exercise</p>
<p>When there is considerable variability in the office BP</p>
<p>Evaluating symptoms consistent with hypotension during treatment</p>
<p>Specific indications for ABPM rather than HBPM:</p> <ul style="list-style-type: none"> <li>● Assessment of nocturnal BP values and dipping status (e.g. suspicion of nocturnal hypertension, such as in sleep apnoea, CKD, diabetes, endocrine hypertension, or autonomic dysfunction)</li> </ul>

## FACTORS CAUSING HYPERTENSION;

Hypertension is classified as a) Primary or Essential Hypertension

b) Secondary Hypertension

## PRIMARY HYPERTENSION:

Primary hypertension has both Environmental and Genetic factors contributing to it.

### Environmental Factors:

- 1) Obesity – weight gain is the strongest and independent risk factor. This is mostly a part of Metabolic syndrome.
- 2) Diet high in Sodium
- 3) Diet low in Potassium, Calcium, Magnesium.
- 4) Stress
- 5) Alcohol consumption
- 6) Low vitamin D
- 7) High Fructose diet
- 8) Lack of exercise

### GENETIC FACTORS:

- 1) Either parent having hypertension : 10-15%
- 2) Both parents having hypertension : 30-40%
- 3) Alpha – adducin gene

## PATHOGENESIS<sup>21</sup>

The above said environmental factors in a genetically predisposed individuals are responsible for the development of hypertension.

Based on pathogenesis of essential hypertension, it can be classified into

- a) Volume related hypertension
- b) Pressure related hypertension

### VOLUME RELATED HYPERTENSION:

SODIUM & WATER increase intracellularly



Increase in Cardiac Output



Increase in renal blood flow



Increased renal hydrostatic pressure



Sodium & Water excretion

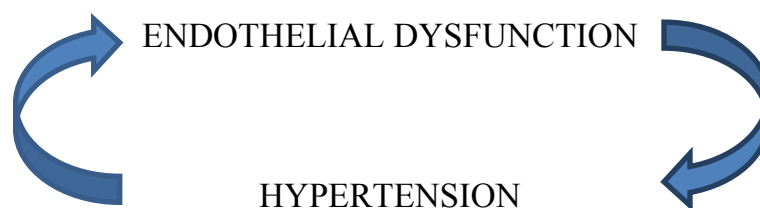
So as long as the kidneys are normally functioning an increase in cardiac output is compensated by pressure natriuresis. So the hypertension in End stage Renal Disease is predominantly due to Volume overload.

## PRESSURE RELATED HYPERTENSION:

Sympathetic overactivity and Renin Angiotensin Aldosterone system contribute to the pressure factors in essential hypertension.

## SYMPATHETIC OVERACTIVITY:

Most important cause of sympathetic overactivity is endothelial dysfunction. This endothelial dysfunction is due to Diabetes mellitus, dyslipidemia, obesity, smoking, oxidative stress, obstructive sleep apnoea syndrome and many other factors. This endothelial dysfunction causes hypertension and hypertension further aggravates endothelial dysfunction. This vicious cycle contributes towards deleterious effects of hypertension.



This dysfunctional endothelium causes defective smooth muscle growth and functional vasoconstriction contributing to increased peripheral vascular resistance and hypertension.

## RENIN ANGIOTENSIN ALDOSTERONE SYSTEM:

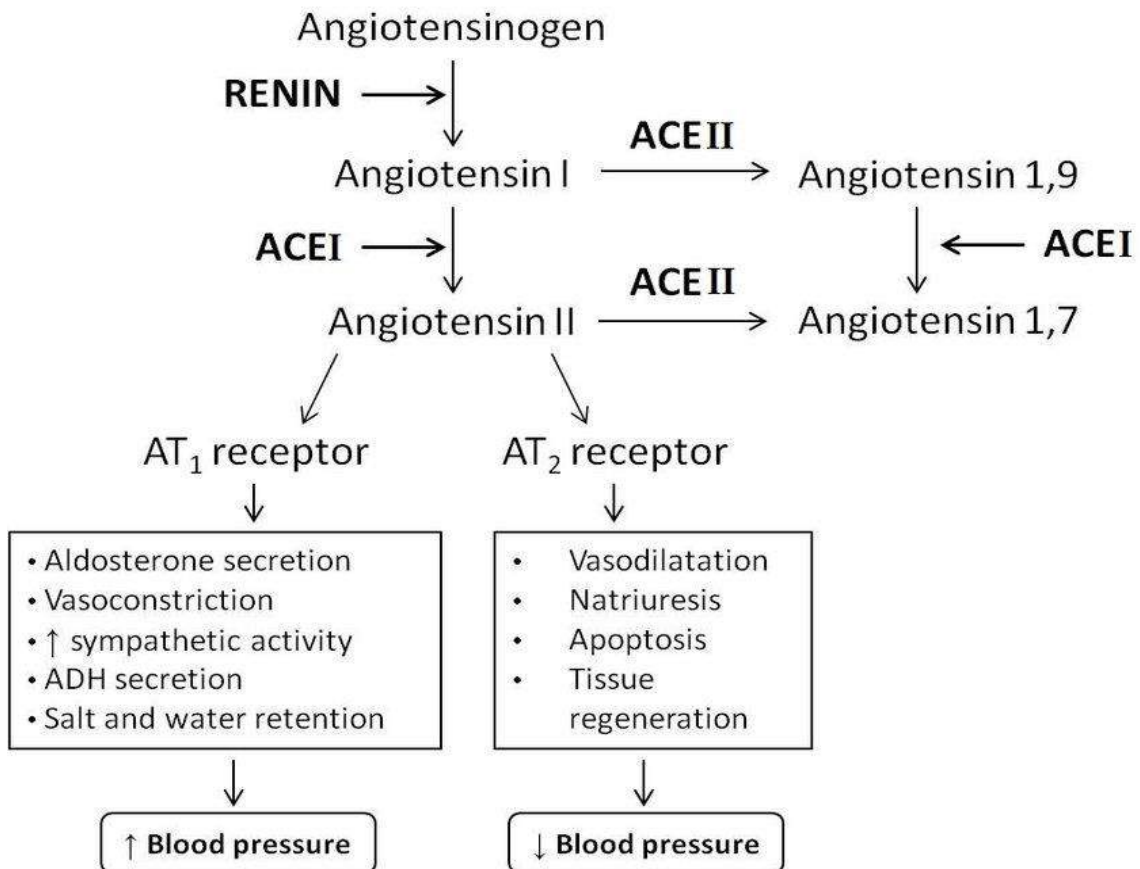
Three stimuli for renin are 1) Beta - 1 Sympathetic stimulation

2) Decreased Renal perfusion pressure

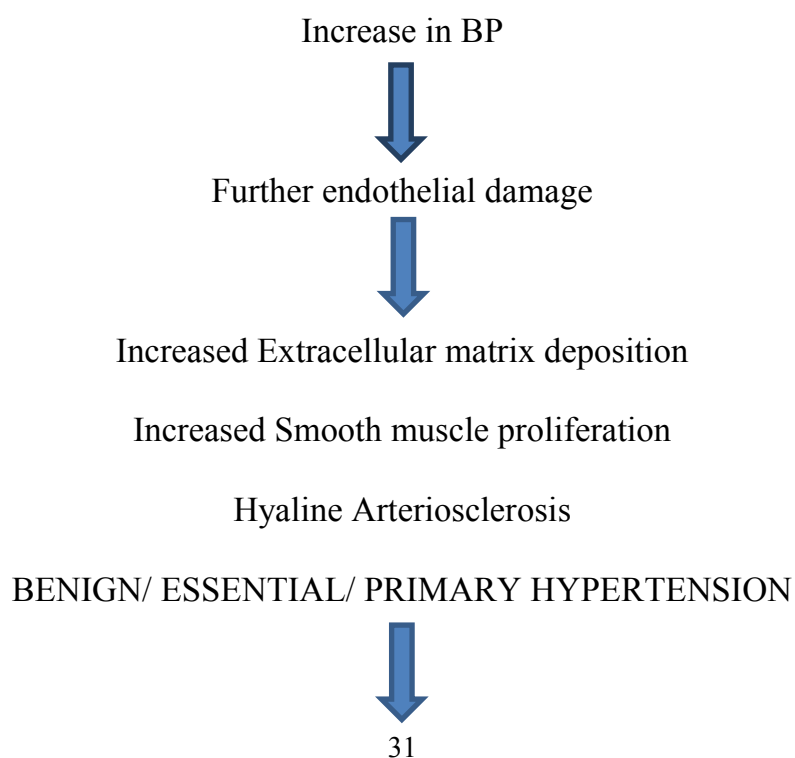
3) Decreased Sodium delivery to Macula densa

4) Prostaglandins





VASCULAR CHANGES IN HYPERTENSION:





Worsening of arteriosclerosis and atherosclerosis

Rupture and Embolisation of atheroma



Clotting and occlusion of small vessels

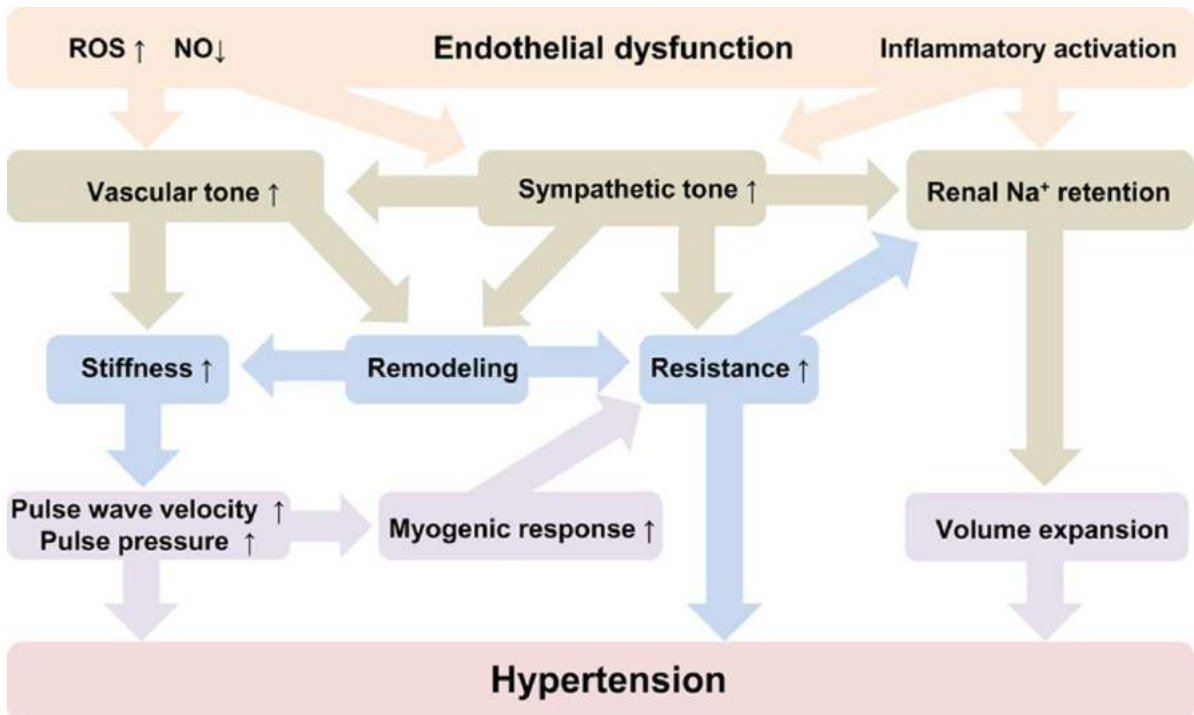


**MALIGNANT HYPERTENSION**

Hyperplastic arteriosclerosis

Fibrinoid necrosis

Onion skin change



## SECONDARY HYPERTENSION:

Of the total, 5-15% are secondary hypertension<sup>22</sup>. Complications in secondary hypertension are more and intense than that of primary hypertension. Broadly the causes of secondary hypertension include

- a) Renal
  - a. Renovascular
  - b. Renoparenchymal
- b) Endocrine
- c) Monogenic
- d) Miscellaneous

Cause	Prevalence in hypertensive patients	Suggestive symptoms and signs	Screening Investigations
Obstructive sleep apnoea	5–10%	Snoring; obesity (can be present in non-obese); morning headache; daytime somnolence	Epworth score and ambulatory polygraphy
Renal parenchymal disease	2–10%	Mostly asymptomatic; diabetes; haematuria, proteinuria, nocturia; anaemia, renal mass in adult polycystic CKD	Plasma creatinine and electrolytes, eGFR; urine dipstick for blood and protein, urinary albumin:creatinine ratio; renal ultrasound
<b>Renovascular disease</b>			
Atherosclerotic renovascular disease	1–10%	Older; widespread atherosclerosis (especially PAD); diabetes; smoking; recurrent flash pulmonary oedema; abdominal bruit	Duplex renal artery Doppler or CT angiography or MR angiography
Fibromuscular dysplasia		Younger; more common in women; abdominal bruit	
<b>Endocrine causes</b>			
Primary Aldosteronism	5 - 15%	Mostly asymptomatic; muscle weakness (rare)	Plasma aldosterone and renin, and aldosterone:renin ratio; hypokalaemia (in a minority): note hypokalaemia can depress aldosterone levels
Phaeochromocytoma	<1%	Episodic symptoms (the 5 'Ps'): paroxysmal hypertension, pounding headache, perspiration, palpitations, and pallor; labile BP; BP surges precipitated by drugs (e.g. beta-blockers, metoclopramide, sympathomimetics, opioids, and tricyclic antidepressants)	Plasma or 24 h urinary fractionated metanephrines
Cushing's syndrome	<1%	Moon face, central obesity, skin atrophy, striae and bruising; diabetes; chronic steroid use	24 h urinary-free cortisol
Thyroid disease (hyper- or hypothyroidism)	1 - 2%	Signs and symptom of hyper- or hypothyroidism	Thyroid function tests
Hyperparathyroidism	<1%	Hypercalcaemia, hypophosphataemia	Parathyroid hormone, Ca <sup>2+</sup>
<b>Other causes</b>			
Coarctation of the aorta	<1%	Usually detected in children or adolescence; different BP ( $\geq 20/10$ mmHg) between upper–lower extremities and/or between right–left arm and delayed radial-femoral femoral pulsation; low ABI interscapular ejection murmur; rib notching on chest X-ray	Echocardiogram

Age group	Per cent with underlying cause	Typical causes
Young children (<12 years)	70 - 85	<ul style="list-style-type: none"> <li>● Renal parenchymal disease</li> <li>● Coarctation of the aorta</li> <li>● Monogenic disorders</li> </ul>
Adolescents (12–18 years)	10–15	<ul style="list-style-type: none"> <li>● Renal parenchymal disease</li> <li>● Coarctation of the aorta</li> <li>● Monogenic disorders</li> </ul>
Young adults (19–40 years)	5–10	<ul style="list-style-type: none"> <li>● Renal parenchymal disease</li> <li>● Fibromuscular dysplasia (especially in women)</li> <li>● Undiagnosed monogenic disorders</li> </ul>
Middle-aged adults (41–65 years)	5–15	<ul style="list-style-type: none"> <li>● Primary aldosteronism</li> <li>● Obstructive sleep apnoea</li> <li>● Cushing's syndrome</li> <li>● Pheochromocytoma</li> <li>● Renal parenchymal disease</li> <li>● Atherosclerotic renovascular disease</li> </ul>
Older adults (>65 years)	5–10	<ul style="list-style-type: none"> <li>● Atherosclerotic renovascular disease</li> <li>● Renal parenchymal disease</li> <li>● Thyroid disease</li> </ul>

Medication/substance	
Oral contraceptive pill	Especially oestrogen containing; cause hypertension in ~5% of women, usually mild but can be severe
Diet pills	For example, phenylpropanolamine and sibutramine
Nasal decongestants	For example, phenylephrine hydrochloride and naphazoline hydrochloride
Stimulant drugs	Amphetamine, cocaine, and ecstasy; these substances usually cause acute rather than chronic hypertension
Liquorice	Chronic excessive liquorice use mimics hyperaldosteronism by stimulating the mineralocorticoid receptor and inhibiting cortisol metabolism
Immunosuppressive medications	For example, cyclosporin A (tacrolimus has less effect on BP and rapamycin has almost no effect on BP) and steroids (e.g. corticosteroids and hydrocortisone)
Antiangiogenic cancer therapies	Antiangiogenic drugs such as VEGF inhibitors (e.g. bevacizumab), tyrosine kinase inhibitors (e.g. sunitinib), and sorafenib have been reported to increase BP
Other drugs and substances that may raise BP	Anabolic steroids, erythropoietin, non-steroidal anti-inflammatory drugs, and herbal remedies (e.g. ephedra and ma huang)

Condition	Phenotype	Mechanism and effect
Liddle syndrome	Hypokalaemia, metabolic alkalosis, low PRA or PRC, low PAC	Increased renal tubular ENaC activity: responds to treatment with amiloride
Apparent mineralocorticoid excess	Hypokalaemia, metabolic alkalosis, low PRA or PRC, low PAC	Decreased 11 $\beta$ -dehydrogenase isoenzyme 2
Gordon syndrome	Hyperkalaemia, metabolic acidosis, low PRA or PRC, low PAC	Overactivity of sodium chloride co-transporter
Geller syndrome	Pregnancy-exacerbated hypertension, low PRA or PRC, low PAC	Agonist effect of progesterone on the mineralocorticoid receptor
Glucocorticoid remediable hypertension	Hypokalaemia, metabolic alkalosis, low PRC or PRA, and increased PAC	Chimeric CYP11 $\beta$ 1 to CYP11 $\beta$ 2 gene: response to treatment with glucocorticoids

## HYPERTENSIVE URGENCY:<sup>23-34</sup>

SBP >180 mmHg or DBP >120mmHg without evidence of any target organ damage. Most common cause is skipping of drugs. Usually requires oral therapy only.

## HYPERTENSIVE EMERGENCY:<sup>23-34</sup>

It is an acute medical emergency. It is diastolic BP >120mmHg with target organ damage. It requires intravenous drug administration for BP lowering.

## ACCELERATED HYPERTENSION:<sup>23-34</sup>

Hypertensive emergency with new onset changes in the fundus, which may be

- Flame shaped haemorrhage
- Cotton wool spots – these are soft exudates. It is due to stasis of axoplasmic flow in the outer nerve fibre layer of retina and thus they are a pseudo exudate.
- Hard exudates – It is a true exudate and it is due to exudation of fluid from capillaries.

## MALIGNANT HYPERTENSION:<sup>23-34</sup>

Accelerated hypertension with Papilloedema constitutes malignant hypertension.

## RESISTANT HYPERTENSION:<sup>23-34</sup>

It is the hypertension without optimal control – preferably less than 140/90 mmHg with the patient despite being on optimal dose 3 anti hypertensives of which one is diuretic. It is usually with a secondary cause. Before diagnosing a resistant hypertension we should first rule out pseudohypertension.

Causes of Pseudo hypertension:

- (1) Poor adherence : Approximately 50% of patients who undergo therapeutic drug monitoring for pseudo-resistant hypertension have poor adherence to recommended medications, which is closely proportional to the number of pills they take.
- (2) Whiter coat phenomena: It is advised to confirm office hypertension with

ABPM or HBPM before confirming the diagnosis of resistant hypertension since white-coat phenomenon, in which BP is raised at the office but controlled at ABPM or HBPM, is not unusual in these individuals.

(3) Wrong technique: An erroneous rise of blood pressure can be caused by poor office BP measuring methodology, such as the use of cuffs that are too small in relation to arm circumference.

(4) Artery calcification: Significant brachial artery calcification, particularly in elderly people with severely calcified arteries.

(5) Clinician inertia which leads to insufficient dosages and irrational combinations.



<b>Characteristics of patients with resistant hypertension</b>	<b>Causes of secondary resistant hypertension</b>	<b>Drugs and substances that may cause raised BP</b>
<p><b>Demographics</b></p> <ul style="list-style-type: none"> <li>● Older age (especially &gt;75 years)</li> <li>● Obesity</li> <li>● More common in black people</li> <li>● Excess dietary sodium intake</li> <li>● High baseline BP and chronicity of uncontrolled hypertension</li> </ul> <p><b>Concomitant disease</b></p> <ul style="list-style-type: none"> <li>● HMOD: LVH and/or CKD</li> <li>● Diabetes</li> <li>● Atherosclerotic vascular disease</li> <li>● Aortic stiffening and isolated systolic hypertension</li> </ul>	<p><b>More common causes</b></p> <ul style="list-style-type: none"> <li>● Primary hyperaldosteronism</li> <li>● Atherosclerotic renovascular disease</li> <li>● Sleep apnoea</li> <li>● CKD</li> </ul> <p><b>Uncommon causes</b></p> <ul style="list-style-type: none"> <li>● Pheochromocytoma</li> <li>● Fibromuscular dysplasia</li> <li>● Aortic coarctation</li> <li>● Cushing's disease</li> <li>● Hyperparathyroidism</li> </ul>	<p><b>Prescribed drugs</b></p> <ul style="list-style-type: none"> <li>● Oral contraceptives</li> <li>● Sympathomimetic agents (e.g. decongestants in proprietary cold remedies)</li> <li>● Non-steroidal anti-inflammatory drugs</li> <li>● Cyclosporin</li> <li>● Erythropoietin</li> <li>● Steroids (e.g. prednisolone and hydrocortisone)</li> <li>● Some cancer therapies</li> </ul> <p><b>Non-prescription drugs</b></p> <ul style="list-style-type: none"> <li>● Recreational drugs (e.g. cocaine, amphetamines, and anabolic steroids)</li> <li>● Excessive liquorice ingestion</li> <li>● Herbal remedies (e.g. ephedra and ma huang)</li> </ul>

<b>Risk factors</b>
Family and personal history of hypertension, CVD, stroke, or renal disease
Family and personal history of associated risk factors (e.g. familial hypercholesterolaemia)
Smoking history
Dietary history and salt intake
Alcohol consumption
Lack of physical exercise/sedentary lifestyle
History of erectile dysfunction
Sleep history, snoring, sleep apnoea (information also from partner)
Previous hypertension in pregnancy/pre-eclampsia
<b>History and symptoms of HMOD, CVD, stroke, and renal disease</b>
Brain and eyes: headache, vertigo, syncope, impaired vision, TIA, sensory or motor deficit, stroke, carotid revascularization, cognitive impairment, dementia (in the elderly)
Heart: chest pain, shortness of breath, oedema, myocardial infarction, coronary revascularization, syncope, history of palpitations, arrhythmias (especially AF), heart failure
Kidney: thirst, polyuria, nocturia, haematuria, urinary tract infections
Peripheral arteries: cold extremities, intermittent claudication, pain-free walking distance, pain at rest, peripheral revascularization
Patient or family history of CKD (e.g. polycystic kidney disease)
<b>History of possible secondary hypertension</b>
Young onset of grade 2 or 3 hypertension (<40 years), or sudden development of hypertension or rapidly worsening BP in older patients
History of renal/urinary tract disease
Recreational drug/substance abuse/concurrent therapies: corticosteroids, nasal vasoconstrictor, chemotherapy, yohimbine, liquorice
Repetitive episodes of sweating, headache, anxiety, or palpitations, suggestive of Pheochromocytoma
History of spontaneous or diuretic-provoked hypokalaemia, episodes of muscle weakness, and tetany (hyperaldosteronism)
Symptoms suggestive of thyroid disease or hyperparathyroidism
History of or current pregnancy and oral contraceptive use
History of sleep apnoea
<b>Antihypertensive Drug Treatment</b>
Current/past antihypertensive medication including effectiveness and intolerance to previous medications
Adherence to therapy

## KEY PHYSICAL EXAMINATION:

Apart from the routine physical examination of the patient the following points are to be done in physical examination:<sup>35-39</sup>

<b>Body habitus</b>
Weight and height measured on a calibrated scale, with calculation of BMI
Waist circumference
<b>Signs of HMOD</b>
Neurological examination and cognitive status
Fundoscopic examination for hypertensive retinopathy
Palpation and auscultation of heart and carotid arteries
Palpation of peripheral arteries
Comparison of BP in both arms (at least once)
<b>Secondary hypertension</b>
Skin inspection: cafe-au-lait patches of neurofibromatosis (phaeochromocytoma)
Kidney palpation for signs of renal enlargement in polycystic kidney disease
Auscultation of heart and renal arteries for murmurs or bruits indicative of aortic coarctation, or renovascular hypertension
Comparison of radial with femoral pulse: to detect radio-femoral delay in aortic coarctation
Signs of Cushing's disease or acromegaly
Signs of thyroid disease

## ROUTINE LABORATORY ASSESSMENT:<sup>35-39</sup>

The routine laboratory assessment in hypertensives are aimed at detecting additional comorbidities like diabetes mellitus, dyslipidemia, electrolyte disturbances, creatinine clearance status, liver function tests. Haematocrit status may point out towards hyperviscosity states. Further urine analysis is done to estimate the albumin creatinine ratio which cost effectively estimates the proteinuria status which is indirectly reflective of glomerular pathology. Also the 12 lead ECG is taken to assess cardiac electrical status and function in hypertensive patients.

<b>Routine laboratory tests</b>
Haemoglobin and/or haematocrit
Fasting blood glucose and glycated HbA <sub>1c</sub>
Blood lipids: total cholesterol, LDL cholesterol, HDL cholesterol
Blood triglycerides
Blood potassium and sodium
Blood uric acid
Blood creatinine and eGFR
Blood liver function tests
Urine analysis: microscopic examination; urinary protein by dipstick test or, ideally, albumin:creatinine ratio
12-lead ECG

## HYPERTENSION MEDIATED ORGAN DAMAGE:

### HYPERTENSION AND HEART:

In hypertensive patients, a persistently elevated left ventricular workload can lead to left ventricular hypertrophy, impaired LV relaxation, left atrial enlargement, increased risk of arrhythmias, particularly AF and heart failure with preserved ejection fraction as well as heart failure with reduced ejection fraction.

### ELECTROCARDIOGRAPHY:

All hypertension patients should get a 12-lead ECG as part of their standard evaluation. The sensitivity of the ECG to LVH detection varies with body weight and is not a very sensitive approach. Even when other CV risk variables and echocardiographic LV mass are taken into account, ECG LVH still offers independent predictive information<sup>40</sup>. A "strain pattern" on an ECG is linked to a higher risk of LVH and other conditions. With increasing levels of hypertension, ECG LVH is more common<sup>41</sup>. LVH cannot be ruled out by the ECG because to its low sensitivity. Echocardiography is advised to know the precise knowledge of heart anatomy and function to guide therapy choices. When LVH is visible on the ECG, it can be utilised to monitor changes in LVH in both treated and untreated patients throughout follow-up<sup>42,43</sup>.

ECG voltage criteria	Criteria for LVH
$S_{V1} + R_{V5}$ (Sokolow–Lyon criterion)	>35 mm
R wave in aVL	$\geq 11$ mm
$S_{V3} + R_{aVL}$ (Cornell voltage) <sup>a</sup> Cornell duration product <sup>b</sup>	>28 mm (men)
	>20 mm (women)
	>2440 mm.ms

## ECHOCARDIOGRAM:

Regression of echocardiographic LVH as a result of hypertension treatment indicates a better prognosis. Echocardiographic left ventricular hypertrophy is a powerful predictor of death in both hypertensive patients and the general population<sup>44,45</sup>. Additionally, left ventricular geometry, left atrial volume, aortic root dimensions, left ventricular systolic and diastolic function, pump efficiency, and output impedance are all revealed by two-dimensional transthoracic echocardiography (TTE)<sup>46,47</sup>. Uncertainty exists over the usefulness of other variables in stratifying CV risk beyond the presence of increased left ventricular mass and left ventricular dilation

Parameter	Measure	Abnormality threshold
LVH	LV mass/height <sup>2.7</sup> (g/m <sup>2.7</sup> )	>50 (men)
		>47 (women)
LVH <sup>a</sup>	LV mass/BSA (g/m <sup>2</sup> )	>115 (men)
		>95 (women)
LV concentric geometry	RWT	≥0.43
LV chamber size	LV end-diastolic diameter/height (cm/m)	>3.4 (men)
		>3.3 (women)
Left atrial size (elliptical)	Left atrial volume/height <sup>2</sup> (mL/m <sup>2</sup> )	>18.5 (men)
		>16.5 (women)

Three-dimensional TTE has better reproducibility than two-dimensional TTE for

quantitative analysis, especially for Left ventricular mass, left ventricular volumes and left ventricular ejection fraction, but much less prognostic validation. The gold standard for quantifying heart anatomical and functional information is cardiac magnetic resonance.

Diastolic dysfunction is typically linked to abnormal left ventricular geometry in hypertension patients, and this dysfunction may be further assessed by combining transmitral flow and tissue Doppler tests. Additionally, there is typically an increase in left atrial size in hypertension individuals, which is linked to diastolic dysfunction, poor cardiovascular outcomes and incidence of atrial fibrillation. A suprasternal view should be carried out as part of the diagnostic process for secondary hypertension in order to detect aortic coarctation.

#### CAROTID ARTERY IMAGING:

CV risk is predicted by carotid intima-media thickness (IMT), which is measured by carotid ultrasonography and the presence of plaques. Both the IMT value at the carotid bifurcations (which predominantly reflects atherosclerosis) and the IMT value at the level of the common carotid artery are affected by this (reflecting primarily hypertension-related hypertrophy). The upper limit of normal changes with age, but a carotid IMT of  $>0.9$  mm is regarded as abnormal<sup>48</sup>. An IMT  $>1.5$  mm, a focused increase in thickness of 0.5 mm or 50% of the surrounding carotid IMT value can all be used to determine the existence of a plaque<sup>49</sup>. Stenotic carotid plaques give higher prognosis accuracy for future myocardial infarction compared to IMT and have a substantial predictive value for both stroke and myocardial infarction, independent of established CV risk factors. Patients will be automatically reclassified from intermediate to high risk if they have carotid plaques.

Routine carotid imaging is not advised, nevertheless, unless it is clinically necessary, such as in cases of carotid bruit, prior TIA, cerebrovascular illness, or as part of the evaluation of individuals who have vascular disease.

#### PULSE WAVE VELOCITY:

The most significant pathophysiological factor influencing isolated systolic hypertension and the age-dependent rise in pulse pressure is large arterial stiffness. The gold standard for assessing the stiffness of major arteries is the carotid-femoral pulse wave velocity (PWV)<sup>50</sup>. In middle-aged hypertension individuals, a pulse wave velocity >10 m/s is regarded as a conservative estimate of severe abnormalities of aortic function. Numerous research have revealed that PWV provides additional benefits above and beyond conventional risk indicators like SCORE and the Framingham risk score. PWV measurement, however, is neither practicable or advised for use in everyday practice.

#### ANKLE BRACHIAL PRESSURE INDEX:

It is possible to measure the ankle-brachial index automatically or manually using a continuous wave Doppler unit and a blood pressure sphygmomanometer. Lower extremity artery disease (LEAD) is indicated by a low ABI (0.9), which is typically a sign of advanced atherosclerosis and has predictive value for CV events<sup>51</sup>. It is linked to an almost two-fold higher rate of major coronary events and 10 year CV mortality compared to the overall rate in each Framingham category. Men who have even asymptomatic LEAD and a poor ABI have a significant incidence of CV morbid and fatal events that is close to 20% in 10 years. The routine use of ABI in hypertensive individuals is not advised, but it should be taken into consideration in those who have LEAD symptoms or signs AND in those who are at moderate risk in whom a positive LEAD test reclassifies



them as high risk group.

#### KIDNEYS IN HYPERTENSION:

After diabetes, hypertension is the second most significant factor in CKD. Primary renal disease that is asymptomatic may potentially occur with hypertension. An rise in serum creatinine is the most typical indicator of a change in renal function. Because a significant loss in renal function must occur before serum creatinine increases, this indicator of renal impairment is insensitive. Additionally, antihypertensive therapy frequently results in an acute rise in serum creatinine of up to 20–30%, especially with renin-angiotensin system (RAS) blockers<sup>52</sup>. This rise in serum creatinine has a functional basis and typically does not reflect manifest renal injury, but its long-term clinical significance is unknown.

Reduced renal function and/or albuminuria are used to make the diagnosis of hypertension-induced renal injury. The 2009 CKD-Epidemiology Collaboration formula is used to determine the estimated glomerular filtration rate (eGFR), which is used to categorise CKD. The most effective way to determine how much urine albumin is being excreted is to assess the albumin: creatinine ratio (ACR), which is done from a spot urine sample, ideally one taken in the morning. Progressively declining eGFR and rising albuminuria are indicators of progressive renal function loss and serve as independent and complementary predictors of rising CV risk and renal disease development<sup>53</sup>. All hypertensive patients should have their serum creatinine, eGFR, and ACR noted, and if CKD is identified, these tests should be repeated at least once a year. In contrast to a normal ACR, one negative urine dipstick test does not rule out albuminuria.

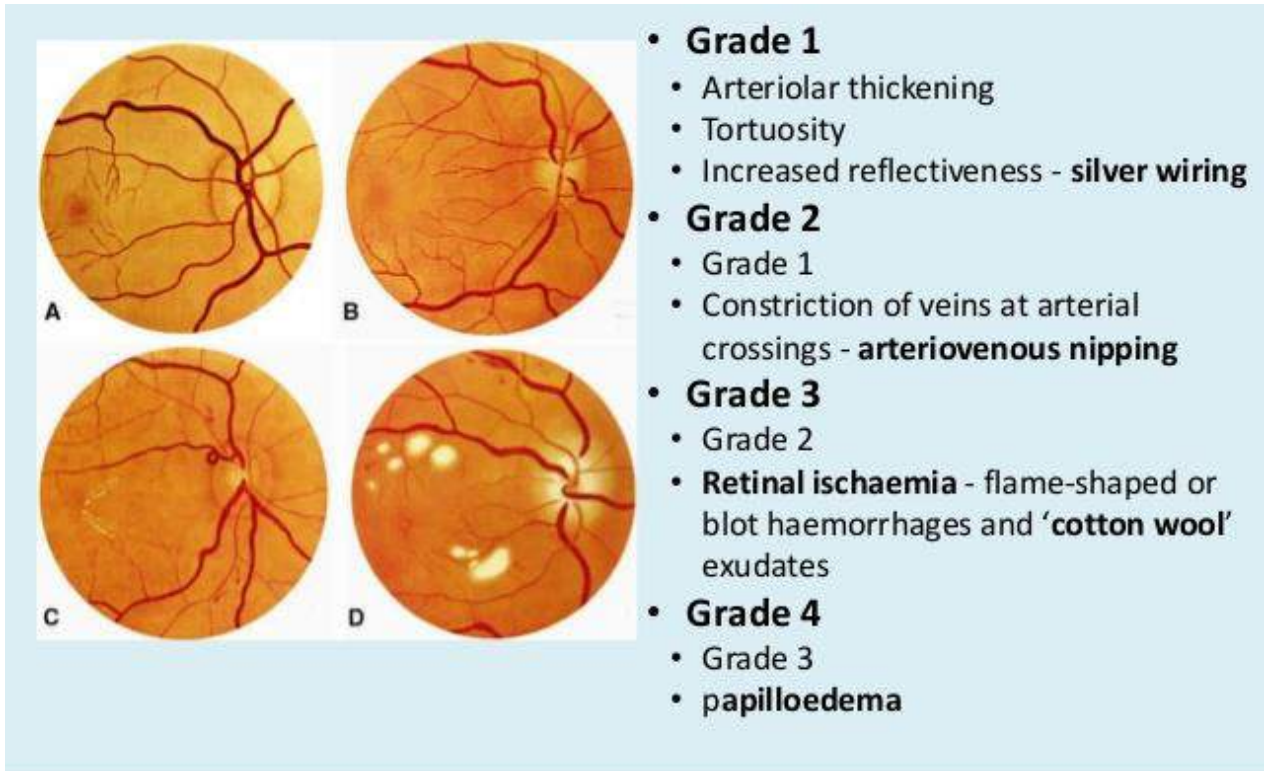
## HYPERTENSIVE RETINOPATHY:

The importance of hypertensive retinopathy via fundoscopy in terms of prognosis has been well established. The detection of severe hypertensive retinopathy, retinal haemorrhages, microaneurysms, hard exudates, cotton wool patches and papilloedema is highly repeatable and strongly prognostic of death<sup>54</sup>. In contrast, even with seasoned observers, there is little interobserver and intraobserver consistency and reduced predictive value for indications of localised or generalised arteriolar constriction and arteriovenous nicking during the early stages of hypertensive retinopathy. Individuals with grade 2 or 3 hypertension or hypertensive patients with diabetes should have a fundoscopy since these patients are more likely to have severe retinopathy. Other hypertensive individuals can be candidates for a fundoscopy. The viability of more frequent fundoscopies using smartphone technology continue to develop as novel methods to view the fundus.

## Classification

### • Keith-Wagener-Barker classification

Grade	Description
Grade 1	Slight narrowing, sclerosis, and tortuosity of the retinal arterioles; mild, asymptomatic hypertension
Grade 2	Definite narrowing, focal constriction, sclerosis, and AV nicking; blood pressure is higher and sustained; few, if any, symptoms referable to blood pressure
Grade 3	Retinopathy (cotton-wool patches, arteriolosclerosis, hemorrhages); blood pressure is higher and more sustained; headaches, vertigo, and nervousness; mild impairment of cardiac, cerebral, and renal function
Grade 4	Neuroretinal edema, including papilledema; Siegrist streaks, Elschnig spots; blood pressure persistently elevated; headaches, asthenia, loss of weight, dyspnea, and visual disturbances; impairment of cardiac, cerebral, and renal function



## BRAIN AND HYPERTENSION<sup>55,56,57</sup>:

The most severe and acute clinical signs of brain injury including transient ischemic attack (TIA) and stroke are widely prevalent in hypertension. Magnetic resonance imaging (MRI) can identify white matter hyperintensities, silent microinfarcts (most of which are tiny and deep, i.e. lacunar infarcts), microhaemorrhages and brain atrophy even in the asymptomatic period of brain damage. As a result of vascular and degenerative dementia, white matter hyperintensities and silent infarcts are linked to an increased risk of stroke and cognitive loss. Although white matter hyperintensity and silent brain infarcts should be looked for in all hypertensive patients with neurological disturbances, cognitive decline and in patients with memory loss, the availability and cost of brain MRI do not permit its widespread use for the evaluation of hypertensive patients.

An MRI should be ordered if there is a family history of middle-aged cerebral

haemorrhage and early-onset dementia. Cognitive evaluation tests should be taken into account in the clinical assessment of hypertensive patients with a history suggesting early cognitive impairment since cognitive impairment in older individuals is, at least in part, connected to hypertension. The Mini-Mental State Examination was once the most often used technique in clinical trials, but more advanced cognitive tests are gradually taking its place since they are more appropriate for regular clinic visits.

Basic screening tests for HMOD	Indication and interpretation
12-lead ECG	Screen for LVH and other possible cardiac abnormalities, and to document heart rate and cardiac rhythm
Urine albumin:creatinine ratio	To detect elevations in albumin excretion indicative of possible renal disease
Blood creatinine and eGFR	To detect possible renal disease
Fundoscopy	To detect hypertensive retinopathy, especially in patients with grade 2 or 3 hypertension
More detailed screening for HMOD	
Echocardiography	To evaluate cardiac structure and function, when this information will influence treatment decisions
Carotid ultrasound	To determine the presence of carotid plaque or stenosis, particularly in patients with cerebrovascular disease or vascular disease elsewhere
Abdominal ultrasound and Doppler studies	<ul style="list-style-type: none"> <li>● To evaluate renal size and structure (e.g. scarring) and exclude renal tract obstruction as possible underlying causes of CKD and hypertension</li> <li>● Evaluate abdominal aorta for evidence of aneurysmal dilatation and vascular disease</li> <li>● Examine adrenal glands for evidence of adenoma or pheochromocytoma (CT or MRI preferred for detailed examination); see section 8.2 regarding screening for secondary hypertension</li> <li>● Renal artery Doppler studies to screen for the presence of renovascular disease, especially in the presence of asymmetric renal size</li> </ul>
PWV	An index of aortic stiffness and underlying arteriosclerosis
ABI	Screen for evidence of LEAD
Cognitive function testing	To evaluate cognition in patients with symptoms suggestive of cognitive impairment
Brain imaging	To evaluate the presence of ischaemic or haemorrhagic brain injury, especially in patients with a history of cerebrovascular disease or cognitive decline

## TREATMENT OF HYPERTENSION:

### LIFESTYLE CHANGES<sup>58,59</sup>:

Healthy lifestyle choices can lower CV risk and can stop or postpone the onset of hypertension. In individuals with grade 1 hypertension, effective lifestyle adjustments may be sufficient to postpone or eliminate the requirement for pharmacological therapy. However, they should never postpone the start of pharmacological therapy in patients with HMOD or those who are at high CV risk. They can also enhance the effectiveness of BPlowering medication. The lack of persistence over time is a significant disadvantage of lifestyle change measurements. Salt restriction, moderate alcohol intake, high consumption of fruits and vegetables, weight loss and maintaining an appropriate body weight, and regular physical activity are the lifestyle changes that have been demonstrated to lower blood pressure. Furthermore, smoking tobacco has an acute prolonged pressor impact that may increase ambulatory blood pressure over the day, although smoking cessation and other lifestyle changes are also crucial beyond blood pressure (i.e. for cardiovascular disease and cancer prevention).

### SALT RESTRICTION<sup>60-67</sup>:

There is proof indicating sodium intake and blood pressure are related, and studies have indicated that having too much sodium (more than 5 grammes, or about one tiny teaspoon, per day) has pressor effect. Age-related increases in SBP and a higher incidence of hypertension may have an impact. Also, sodium restriction has been demonstrated in several studies to reduce blood pressure. A recent meta-analysis of these studies revealed that those with hypertension saw a more dramatic impact (-5.4/ -2.8 mmHg) with a

decrease of 1.75 g sodium per day (4.4 g salt/day) in SBP/DBP. Because of low dietary durability, the positive impact of decreasing salt consumption on BP tends to fade with time. It is more effective for lowering blood pressure in older patients, black patients, and those with diabetes, metabolic syndrome, or kidney disease. Effective sodium restriction may reduce the dosage of BP-lowering medications required to regulate blood pressure in persons on hypertension treatment. Reduced dietary sodium's impact on CV events is yet unknown. Epidemiological studies have not found any proof that consuming significantly minimal sodium may be harmful. Intake of salt is typically between 3.5 and 5.5 grammes per day (with equal to 9–12 grammes of salt per day), with noticeable variations across areas within countries as well as between nations. ESH suggests keeping sodium consumption under 2.0 grammes per day (corresponding to around 5.0 g of salt per day) in the average attempting to accomplish this objective in all hypertensive patients. Effective salt lowering is challenging, and there is frequently inadequate understanding of which foods are rich in salt. Advice ought to be advised to stay away from dishes with a high amount of salt.

#### ALCOHOL RESTRICTION<sup>68</sup>:

Alcohol use has a long-standing positive linear relationship with blood pressure, the prevalence of hypertension, and the risk of cardiovascular disease.

Binge drinking can exert significant pressor effect. According to the Prevention and Treatment of Hypertension Study (PATHS), the intervention group had a moderate 1.2/0.7 mmHg lower blood pressure than the control group at the conclusion of the six-month period. Alcohol intake should be restricted for hypertensive males to no more than 14 units per week and for hypertensive women to no more than 8 units per week (1 unit is

equal to 125 mL of wine or 250 mL of beer). Additionally encouraged are alcohol-free days during the week and avoiding excessive drinking.

#### OTHER DIETARY HABITS<sup>69-71</sup>:

Regular intake of sugar-sweetened soft drinks has been linked to increased risk for CV disease, metabolic syndrome, and obesity. It is best to dissuade people from drinking these beverages.

Patients with hypertension should be encouraged to consume a healthy, balanced diet that includes fish, whole grains, legumes, fresh fruits, vegetables, low-fat dairy products and polyunsaturated fatty acids, especially olive oil, as well as to consume less red meat and unsaturated fats.

#### WEIGHT REDUCTION<sup>72-74</sup>:

Hypertension is linked to excessive weight gain, and lowering weight to an optimal body weight lowers BP. A 5.1 kg weight loss on average resulted in mean SBP and DBP reductions of 4.4 and 3.6 mmHg, respectively. Obesity and CV mortality risk are both elevated in both overweight and obese individuals. For the treatment of metabolic risk factors, weight loss is advised in overweight and obese hypertensive patients; nevertheless, for many, weight stabilisation may be a fair goal.

Although the ideal BMI is unknown, it is advised to maintain a healthy body weight (BMI of 20 to 25 kg/m<sup>2</sup> in patients under 60 years old; higher in older patients) and waist circumference (94 cm for men and 80 cm for women) for both hypertensive patients and non-hypertensive people to lower blood pressure. Losing weight can also lower CV risk and increase antihypertensive drug effectiveness. A multidisciplinary strategy for weight loss should be used, including food recommendations, consistent

exercise, and motivational counselling. Anti-obesity medications and bariatric surgery, which appears to lower CV risk in individuals who are extremely obese, can both help people lose weight. Physical exercise causes a sharp increase in BP, especially SBP, which is followed by a quick drop down to baseline BP. According to epidemiological research, regular aerobic exercise may help to reduce CV risk and death as well as prevent and cure hypertension. In contrast to other forms of exercise, endurance training lowers blood pressure greater in participants with hypertension (8.3/5.2 mmHg). Based on the data, it is recommended that hypertension patients be encouraged to engage in 5-7 days per week of at least 30 minutes of moderate-intensity dynamic aerobic activity, such as walking, jogging, cycling, or swimming. It may also be advisable to perform strength training activities two to three days a week.

#### PHARMACOTHERAPY:

To get the best BP control, the majority of patients will need pharmacological therapy in addition to lifestyle modifications. The five main drug classes previously recommended for the treatment of hypertension were ACE inhibitors, ARBs, beta-blockers, CCBs, and diuretics (thiazides and thiazide-like diuretics such as chlortalidone and indapamide), based on<sup>75</sup>:

- i) their demonstrated ability to lower blood pressure;
- ii) evidence from placebo-controlled studies that they reduce CV events;
- iii) the evidence of broad equivalence on overall CV morbidity and mortality, with the conclusion that the benefit from using them primarily comes from lowering blood pressure.



<b>Recommendations</b>	<b>Class<sup>a</sup></b>	<b>Level<sup>b</sup></b>
Salt restriction to <5 g per day is recommended. <sup>248,250,255,258</sup>	<b>I</b>	<b>A</b>
It is recommended to restrict alcohol consumption to: <ul style="list-style-type: none"> <li>● Less than 14 units per week for men.</li> <li>● Less than 8 units per week for women.<sup>35</sup></li> </ul>	<b>I</b>	<b>A</b>
It is recommended to avoid binge drinking.	<b>III</b>	<b>C</b>
Increased consumption of vegetables, fresh fruits, fish, nuts, and unsaturated fatty acids (olive oil); low consumption of red meat; and consumption of low-fat dairy products are recommended. <sup>262,265</sup>	<b>I</b>	<b>A</b>
Body-weight control is indicated to avoid obesity (BMI >30 kg/m <sup>2</sup> or waist circumference >102 cm in men and >88 cm in women), as is aiming at healthy BMI (about 20–25 kg/m <sup>2</sup> ) and waist circumference values (<94 cm in men and <80 cm in women) to reduce BP and CV risk. <sup>262,271,273,290</sup>	<b>I</b>	<b>A</b>
Regular aerobic exercise (e.g. at least 30 min of moderate dynamic exercise on 5–7 days per week) is recommended. <sup>262,278,279</sup>	<b>I</b>	<b>A</b>
Smoking cessation, supportive care, and referral to smoking cessation programs are recommended. <sup>286,288,291</sup>	<b>I</b>	<b>B</b>

Combination therapy is almost always more successful than monotherapy at lowering blood pressure; in fact, even low-dosage combination therapy is frequently more effective than monotherapy at its highest dose. Additionally, the variability of the BP response to first treatment is reduced and a steeper dosage response is provided by the combination of drugs targeting several pathways, such as inhibiting the RAS as well as producing vasodilatation and/or diuresis. Even when administered to individuals with grade 1 hypertension, when adverse events necessitating treatment cessation are uncommon, two-drug combinations have been found to be safe and well tolerated with no or a little increase in the incidence of hypotensive episodes.

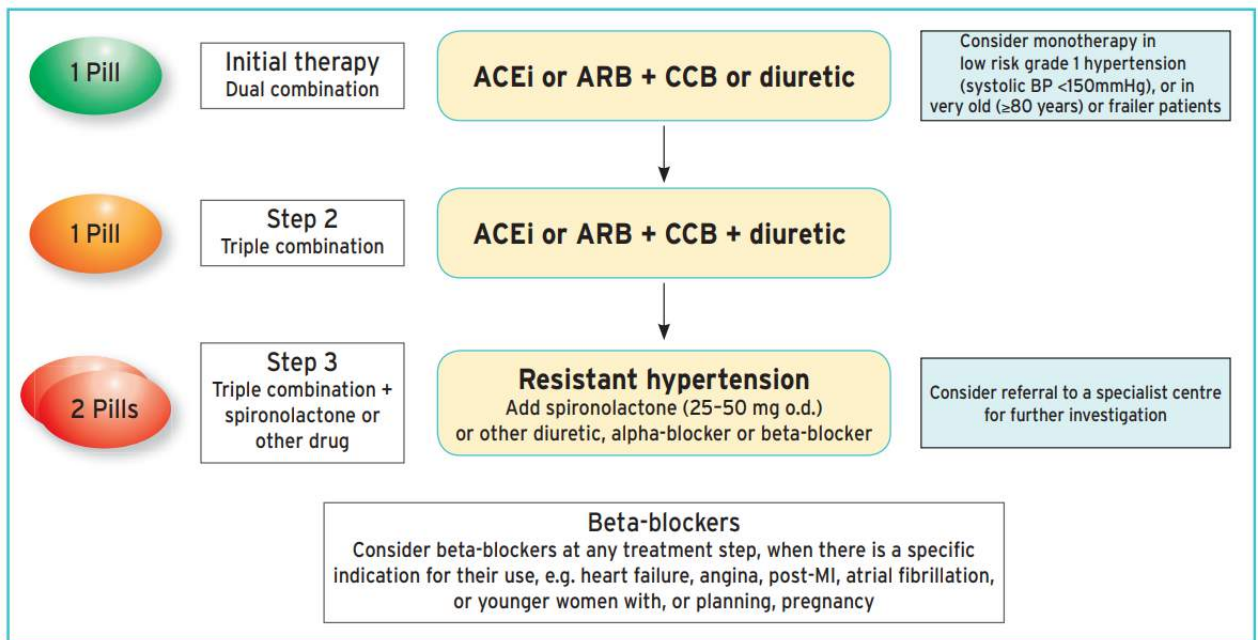
Trial	Comparator	Type of patients	SBP difference (mmHg)	Outcomes [change in relative risk (%)]
<b>ACE inhibitor and diuretic combination</b>				
PROGRESS <sup>27</sup>	Placebo	Previous stroke or TIA	-9	-28% strokes ( <i>P</i> < 0.001)
ADVANCE <sup>229</sup>	Placebo	Diabetes	-5.6	-9% micro/macrovacular events ( <i>P</i> = 0.04)
HYVET <sup>220</sup>	Placebo	Hypertensive; ≥80 years	-15	-34% CV events ( <i>P</i> < 0.001)
<b>ARB and diuretic combination</b>				
SCOPE <sup>330</sup>	Diuretic + placebo	Hypertensive; ≥70 years	-3.2	-28% non-fatal strokes ( <i>P</i> = 0.04)
<b>CCB and diuretic combination</b>				
FEVER <sup>331</sup>	Diuretic + placebo	Hypertensive	-4	-27% CV events ( <i>P</i> < 0.001)
<b>ACE inhibitor and CCB combination</b>				
Syst-Eur <sup>332</sup>	Placebo	Older with ISH	-10	-31% CV events ( <i>P</i> < 0.001)
Syst-China <sup>333</sup>	Placebo	Older with ISH	-9	-37% CV events ( <i>P</i> < 0.004)
<b>Beta-blocker and diuretic combination</b>				
Coope and Warrender <sup>322</sup>	Placebo	Older hypertensive	-18	-42% strokes ( <i>P</i> < 0.03)
SHEP <sup>323</sup>	Placebo	Older with ISH	-13	-36% strokes ( <i>P</i> < 0.001)
STOP-H <sup>324</sup>	Placebo	Older hypertensive	-23	-40% CV events ( <i>P</i> = 0.003)
STOP-H 2 <sup>334</sup>	ACE inhibitor or conventional antihypertensive	Hypertensive	0	NS difference in CV events
<b>Combination of two RAS blockers/ACE inhibitor + ARB or RAS blocker + renin inhibitor)</b>				
ONTARGET <sup>299</sup>	ACE inhibitor or ARB	High-risk patients		More renal events
ALTITUDE <sup>291</sup>	ACE inhibitor or ARB	High-risk diabetic patients		More renal events

Trial	Comparator	Type of patients	SBP difference (mmHg)	Outcomes [change in relative risk (%)]
<b>ACE inhibitor and diuretic combination</b>				
CAPP <sup>335</sup>	BB + diuretic	Hypertensive	+3	+5% CV events (NS)
ACCOMPLISH <sup>327</sup>	ACE inhibitor + CCB	Hypertensive with risk factors	+1	+21% CV events ( <i>P</i> <0.001)
<b>ARB and diuretic combination</b>				
LIFE <sup>317</sup>	BB + diuretic	Hypertensive with LVH	-1	-26% stroke ( <i>P</i> <0.001)
<b>CCB and diuretic combination</b>				
ELSA <sup>336</sup>	BB + diuretic	Hypertensive	0	NS difference in CV events
CONVINCE <sup>233</sup>	BB + diuretic	Hypertensive with risk factors	0	NS difference in CV events
VALUE <sup>337</sup>	ARB + diuretic	High-risk hypertensive	-2.2	-3% CV events ( <i>P</i> = NS)
COPE <sup>338</sup>	CCB + BB	Hypertensive	+0.7	NS difference in CV events or stroke
<b>ACE inhibitor and CCB combination</b>				
NORDIL <sup>339</sup>	BB + diuretic	Hypertensive	+3	NS difference in CV events
INVEST <sup>340</sup>	BB + diuretic	Hypertensive with CAD	0	NS difference in CV events
ASCOT <sup>318</sup>	BB + diuretic	Hypertensive with risk factors	-3	-16% CV events ( <i>P</i> <0.001)
ACCOMPLISH <sup>327</sup>	ACE inhibitor + diuretic	Hypertensive with risk factors	-1	-21% CV events ( <i>P</i> <0.001)
<b>Beta-blocker and diuretic combination</b>				
CAPP <sup>335</sup>	ACE inhibitor + diuretic	Hypertensive	-3	-5% CV events ( <i>P</i> = NS)
LIFE <sup>317</sup>	ARB + diuretic	Hypertensive with LVH	+1	+26% stroke ( <i>P</i> <0.001)
ALLHAT <sup>316</sup>	ACE inhibitor + BB	Hypertensive with risk factors	-2	NS difference in CV events
ALLHAT <sup>316</sup>	CCB + BB	Hypertensive with risk factors	-1	NS difference in CV events
CONVINCE <sup>233</sup>	CCB + diuretic	Hypertensive with risk factors	0	NS difference in CV events
NORDIL <sup>339</sup>	ACE inhibitor + CCB	Hypertensive	-3	NS difference in CV events
INVEST <sup>340</sup>	ACE inhibitor + CCB	Hypertensive with CAD	0	NS difference in CV events
ASCOT <sup>318</sup>	ACE inhibitor + CCB	Hypertensive with risk factors	+3	+16% CV events ( <i>P</i> <0.001)
<b>Beta-blocker and CCB combination</b>				
COPE <sup>329</sup>	ARB + CCB	Hypertensive	+0.8	NS difference in CV events or stroke
<b>ARB and CCB combination</b>				
COPE <sup>329</sup>	CCB + diuretic	Hypertensive	-0.7	NS difference in CV events or stroke
COPE <sup>329</sup>	CCB + BB	Hypertensive	-0.8	NS difference in CV events or stroke
COLM <sup>328</sup>	ARB + diuretic	Older hypertensive	0	NS difference in CV events

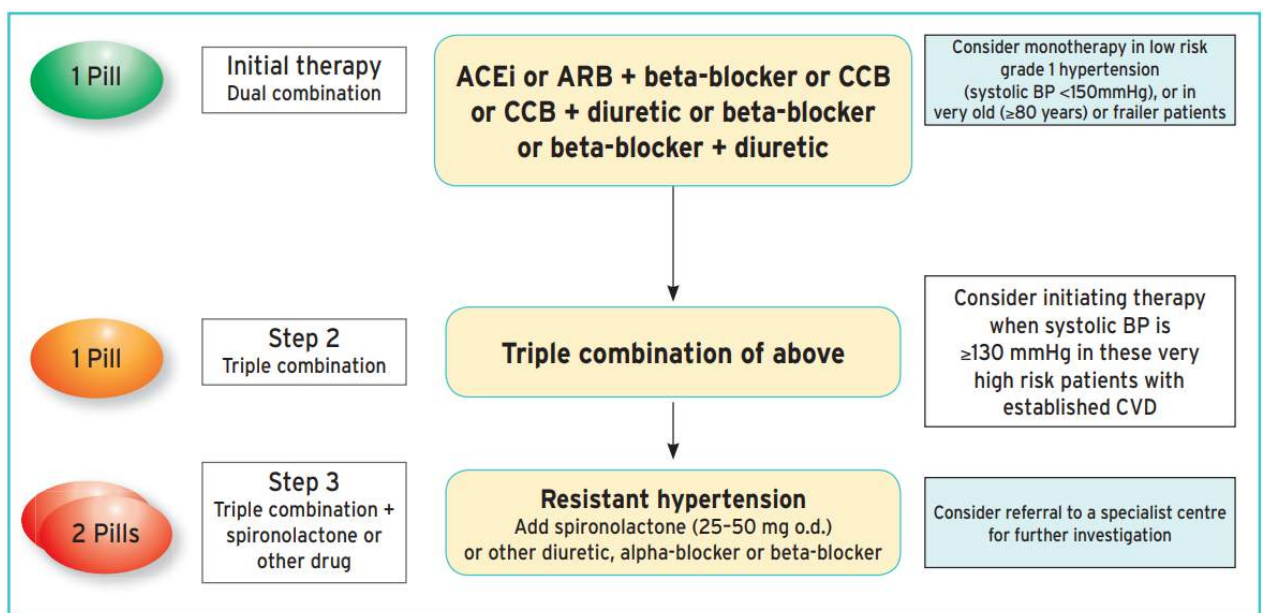
Observational data shows that the length of time needed to attain blood pressure control is a significant factor in clinical outcomes, particularly in individuals at greater risk; a shorter time to control is linked with reduced risk. Additionally, there is evidence from the general hypertensive population that individuals who begin treatment with a two-drug combination have more frequent BP control after a year than those who begin with initial monotherapy. This is likely due to the initial combination treatment's association with better long-term adherence to the recommended treatment regimen and the fact that the initial administration of two drugs prevents therapeutic inertia (i.e., a patient's and clinician's resistance to switching from one drug to another when BP is not under control).

Age group	Office SBP treatment target ranges (mmHg)					Office DBP treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke <sup>a</sup> /TIA	
18 - 65 years	<b>Target to 130</b> <i>or lower if tolerated</i> Not <120	<b>Target to 130</b> <i>or lower if tolerated</i> Not <120	<b>Target to &lt;140 to 130</b> <i>if tolerated</i>	<b>Target to 130</b> <i>or lower if tolerated</i> Not <120	<b>Target to 130</b> <i>or lower if tolerated</i> Not <120	70–79
65 - 79 years <sup>b</sup>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	70–79
≥80 years <sup>b</sup>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	<b>Target to 130-139</b> <i>if tolerated</i>	70–79
<b>Office DBP treatment target range (mmHg)</b>	70–79	70–79	70–79	70–79	70–79	

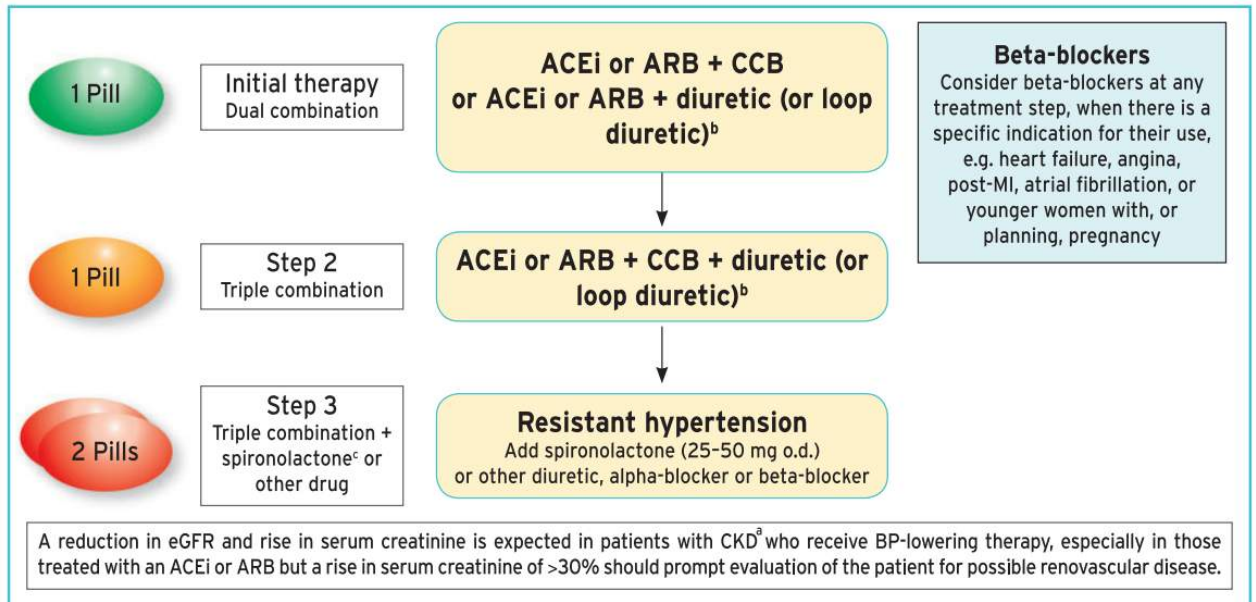
## HYPERTENSION WITHOUT COMPLICATIONS<sup>1</sup>:



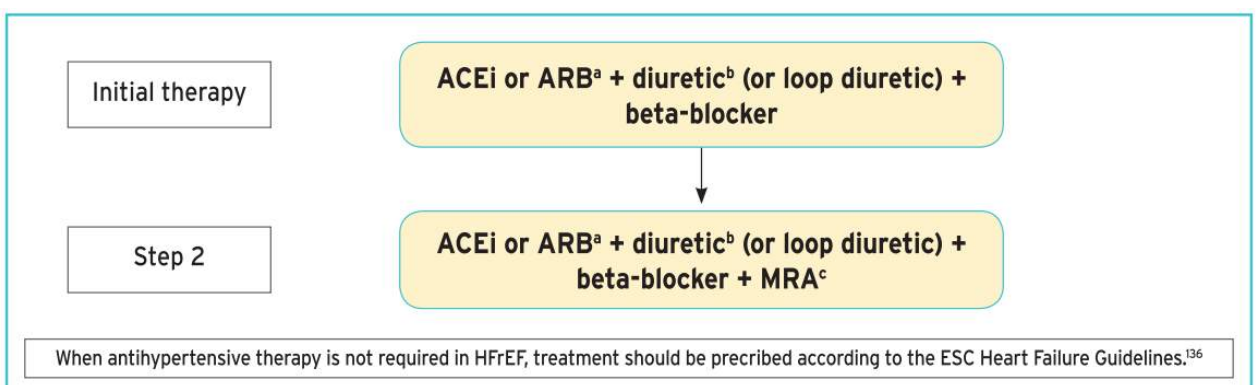
## HYPERTENSION WITH CORONARY ARTERY DISEASE<sup>1</sup>:



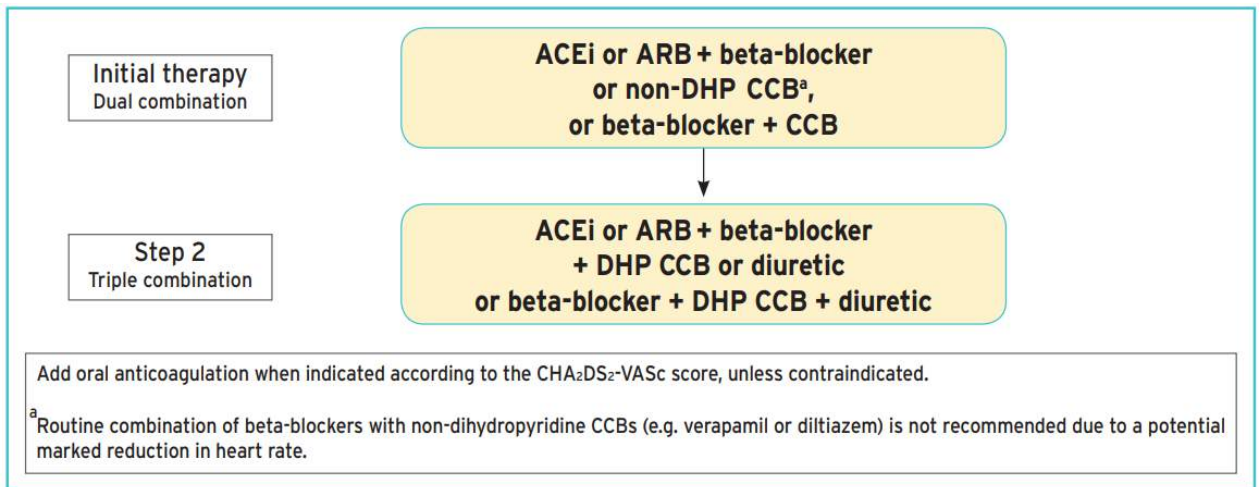
## HYPERTENSION IN CHRONIC KIDNEY DISEASE<sup>1</sup>:



## HYPERTENSION AND HEAR FAILURE WITH REDUCED EJECTION FRACTION<sup>1</sup>:



## HYPERTENSION AND ATRIAL FIBRILLATION<sup>1</sup>:



## **METABOLIC SYNDROME**<sup>76-80</sup>:

The metabolic syndrome, also known as syndrome X or insulin resistance syndrome, is a collection of metabolic disorders that elevates one's risk of developing diabetes and cardiovascular disease. Central obesity, low levels of high-density lipoprotein (HDL) cholesterol, hypertriglyceridemia, hyperglycemia and hypertension are the main characteristics of the metabolic syndrome. The risk of diabetes (insulin resistance ) and CVD are strongly related to intraabdominal circumference. The distribution of adipose tissue between subcutaneous (SC) and visceral depots varies significantly for any given waist circumference. The various age and ethnicity of the populations are investigated with various diagnostic standards and these all have an impact on estimation of prevalence of the metabolic syndrome globally. In general, as people get older, the prevalence of the metabolic syndrome rises. Growing rates of obesity and an anticipated rise in the frequency of the metabolic syndrome are linked to increased global industrialisation, particularly as the population ages. Additionally, the metabolic syndrome's characteristics in a younger population, who are currently predicted to be present in 12% and 30% of obese and overweight children, are reflected in the increased prevalence and severity of obesity among them. In all population around the globe the prevalence of metabolic syndrome rose with age, rising from 19.5% in the 20–39 age group to 48.6% in the over-60 age group<sup>81</sup>. Our study is aimed at estimating the prevalence of metabolic syndrome among hypertensive adult population of age group 20 - 45 years presenting to our hospital's hypertension OP department.



## VARIOUS DIAGNOSTIC CRITERIA FOR METABOLIC SYNDROME:

	WHO (1999)	NCEP-ATP III (2001)	NCEP-R (2004)	IDF (2005)	AACE
Obesity	WHR >0.90 (male) >0.85 (female) or BMI>30 kg/m <sup>2</sup>	WC ≥102 cm (male) ≥88 cm (female)	WC ≥102 cm (male) ≥88 cm (female)	[REQUIREMENT] WC ≥94 cm (male) ≥80 cm (female)	Overweight/Obesity BMI≥25 kg/m <sup>2</sup>
Serum triglycerides	≥150 mg/dl	≥150 mg/dl	≥150 mg/dl or medication	≥150 mg/dl or medication	≥150 mg/dl
Serum HDL	<35 mg/dl (male)	<40 mg/dl (male)	<40 mg/dl (male)	<40 mg/dl (male)	<40 mg/dl (male)
Cholesterol	<39 mg/dl (female)	<50 mg/dl (female)	<50 mg/dl (female) or medication	<50mg/dl (female) or medication	<50 mg/dl (female)
Blood pressure	≥140/90 mmHg	≥130/85 mmHg or medication	≥130/85 mmHg or medication	≥130/85 mmHg or medication	≥130/85 mmHg or medication
Fasting plasma glucose	[REQUIREMENT] FPG≥110 mg/dl	≥110 mg/dl	≥100 mg/dl	≥100 mg/dl or previously diagnosed T2DM	110-126 mg/dl
Other risk factors	Urinary albumin excretion rate≥20 µg/min or albumin/creatinine ratio≥30 mg/g				Family history of T2DM, HTN, or CVD. Polycystic ovary syndrome, sedentary life style, Advancing age and ethnic groups having high risk for DM or CVD Physician's judgement
Diagnosis	Impaired FPG+any 2 criteria	Any 3 criteria	Any 3 criteria	WC+any 2 criteria	Physician's judgement

T2DM: Type 2 diabetes mellitus, HTN: Hypertension, CVD: Cerebrovascular accident, DM: Diabetes mellitus, WC: Waist circumference. NCEP ATP III: National cholesterol education program adult treatment panel III, NCEP-R: NCEP-R: revised NCEP, IDF: International diabetes federation, AACE: American association of clinical endocrinologists, WHO: World Health Organization, WHR: Waist-to-hip ratio, BMI: Body mass index, HDL: High density lipoprotein

## RISK FACTORS<sup>80-95</sup>:

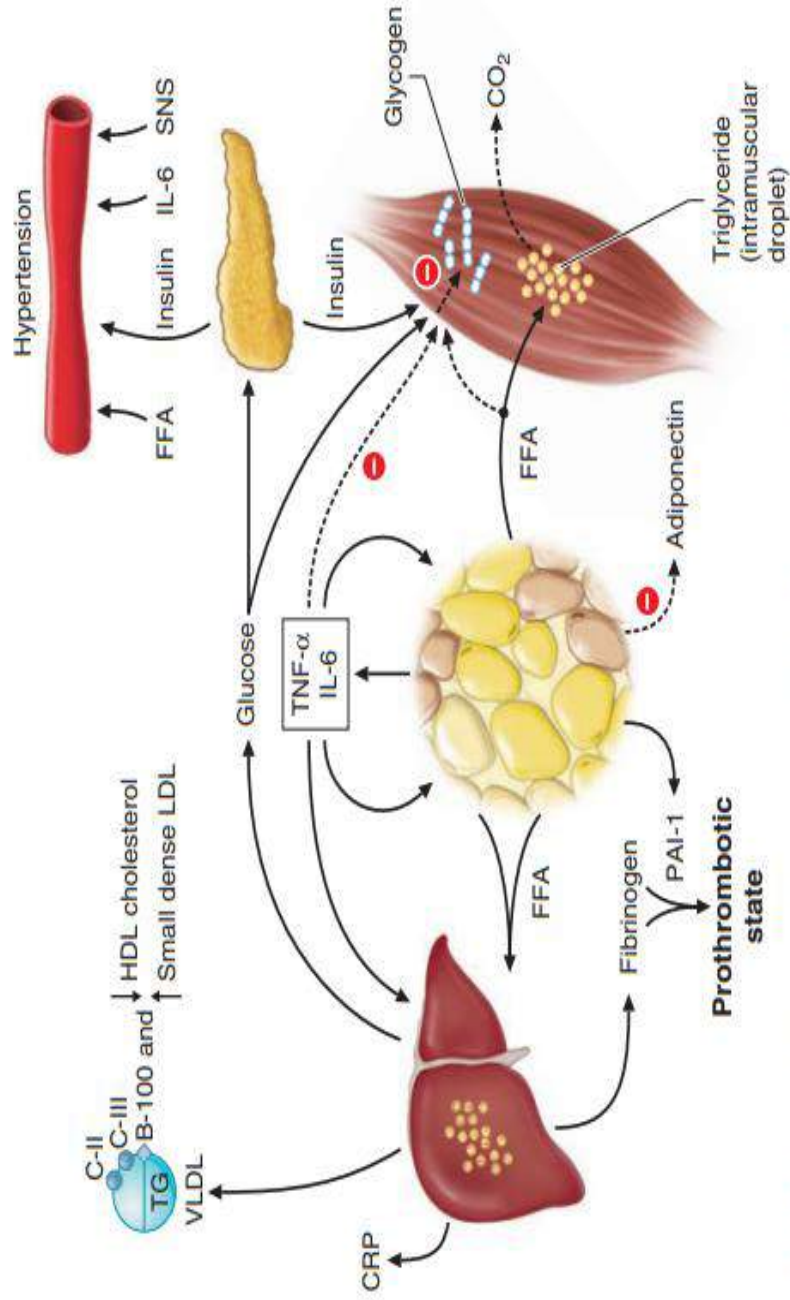
1. **CENTRAL OBESITY<sup>94</sup>**: One of the hallmarks of the syndrome is central adiposity, and its prevalence is due to the significant correlation between rising adiposity and waist circumference. Despite the significance of obesity, persons with normal weights might still have the metabolic syndrome and be insulin-resistant. For groups in Central America, Southeast Asia, and India, this phenotype is particularly pronounced.
2. **SEDENTARY LIFESTYLE**: Predictors of CVD events and the associated risk of mortality include physical inactivity and lower cardiorespiratory fitness. A sedentary lifestyle is linked to all components of the metabolic syndrome. Those who have screen time >4 h daily have a twofold greater risk of developing the

metabolic syndrome compared to those who do so for 1 h per day.

3. AGEING: Nearly 50% of Americans over the age of 60 are impacted by the metabolic syndrome, and women are more frequently affected than males at this age. In the majority of communities across the world, the prevalence of the condition is age dependent.
  
4. DIABETES MELLITUS<sup>85,86</sup>: The metabolic syndrome is present in the vast majority (about 75%) of people with type 2 diabetes or impaired glucose tolerance. The prevalence of CVD is greater in these groups when the metabolic syndrome is present compared to individuals with type 2 diabetes or impaired glucose tolerance who do not have the syndrome.

<b>Metabolic Syndrome: NCEP-ATPIII Criteria</b> [JAMA 2001; 285: 2486–97.] [Circulation 2004; 109: 433–8. Circulation 2005; 112: 2735–52.]	
At least three of the following five items:	
Glucose or Insulin abnormalities as defined by:	Fasting plasma glucose $\geq 100$ mg/dL
Central Obesity as defined by:	Waist circumference $\geq 102$ cm (40 in) (men) $\geq 88$ cm (35 in) (women)
Dyslipidemia as defined by:	Triglycerides $\geq 150$ mg/dL
Dyslipidemia as defined by:	HDL $< 40$ mg/dL (men), HDL $< 50$ mg/dL (women)
Elevated Blood Pressure as defined by:	BP $\geq 130/85$ mmHg
<i>Patients must either meet the indicated criteria for a risk factor OR be on drug treatment for that risk factor.</i>	

5. LIPODYSTROPHY: The metabolic syndrome is generally linked to conditions of lipodystrophy. Furthermore, the metabolic syndrome is a typical presentation in these individuals. Severe insulin resistance and many of the symptoms of the metabolic syndrome may result from both genetic lipodystrophy (such as Berardinelli-Seip congenital lipodystrophy and Dunnigan familial partial lipodystrophy) and acquired lipodystrophy (such as HIV-related lipodystrophy and in anti retro viral related lipodystrophy).



**FIGURE 408-2 Pathophysiology of the metabolic syndrome.** Free fatty acids (FFAs) are released in abundance from an expanded adipose tissue mass. In the liver, FFAs result in increased production of glucose and triglycerides and secretion of very-low-density lipoproteins (VLDLs). Associated lipid/lipoprotein abnormalities include reductions in high-density lipoprotein (HDL) cholesterol and an increased low-density lipoprotein (LDL) particle number. FFAs also reduce insulin sensitivity in muscle by inhibiting insulin-mediated glucose uptake. Associated defects include a reduction in glucose partitioning to glycogen and increased lipid accumulation in triglyceride (TG). The increase in circulating glucose, and to some extent FFAs, increases pancreatic insulin secretion, resulting in hyperinsulinemia. Hyperinsulinemia may result in enhanced sodium reabsorption and increased sympathetic nervous system (SNS) activity and contribute to hypertension, as might higher levels of circulating FFAs. The proinflammatory state is superimposed and contributory to the insulin resistance produced by excessive FFAs. The enhanced secretion of interleukin 6 (IL-6) and tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ) produced by adipocytes and monocyte-derived macrophages results in more insulin resistance and lipolysis of adipose tissue triglyceride stores to circulating FFAs. IL-6 and other cytokines also enhance hepatic glucose production, VLDL production by the liver, hypertension, and insulin resistance in muscle. Insulin resistance also contributes to increased triglyceride accumulation in the liver (nonalcoholic fatty liver disease). Cytokines and FFAs also increase hepatic production of fibrinogen and adipocyte production of plasminogen activator inhibitor 1 (PAI-1), resulting in a pro-thrombotic state. Higher levels of circulating cytokines stimulate hepatic production of C-reactive protein (CRP). Reduced production of the anti-inflammatory and insulin-sensitizing cytokine adiponectin is also associated with the metabolic syndrome. (Reproduced with permission from RH Eckel et al: *The metabolic syndrome*. Lancet 365:1415, 2005.)

# *Methodology*

## **OBJECTIVE:**

### **Primary objective:**

Among the patients presenting to Department of Medicine, Government Stanley Medical College hospital between April 2021 and March 2022.

- 1.To study the prevalence of metabolic syndrome in young hypertensive adults.

### **Secondary objective:**

- 1.To study the compliance to drugs and lifestyle modifications in hypertensive patients with and without metabolic syndrome
- 2.To explore the reasons for poor compliance to treatment (Drugs and life style modifications) among patients with metabolic syndrome

### **Study design**

The study design will be a mixed methods study. Quantitative research component will be followed by the qualitative research method. This particular type was chosen as the study findings of the qualitative method will be explained by the in-depth interviews administered to the part of purposively selected study participants.

- i) Quantitative component of it will be a cross sectional study that addresses the first and second objectives of the study.
- ii) Descriptive qualitative study using in-depth interview

will be done to study the third objective.

**Study setting** : Department of Medicine, Govt Stanley Medical college & Hospital

**Study period** : April 2021 - March 2022

**Study Population** : **Inclusion Criteria:**

1. Individuals of age 20-45yrs with Systemic hypertension (BP>140/90 as per 2020 ACC- AHA guidelines) reporting to Department of General Medicine.

**Exclusion criteria:**

1. Patients with age less than 20 yrs and above 45 yrs
2. Patients with previously diagnosed diabetes mellitus.
3. Patients with secondary hypertension
4. Patients with secondary cause of obesity
5. Patients on steroidal or other medications likely to cause elevated plasma glucose
6. Any acute illness
7. Pregnant women
8. Patients not willing to participate in the study
9. Patients not giving consent for the study.

**Sample size** : We estimated sample size using the formula

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

$Z_{\alpha}$  is 1.96 for 95% confidence level

P is the anticipated frequency

d is the absolute precision

Sample size was calculated based on a previous study done by Osuji et al using the formula  $Z^2 pq/L^2$  Where p is 31.2%, q is 68.8% L ( Absolute precision of 5) the sample size was 330 and adding 10% for non response rate the final sample size was rounded off to 365.

For qualitative component 8 to 10 patients from the poorly compliant group will be selected for in depth interview

**Sampling technique** : We will include eligible individuals by stratified random sampling.



## **OPERATIONAL DEFINITION:**

**Systemic Hypertension:** individuals with BP >140/90mmHg

**Metabolic Syndrome :**

**(NCEP ATP III )**

Three or more of the following:-

- 1) Central obesity: - waist circumference >102 cm (M), >88 cm (F)
- 2) Hypertriglyceridemia: - Triglycerides  $\geq$  150 mg/dl or specific medication.
- 3) Low HDL cholesterol : - <40 mg/dl (male), < 50 mg/dl (female) or specific medication.
- 4) Hypertension: -  $\geq$  130 mm of Hg Systolic or  $\geq$  85 mm of Hg Diastolic
- 5) Fasting plasma glucose level: -  $\geq$  100 mg/dl or specific medication or previously diagnosed type 2 diabetes mellitus

## **ETHICAL CONSIDERATION:**

Patients will be given a Patient Information sheet and informed consent form that will be verbally explained to the patients orally in a language they understand.

Confidentiality will be maintained.

## **PERMISSION:**

Written permission were obtained from the Heads of Department of General Medicine, Pathology and Biochemistry.

## **ETHICAL COMMITTEE:**

Permission from Institutional Ethical Committee (IEC) is obtained.

**DATA COLLECTION:**

We will start the study after the approval from IEC. After obtaining written and informed consent from the adults presenting with hypertension, we will interview them with the structured questionnaire to obtain information on socio-demographic factors, clinical history, physical examination findings, adherence to medication. We will enter the data in MS Excel. . In-depth interview (IDI) will be conducted among the purposively selected non compliant participants for the qualitative study component. After obtaining their consent, the interviews will be audio recorded for the purpose of doing content analysis.

**CONFLICT OF INTEREST:** None to declare

**FINANCIAL DISCLOSURE:** None to declare

## DATA ANALYSIS:

VARIABLES	ANALYSIS PLAN
<p><b>DEPENDANT VARIABLES:</b></p> <ul style="list-style-type: none"><li>• Proportion of patients with metabolic syndrome among young hypertensives</li><li>• Compliance status of the participants of the study</li></ul>	<ul style="list-style-type: none"><li>• Frequency and proportion</li><li>• Unpaired T test will be used for continuous variables and chi square test will be used for categorical variables</li></ul>
<p><b>INDEPENDENT VARIABLES</b></p> <ul style="list-style-type: none"><li>• Age, height, weight, waist circumference, Triglyceride levels, HDL levels, fasting blood sugar, post prandial blood sugar</li><li>• Stress, compliance to drug, physical activity, diet, tobacco use, alcohol consumption</li><li>• Comparison of compliance of patients with and without metabolic syndrome</li><li>• Reasons for poor compliance to treatment</li></ul>	<ul style="list-style-type: none"><li>• Mean and standard deviation</li><li>• Frequency and proportion</li><li>• Mann whitney U test</li><li>• Guidelines by UCLA center for health policy research</li></ul>

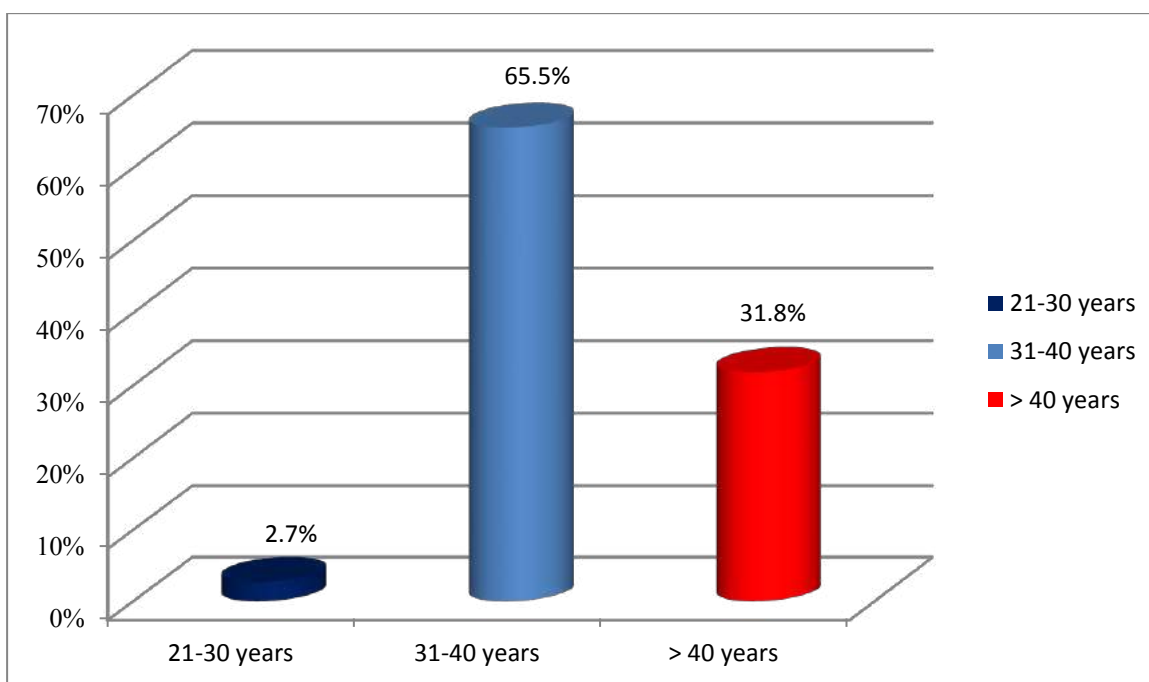
# *Observation and Results*

**Table 1: Age wise distribution of the study participants (N-365)**

Age	Frequency	Percentage (%)
21-30 years	10	2.7
31-40 years	239	65.5
> 40 years	116	31.8
<b>Total</b>	<b>365</b>	<b>100</b>

In our study majority of the participants enrolled were of age between 31 to 40 years (65.5%) followed by 41 to 45 years (31.8%). In our study we have enrolled 10 participants of age between 21 to 30 years.

**Figure 1: Age wise distribution of the study participants**



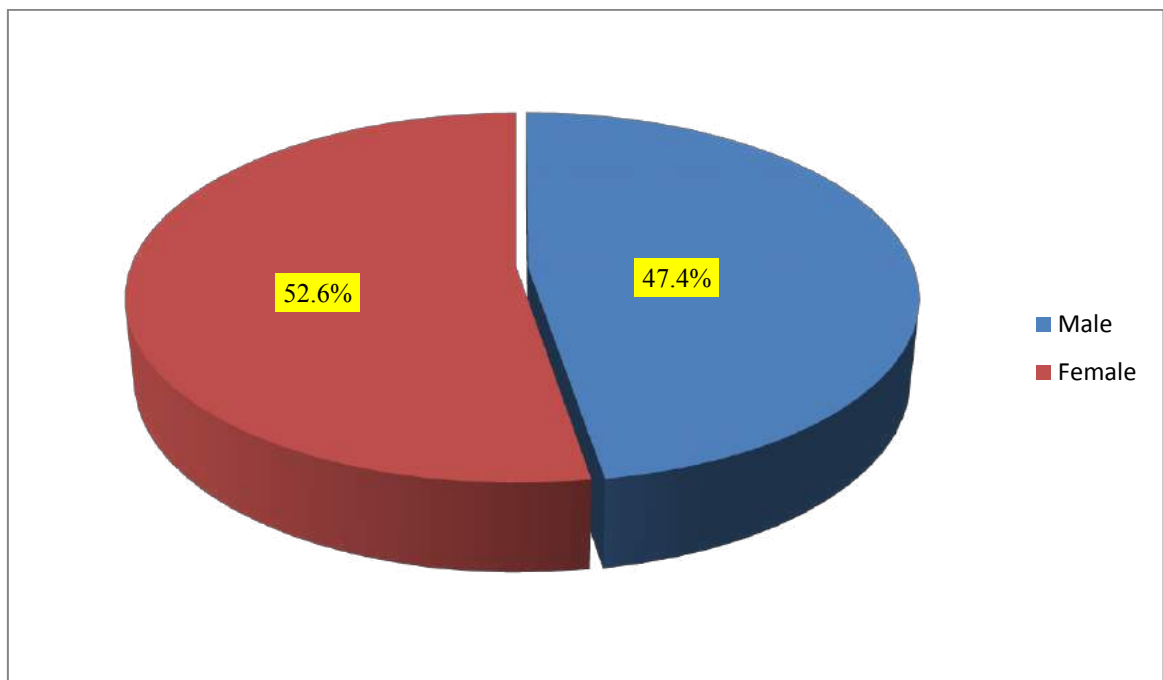
**Table 2: Sex wise distribution of the study participants (N-365)**

<b>Sex</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>Male</b>	173	47.4
<b>Female</b>	192	52.6
<b>Total</b>	365	100

Of the total 365 participants enrolled 52.6% were female and 47.4% were male subjects.

We see that prevalence of hypertension is slightly more in female population as per our study

**Figure 2: Sex wise distribution of the study participants**

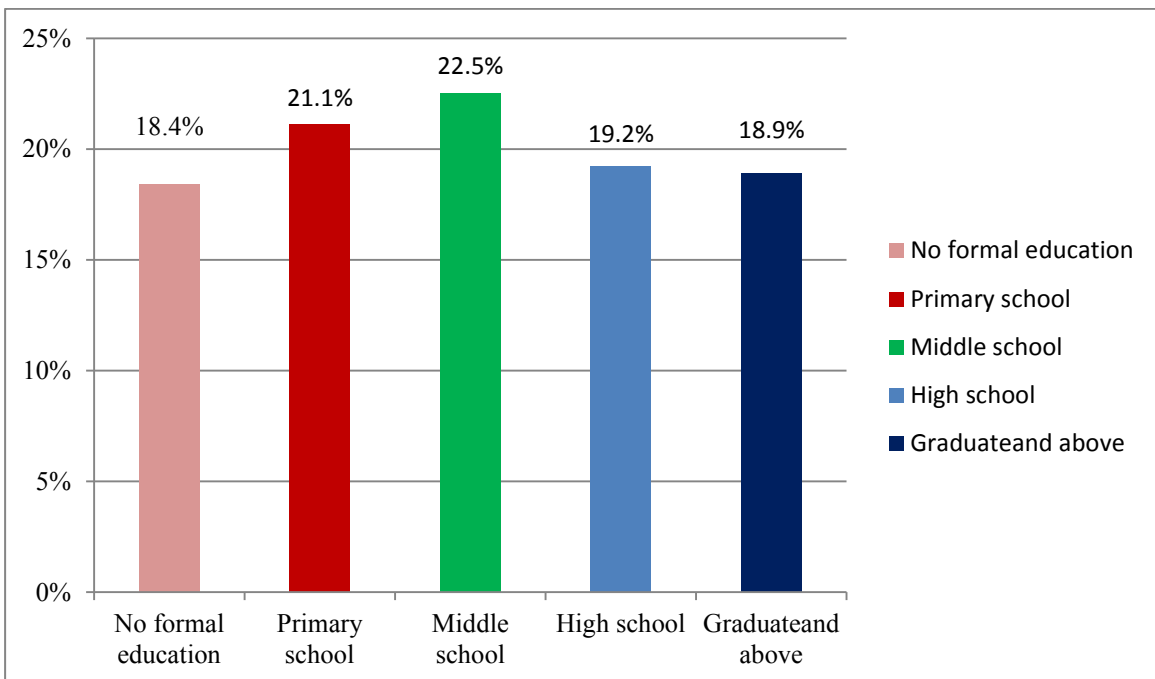


**Table 3: Educational status of the study participants (N-365)**

<b>Education</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>No formal education</b>	67	18.4
<b>Primary school</b>	77	21.1
<b>Middle school</b>	82	22.5
<b>High school</b>	70	19.2
<b>Graduate and above</b>	69	18.9
<b>Total</b>	365	100

Of the total participants, majority of them have completed middle schooling and primary schooling alone. 67 participants had no formal education and 69 participants were graduates.

**Figure 3: Educational status of the study participants**

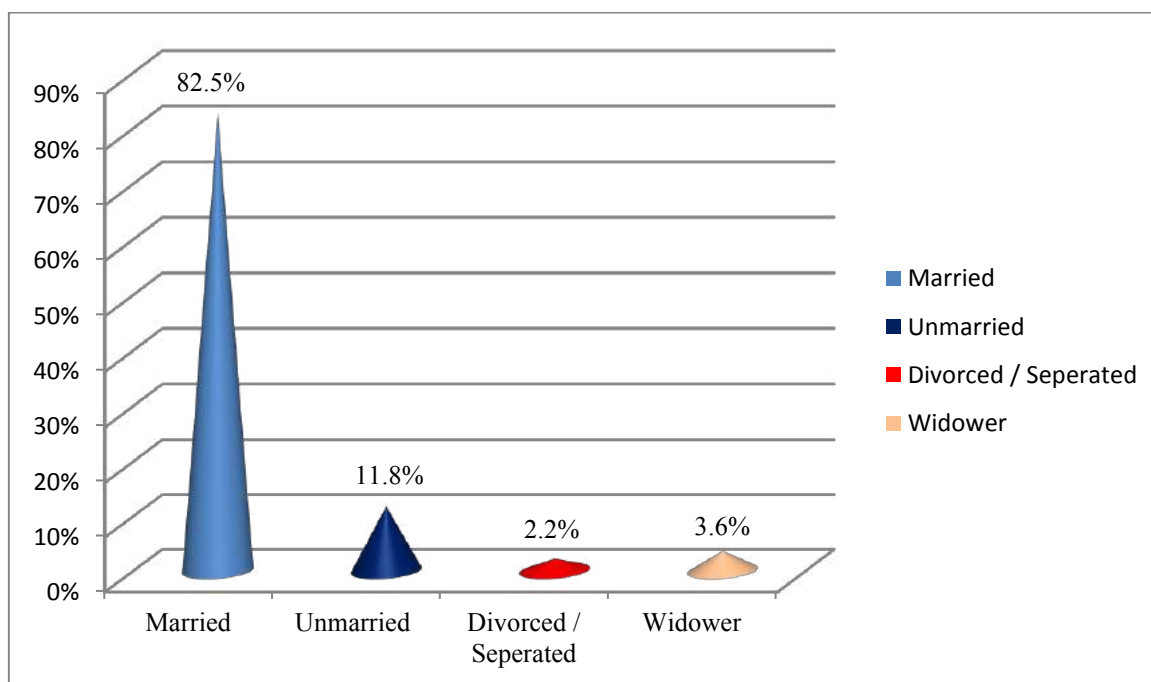


**Table 4: Marital status of the study participants (N-365)**

<b>Marital status</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>Married</b>	301	82.5
<b>Unmarried</b>	43	11.8
<b>Divorced / Seperated</b>	8	2.2
<b>Widower</b>	13	3.6
<b>Total</b>	365	100

Of the total 365, 301 were married; 43 were unmarried, 8 were divorced, 13 were widowed.

**Figure 4: Marital status of the study participants**



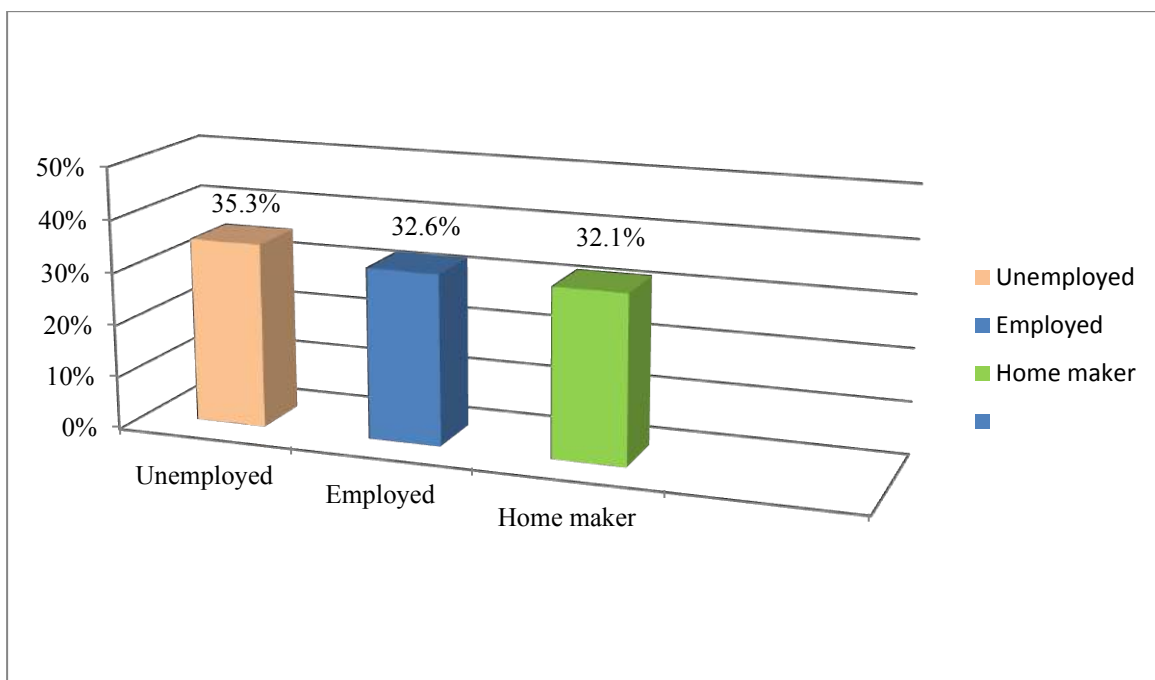


**Table 5: Occupation of the study participants (N-365)**

<b>Occupation</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>Unemployed</b>	129	35.3
<b>Employed</b>	119	32.6
<b>Home Maker</b>	117	32.1
<b>Total</b>	365	100

In our study, 129 participants were unemployed, 119 were employed and 117 were home makers with their spouse working on.

**Figure 5: Occupation of the study participants**

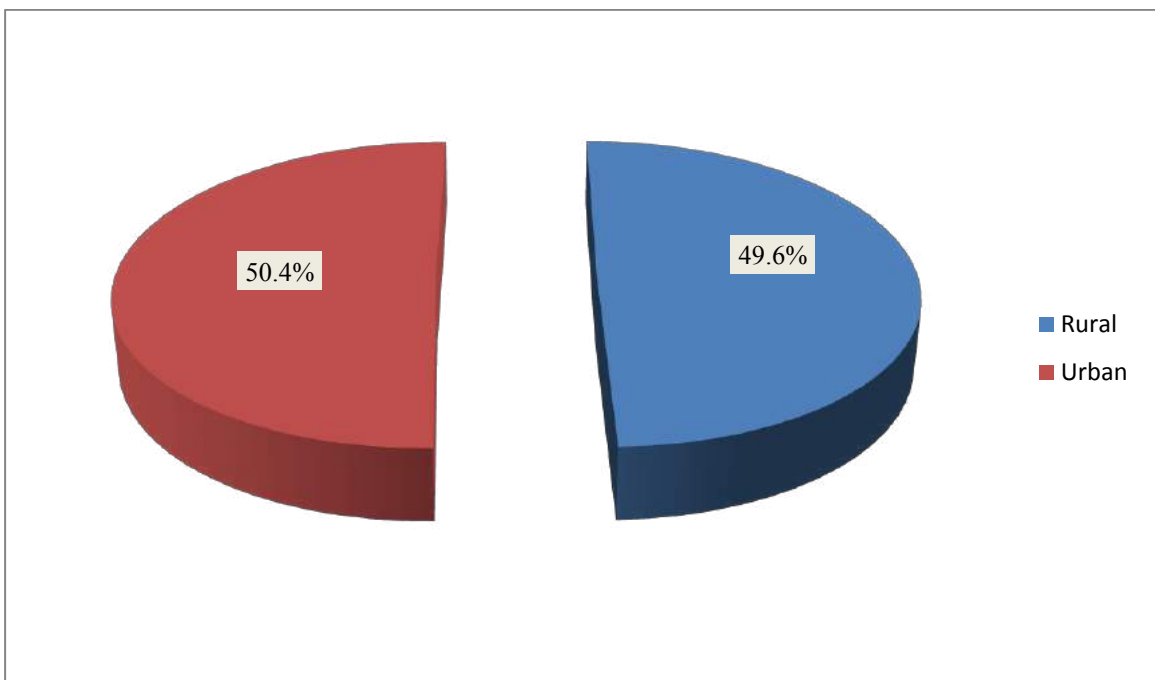


**Table 6: Place of residence of the study participants (N-365)**

Place of residence	Frequency	Percentage (%)
<b>Rural</b>	181	49.6
<b>Urban</b>	184	50.4
<b>Total</b>	365	100

Our study has 181 participants from the rural side and 184 participants from the urban side.

**Figure 6: Place of residence of the study participants**

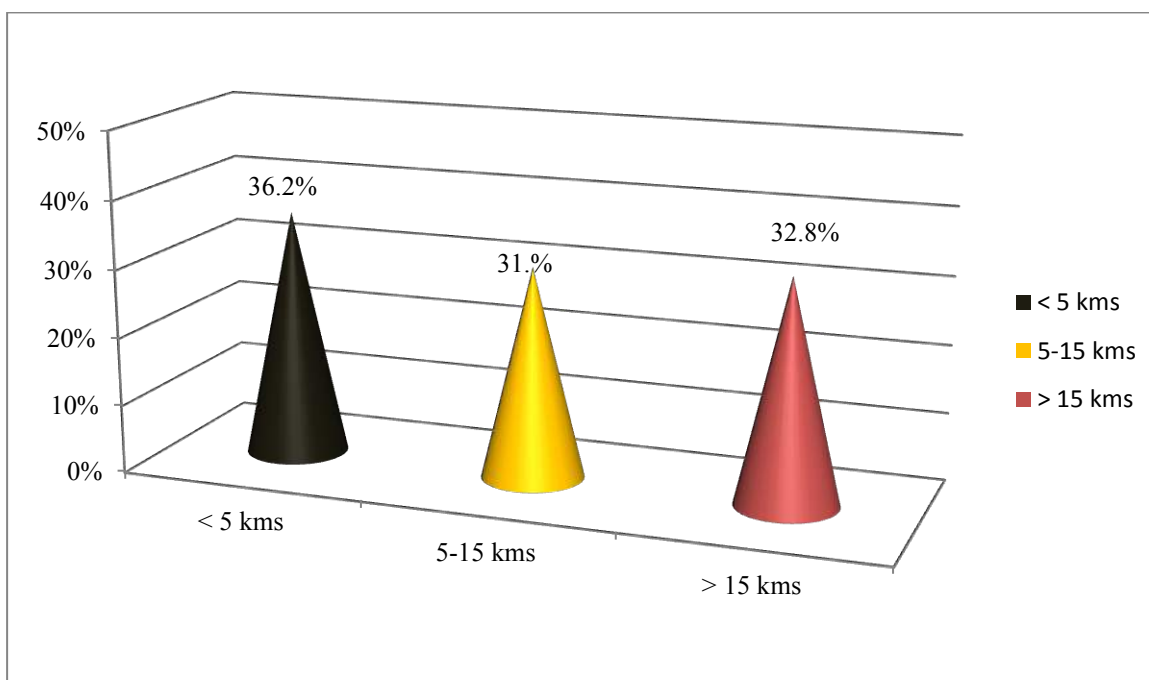


**Table 7: Distance travelled from house to reach the hospital among the study participants (N-365)**

Distance	Frequency	Percentage (%)
< 5 km	132	36.2
5-15 km	113	31
> 15 km	120	32.8
<b>Total</b>	<b>365</b>	<b>100</b>

Around 132 participants have the hospital within 5 km from their home while 113 participants have their home in 5 – 15 km radius of hospital. Also 120 patients have the hospital from which they are getting drugs around 15 km from their residence

**Figure 7: Distance travelled from house to reach the hospital among the study participants**

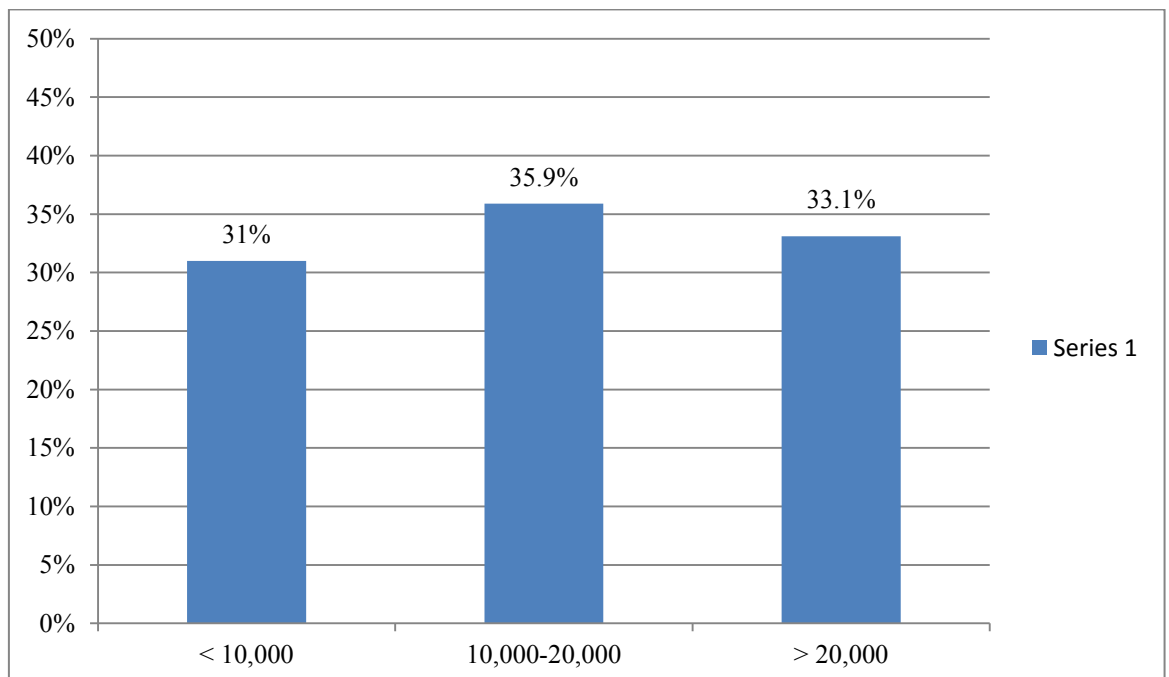


**Table 8: Income per month among the study participants (N-365)**

<b>Income per month</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>&lt; 10,000</b>	113	31
<b>10,000-20,000</b>	131	35.9
<b>&gt; 20,000</b>	121	33.1
<b>Total</b>	365	100

While enquiring about the family income status around 131 persons have their family income between 10000 and 20000. 113 patients have their family income less than 10000 and 121 have their family income above 20000.

**Figure 8: Income per month among the study participants**

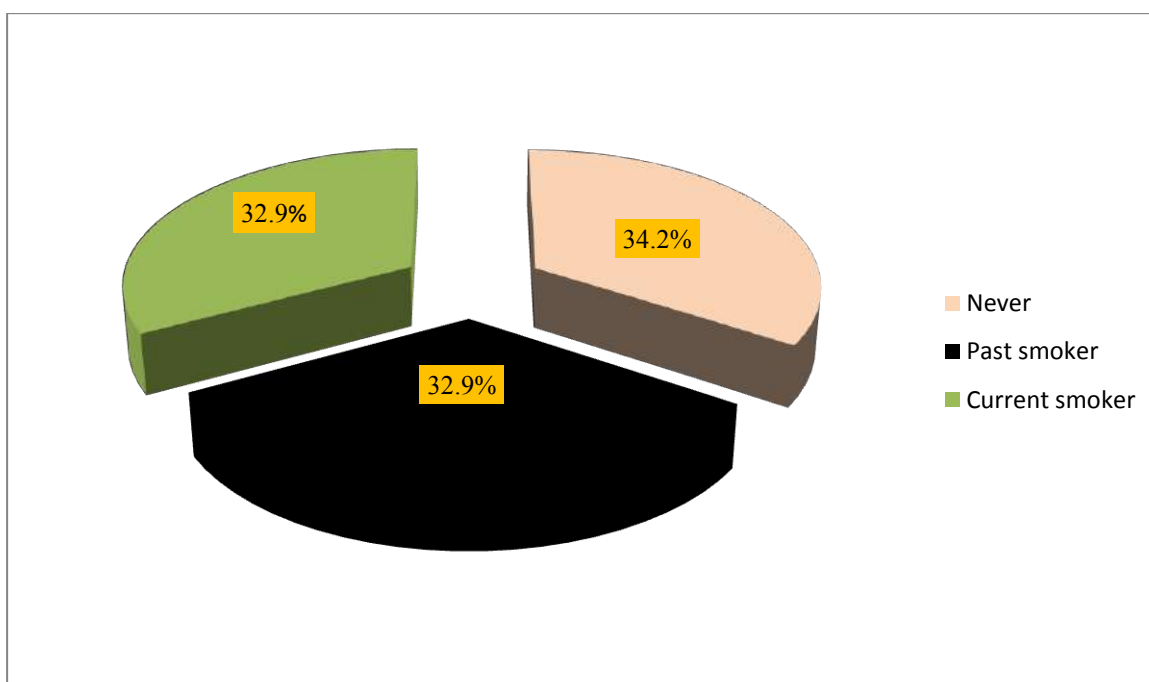


**Table 9: Smoking history (Active and Passive) among the study participants**

Smoking	Frequency	Percentage (%)
Never	125	34.2
Past smoker	120	32.9
Current smoker	120	32.9
<b>Total</b>	<b>365</b>	<b>100</b>

We have taken both active and passive smoking status into consideration as both have adverse effects on patients. 125 participants have never smoked in their lifetime. 240 were exposed to either active or passive smoking. Among them 120 have abstained from smoking for the past one month.

**Figure 9: Smoking (Active and Passive) among the study participants**

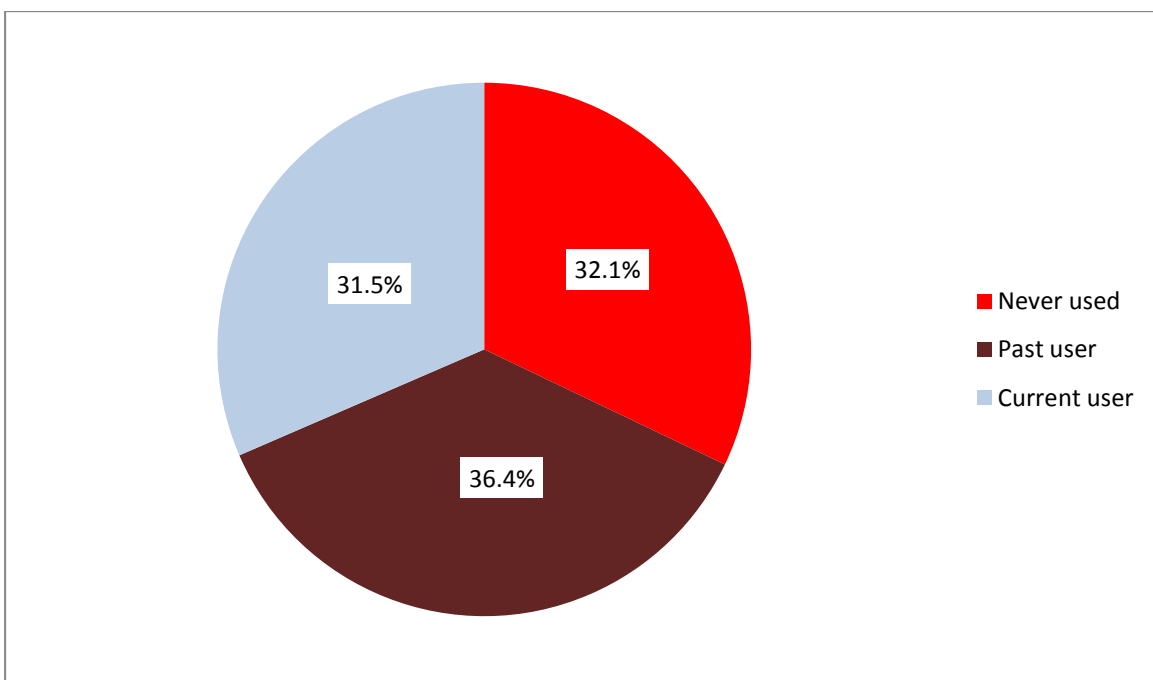


**Table 10: Alcohol use among the study participants**

<b>Alcohol use</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>Never used</b>	117	32.1
<b>Past user</b>	133	36.4
<b>Current user</b>	115	31.5
<b>Total</b>	365	100

Alcohol consumption has proven adverse effects on cardiovascular system. We have taken into account of people consuming all type of alcohol including country liquor. To our surprise 68.5% of the participants were consuming alcohol now or then. 32% of participants have never been exposed to alcohol.

**Figure 10: Alcohol use among the study participants**

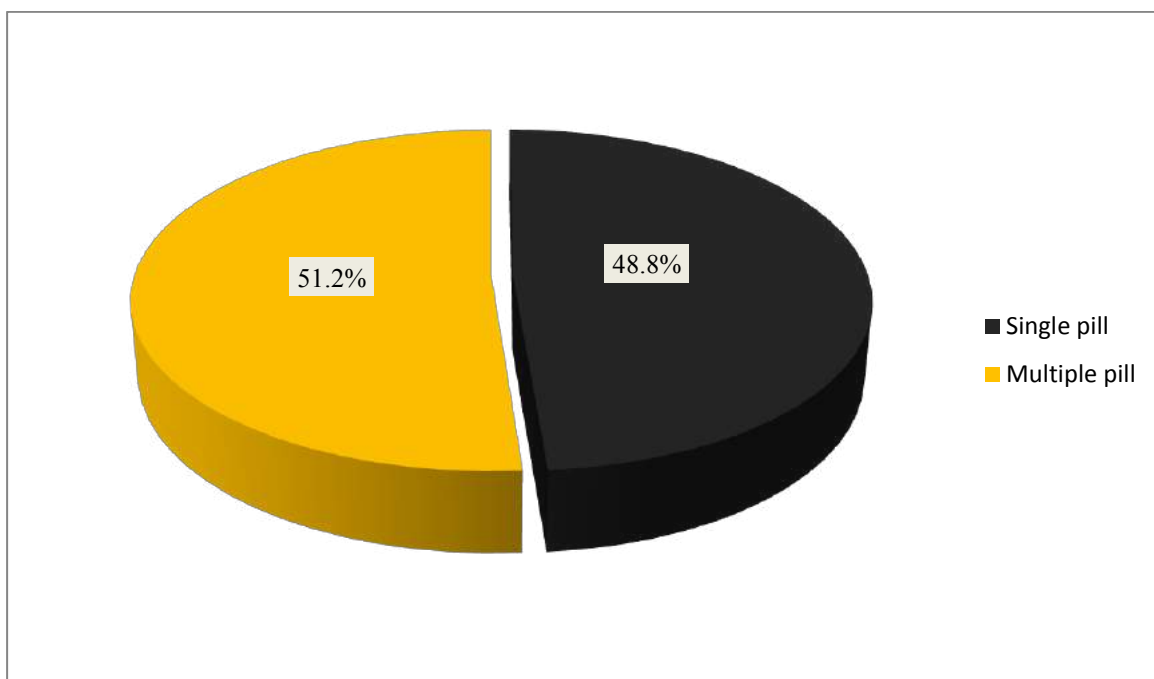


**Table 11: Type of medication use among the study participants (N-365)**

Type of medication	Frequency	Percentage (%)
Single pill	178	48.8
Multiple pill	187	51.2
<b>Total</b>	<b>365</b>	<b>100</b>

In our study we have recorded whether the participants have been prescribed medications. All of them were prescribed oral medications. 178 were prescribed single pill and 187 were prescribed multiple pills for anti hypertensive therapy. It is found that multiple pill intake reduces the compliance of treatment.

**Figure 11: Type of medication use among the study participants**

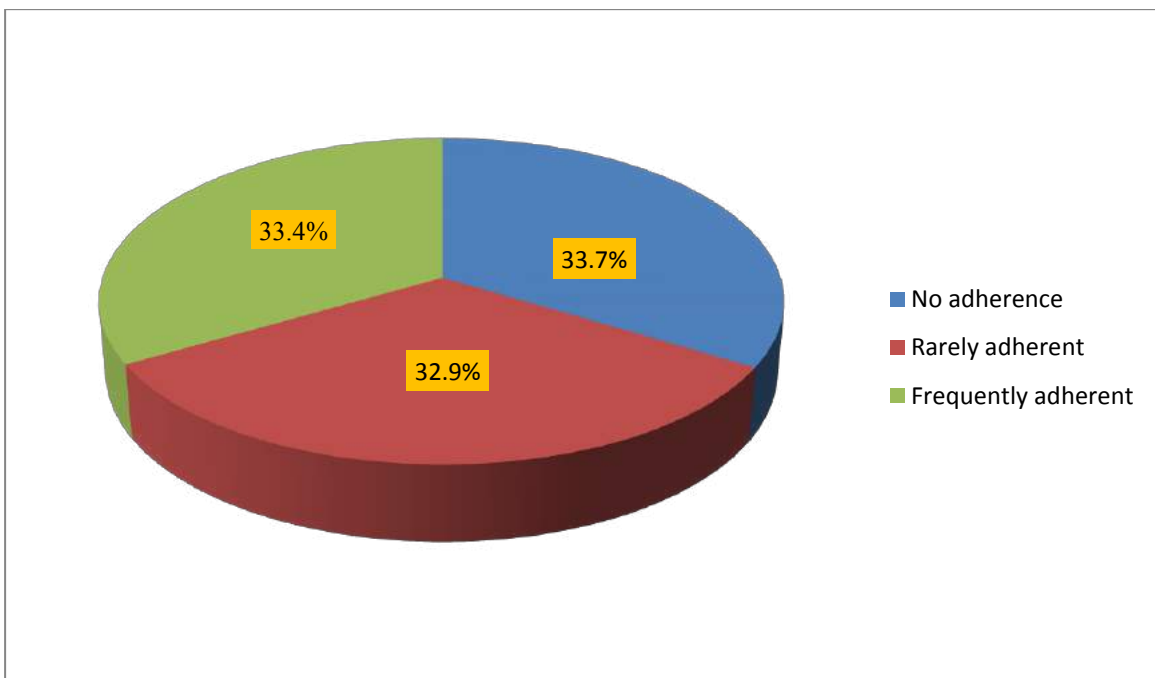


**Table 12: Compliance to lifestyle modification among the study participants (N-365)**

<b>Compliance to lifestyle modification</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>No adherence</b>	123	33.7
<b>Rarely adherent</b>	120	32.9
<b>Frequently adherent</b>	122	33.4
<b>Total</b>	365	100

Lifestyle modifications form the important measure in treatment of hypertension. Without lifestyle modifications any drug therapy will be inefficient. Infact lifestyle modification is the key pillar of blood pressure treatment. 123 participants of our study don't adhere to any of the lifestyle modifications advised. 120 participants were rarely adherent. Only 122 of the total 365 participants were frequently adherent to the lifestyle modifications.

**Figure 12: Compliance to lifestyle modification among the study participants**





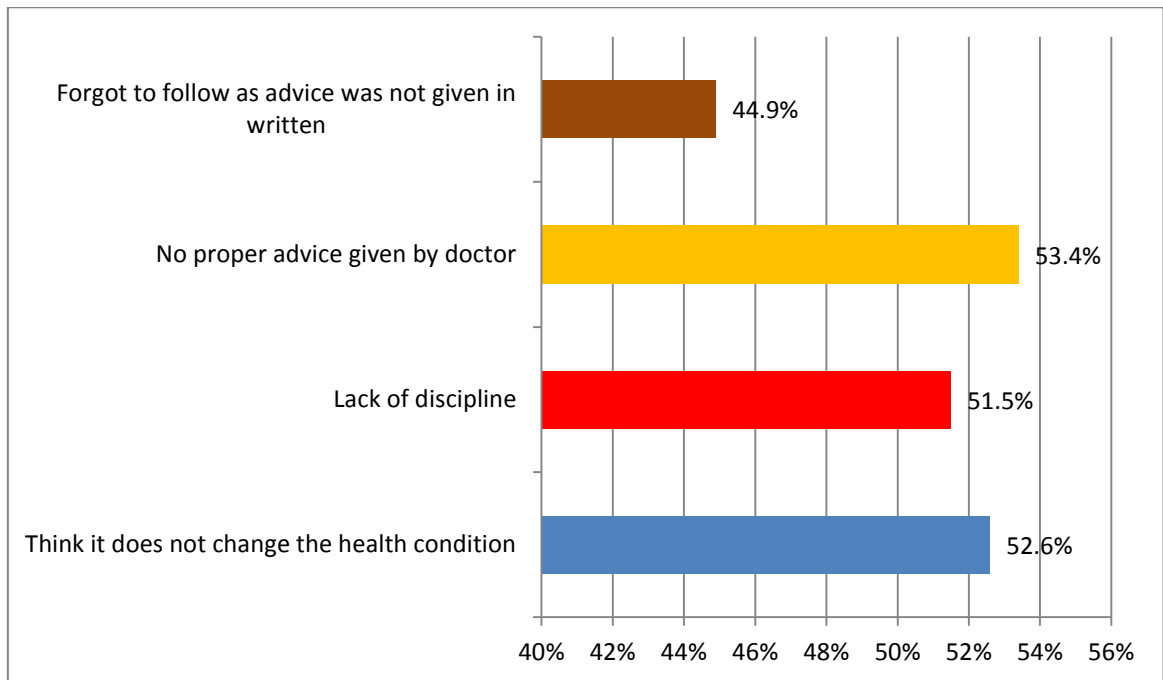
**Table 13: Reasons for poor adherence to dietary advice among the study participants**

Reasons for poor adherence to dietary advice	Frequency	Percentage (%)
<b>Lack of discipline</b>	188	51.5
<b>No proper advice given by doctor</b>	195	53.4
<b>Forgot to follow as advice was not given in written</b>	164	44.9
<b>Think it does not change the health condition</b>	192	52.6

**\* Multiple responses allowed**

In our study participants were enquired about the reason for poor adherence to dietary advice. It is surprising to note that 52.6% of the participants were of the opinion that dietary modification does not change the health condition. 195 participants said that no proper advice was given to them regarding dietary modification while 164 said that they forgot the dietary advice as it was not given in written. 188 participants admitted that they were unable to follow the dietary advice due to lack of discipline.

**Figure 13: Reasons for poor adherence to dietary advice among the study participants**

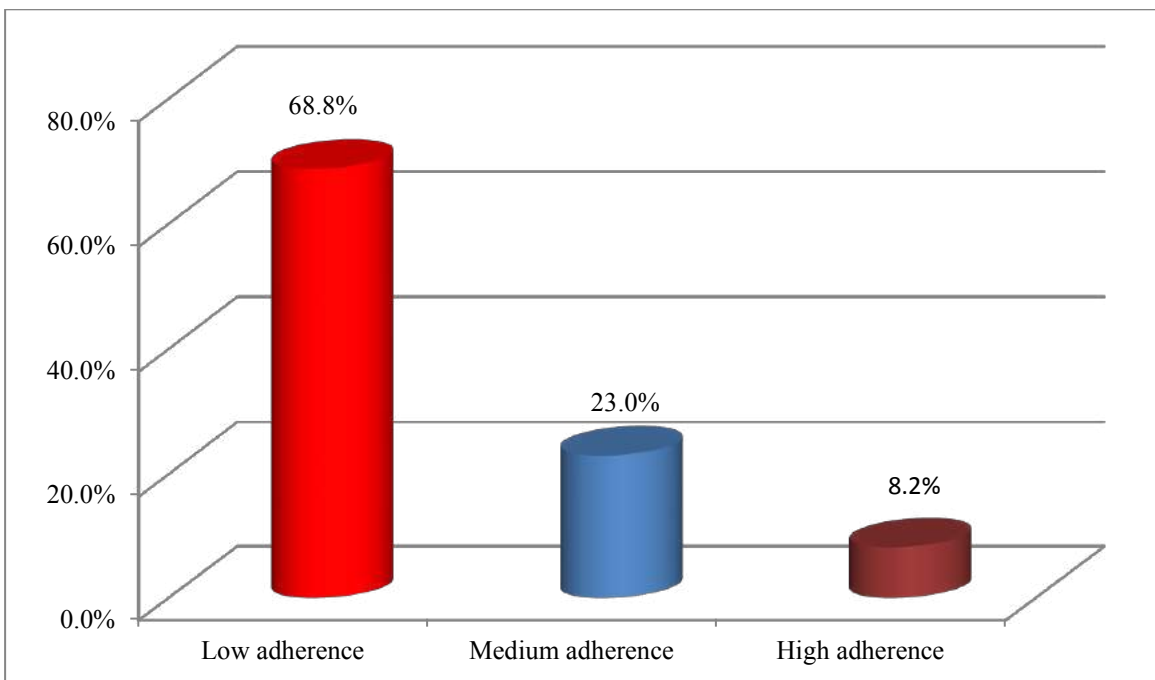


**Table 14: Adherence to hypertension medication among the study participants (N-365)**

<b>Adherence to medication (MMAS Score)</b>	<b>Frequency</b>	<b>Percentage (%)</b>
Low (<6)	251	68.8
Medium (6 & 7)	84	23
High (equal to 8)	30	8.2
<b>Total</b>	<b>365</b>	<b>100</b>

Our study participants were assessed for drug compliance using the Morisky Medication Adherence Score. It contains 8 questions and based upon response numerical scoring is assigned to each question. It is found that 251 participants comprising of about 68.8% of participants were of low adherence to anti hypertensive therapy. 84 participants were of medium adherence. Only 30 participants out of 365 were highly adherent to anti hypertensive medications.

**Figure 14: Adherence to hypertension medication among the study participants**

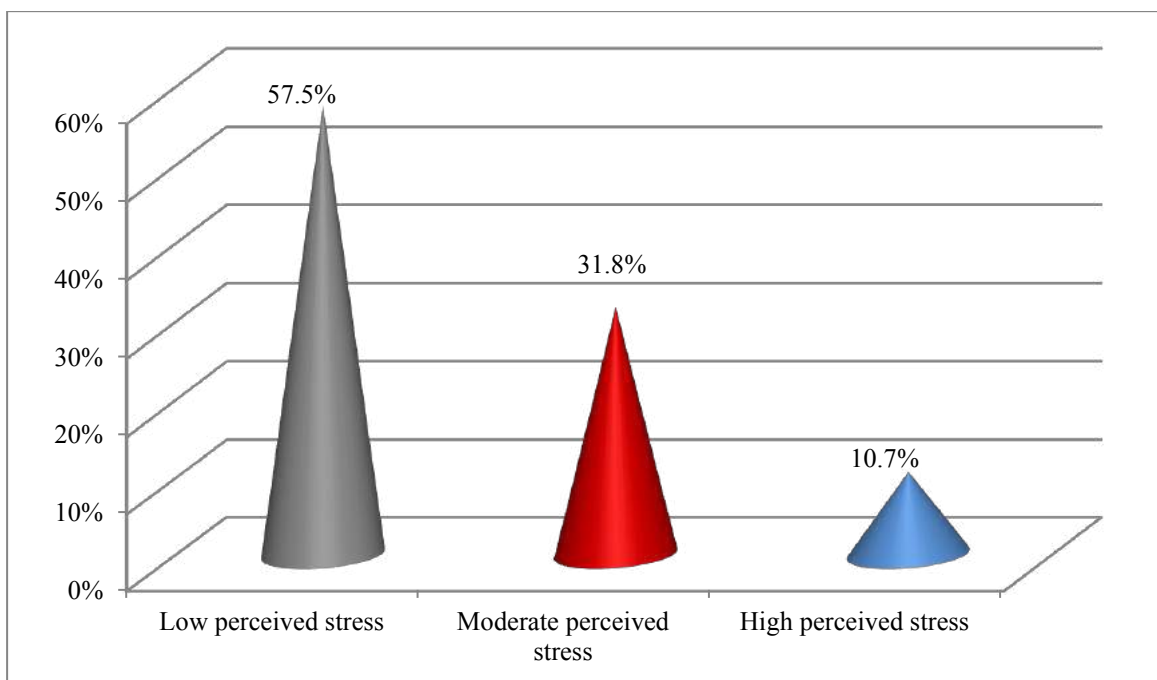


**Table 15: Perceived stress prevalence among the study participants (N-365)**

Perceived stress level (PSS score)	Frequency	Percentage (%)
Low (0-13)	210	57.5
Moderate (14-26)	116	31.8
High (27-40)	39	10.7
<b>Total</b>	<b>365</b>	<b>100</b>

Environmental factors – most importantly stress factor contribute towards the development of hypertension. In our study Perceived stress scale is employed for stress assessment. 210 people are with low stress while 116 participants are with moderate stress. Around 10.7% - 39 are with high stress. For the people with moderate and high amount of stress Psychiatry counselling were given.

**Figure 15: Perceived stress prevalence among the study participants**

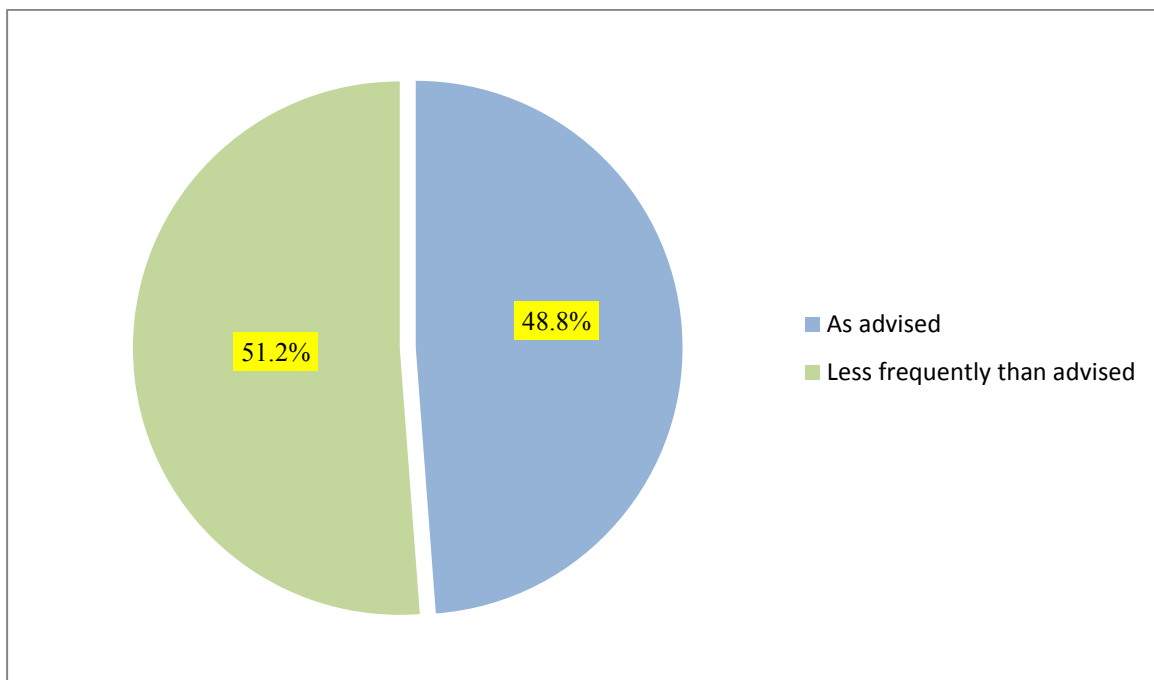


**Table 16: Adherence to follow up for treatment among the study participants (N-365)**

<b>Adherence to follow-up</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>As advised</b>	178	48.8
<b>Less frequently than advised</b>	187	51.2
<b>Total</b>	365	100

Adherence to treatment follow up visits were recorded in our study. 178 participants adhere to the treatment follow up schedule as advised by the doctor. 187 participants come for review less frequently than advised by doctor. In spite of poor drug adherence at home, a high proportion of people come to review on time because next visit date is clearly mentioned in the treatment record note given to the patients.

**Figure 16: Adherence to follow up for treatment among the study participants**



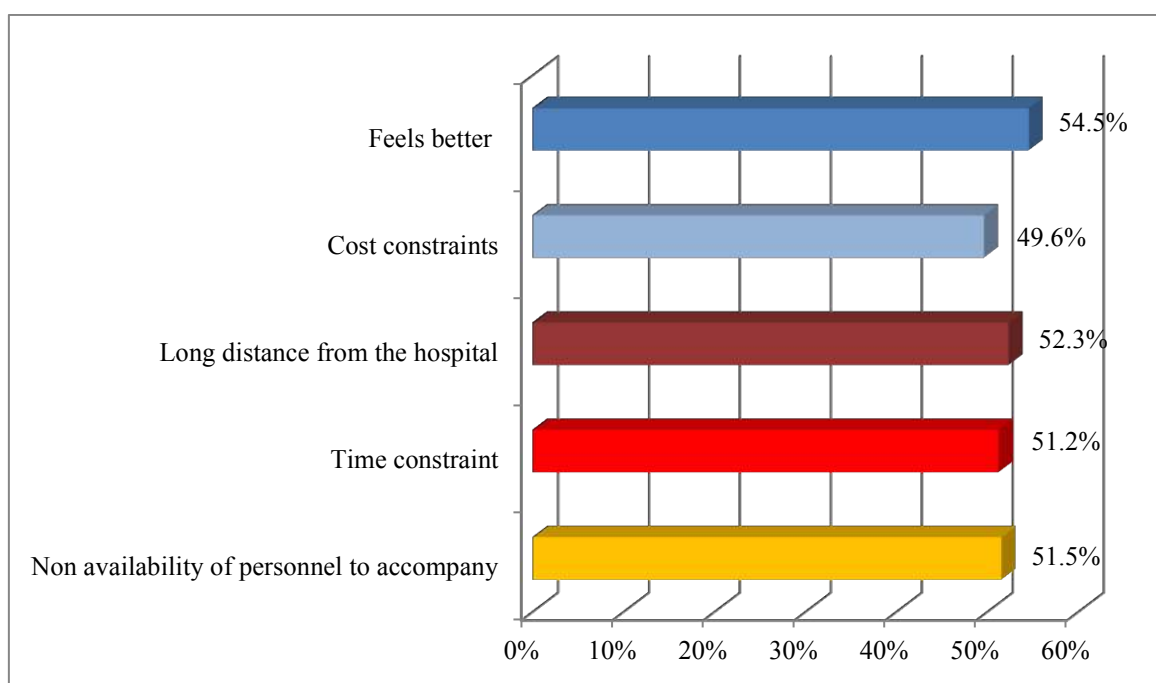
**Table 17: Reasons for poor adherence to follow up for treatment among the study participants (N-365)**

<b>Reasons for poor adherence to follow up</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>Non availability of personnel to accompany</b>	188	51.5
<b>Time constraint</b>	187	51.2
<b>Long distance from the hospital</b>	191	52.3
<b>Cost constraints</b>	181	49.6
<b>Feels better</b>	199	54.5

**\* Multiple responses allowed**

It is found that in our study more than half of study participants (51.2%) were coming to follow up less frequently than advised by doctor. Also those who frequently adhere to the treatment schedule sometimes miss the regular dates for follow up. While enquiring about the reasons for poor follow up long distance to hospital was the primary reason behind it. More than half of the participants have time constraints. For elderly people non availability of the personnel to accompany was the main reason. Cost constraints were also significant cause for poor follow up. To address these issues Mobile Medical Unit and Makkalai Thedi Maruthuvam scheme are implemented from Directorate of Public Health side.

**Figure 17: Reasons for poor adherence to follow up for treatment among the study participants**

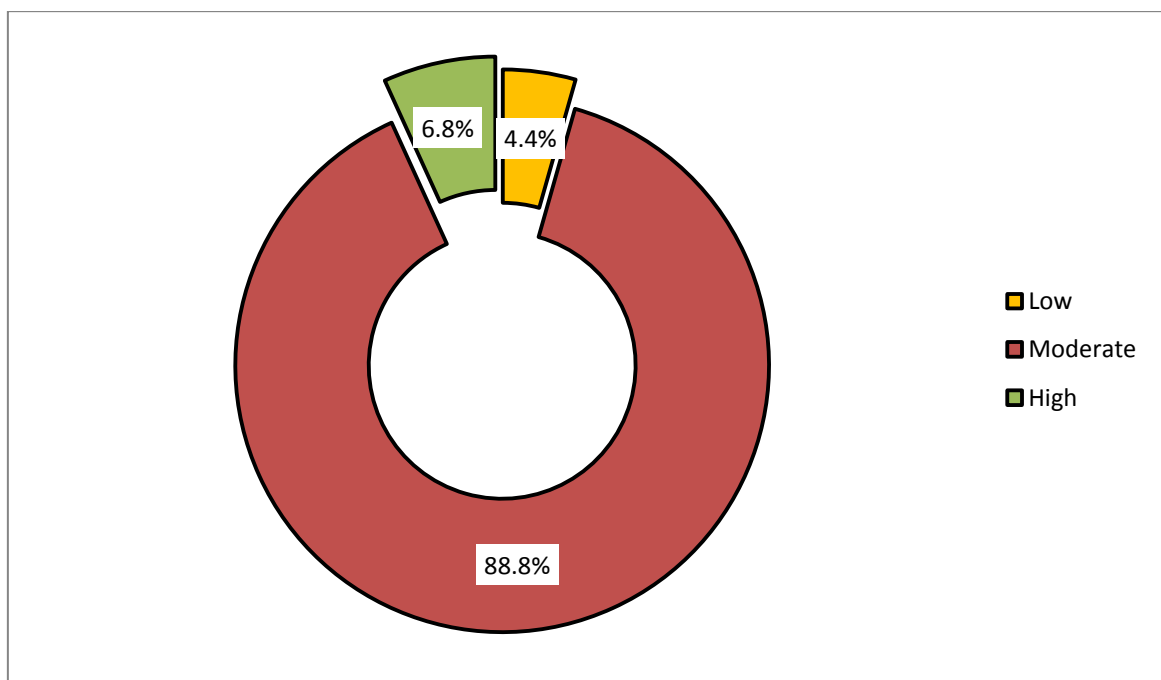


**Table 18: Level of physical activity as per IPAQ among the study participants (N-365)**

Level of physical activity	Frequency	Percentage (%)
Low (0-599 METs)	16	4.4
Moderate (600-2999 METs)	324	88.8
High ( $\geq 3000$ METs)	25	6.8
<b>Total</b>	<b>365</b>	<b>100</b>

International Physical Activity Questionnaire was employed to calculate the physical activity level among the study participants. Most of the participants 88.8% - 324 of the 365 participants perform moderate physical activity. 16 participants are with low physical activity while 25 participants (6.8%) are with high physical activity.

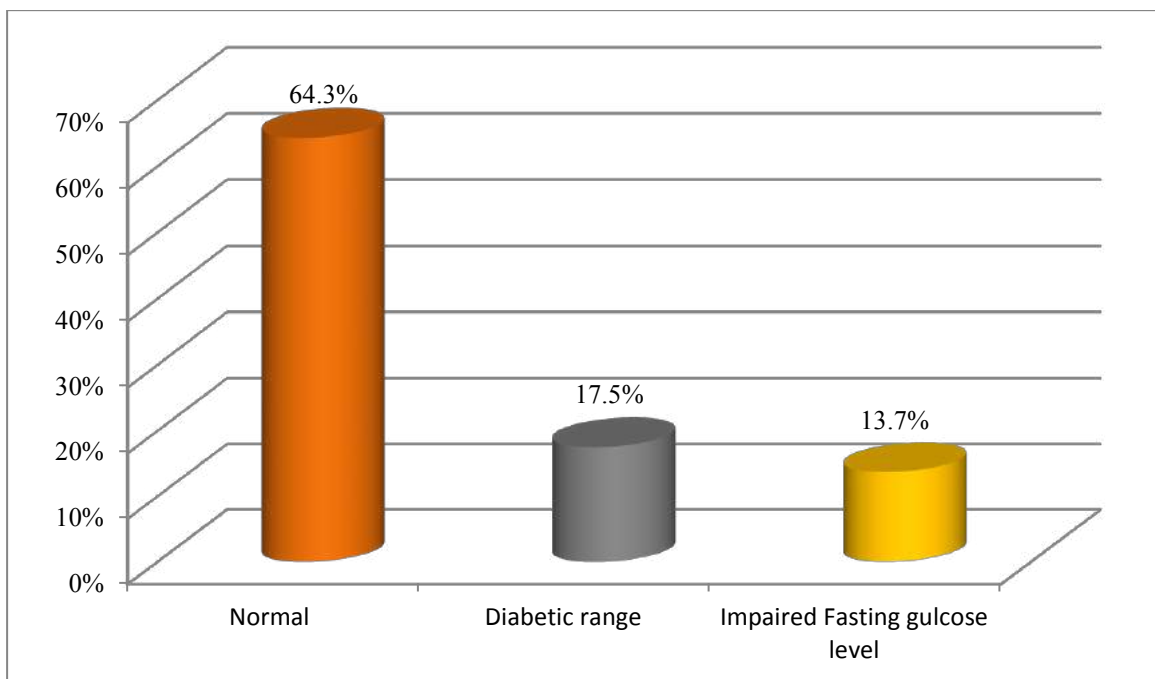
**Figure 18: Level of physical activity as per IPAQ among the study participants**



**Table 19: Distribution of continuous variables among the study participants (N-365)**

<b>Variable</b>	<b>Minimum</b>	<b>Maximum</b>	<b>Mean</b>	<b>Standard deviation</b>
<b>Age</b>	22	45	37.5	4.68
<b>Waist circumference</b>	78	105	91.9	7.05
<b>Triglyceride</b>	85	250	159.3	35.10
<b>HDL</b>	23	58	39.5	8.23
<b>SBP</b>	126	186	155.1	15.80
<b>DBP</b>	76	110	91.1	9.33
<b>FBS</b>	75	221	98.8	16.75

**Figure 19: Distribution of FBS among the study participants (N-365)**



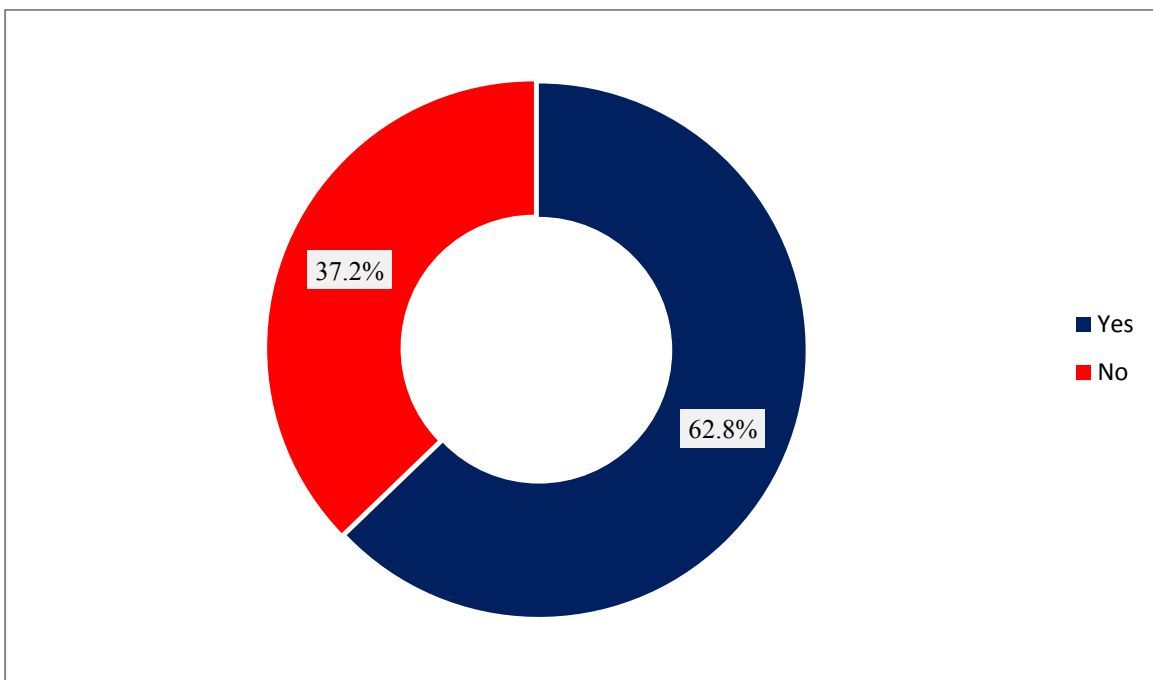


**Table 20: Prevalence of Metabolic syndrome among the study participants (N-365)**

<b>Metabolic syndrome</b>	<b>Frequency</b>	<b>Percentage (%)</b>
<b>Yes</b>	229	62.8
<b>No</b>	136	37.2
<b>Total</b>	365	100

Coming to the primary objective of our study, 229 (62.8%) of the total 365 participants with hypertension were found to be with hypertension.

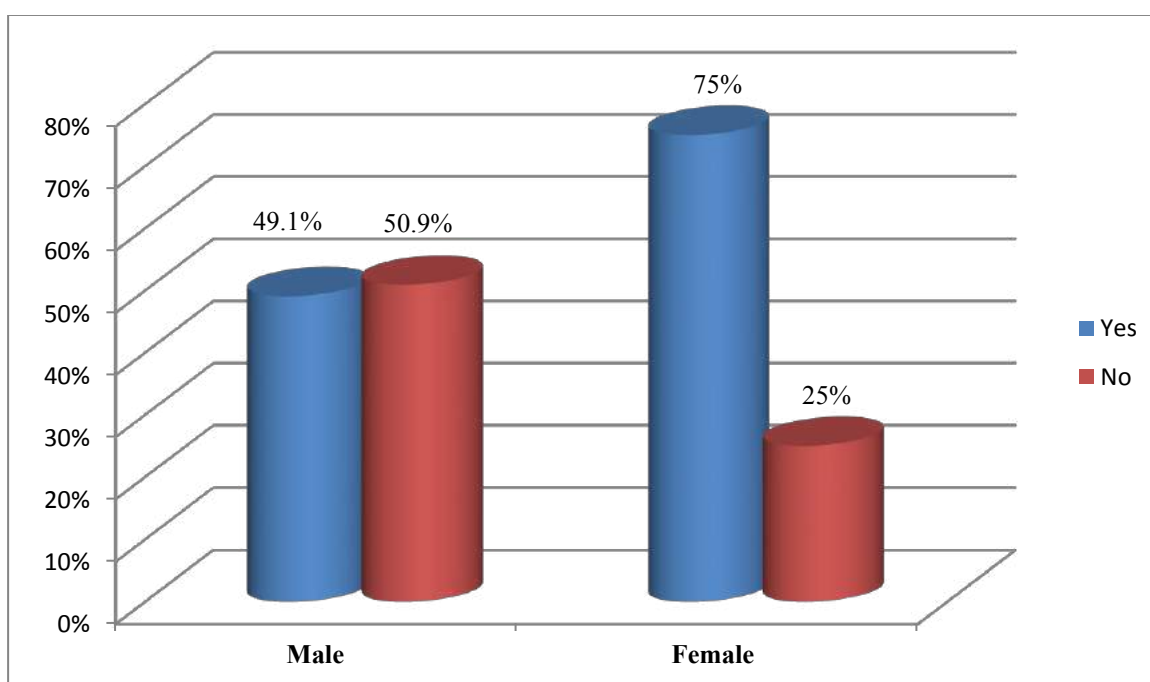
**Figure 20: Prevalence of Metabolic syndrome among the study participants**



**Table 21 : Sex wise distribution of Metabolic Syndrome (N-365)**

Variable	Metabolic Syndrome	
	Yes (229)	No (136)
Sex		
Male	85	88
Female	144	48

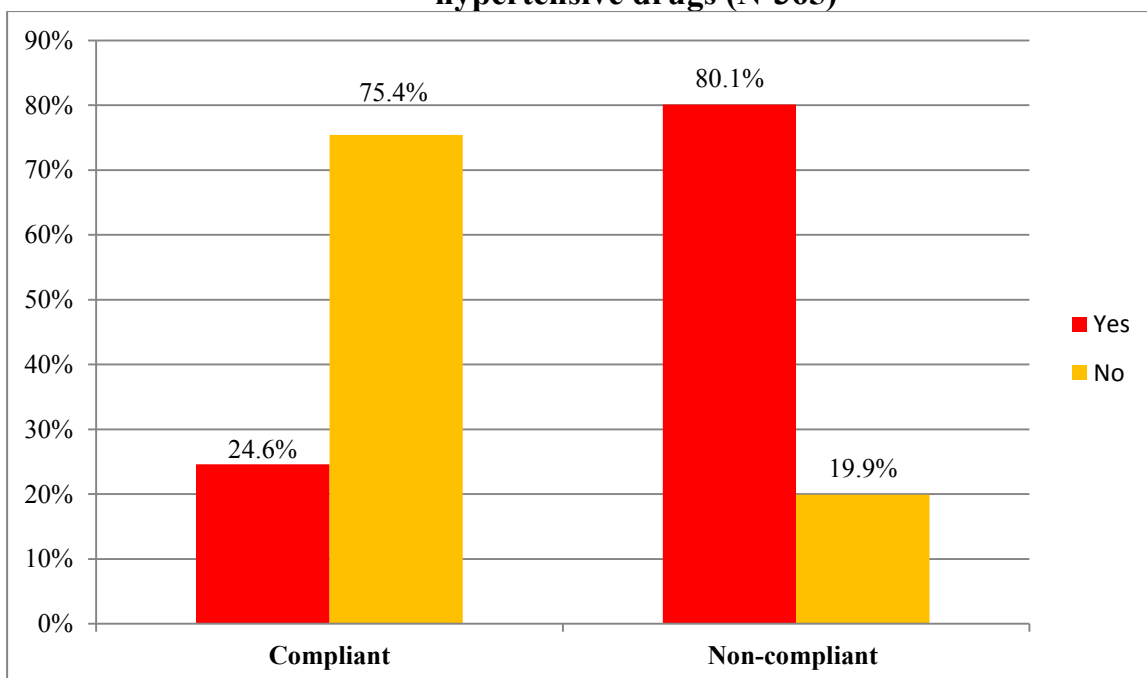
**Figure 21: Sex wise distribution of Metabolic Syndrome (N-365)**



**Table 22: Distribution of Metabolic Syndrome as per patient compliance to anti-hypertensive drugs (N-365)**

Variable	Metabolic Syndrome	
	Yes (229)	No (136)
<b>Compliance to Anti-hypertensive drugs</b>		
<b>Yes</b>	28	86
<b>No</b>	201	50

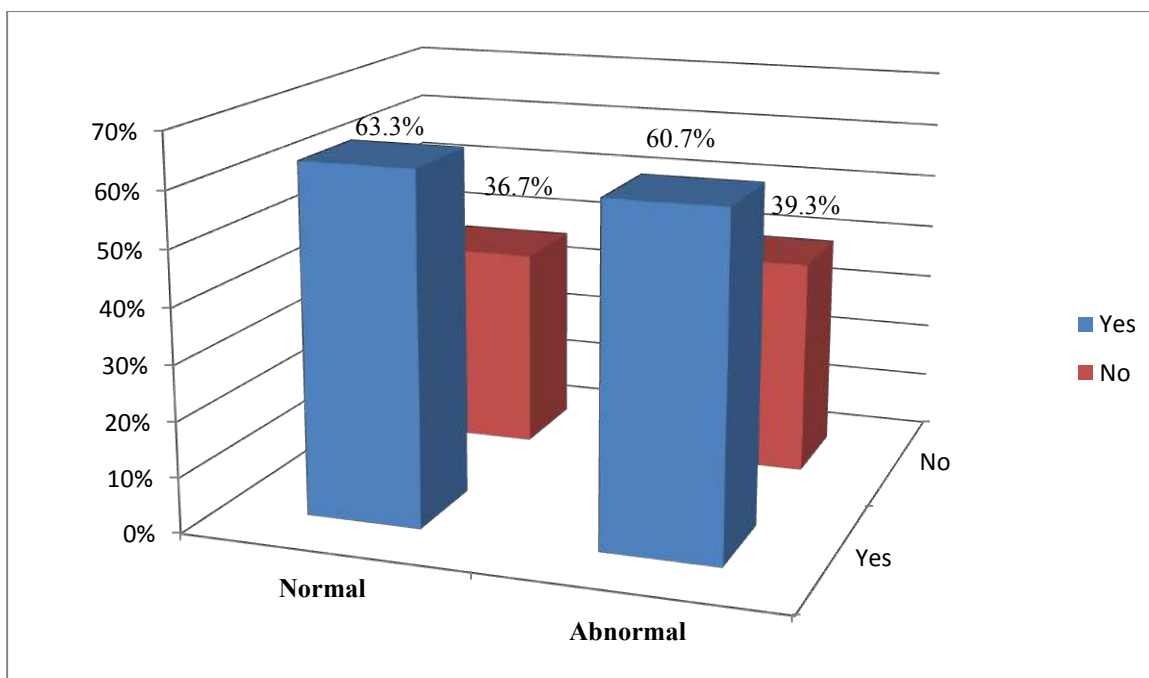
**Figure 22: Distribution of Metabolic Syndrome as per patient compliance to anti-hypertensive drugs (N-365)**



**Table 23: Distribution of Metabolic Syndrome as per waist circumference (N-365)**

Variable	Metabolic Syndrome	
	Yes (229)	No (136)
<b>Waist Circumference</b>		
Normal (Male $\leq$ 102 cms and Female $\leq$ 88 cms)	178	103
Abnormal (Male $>$ 102 cms and Female $>$ 88 cms)	51	33

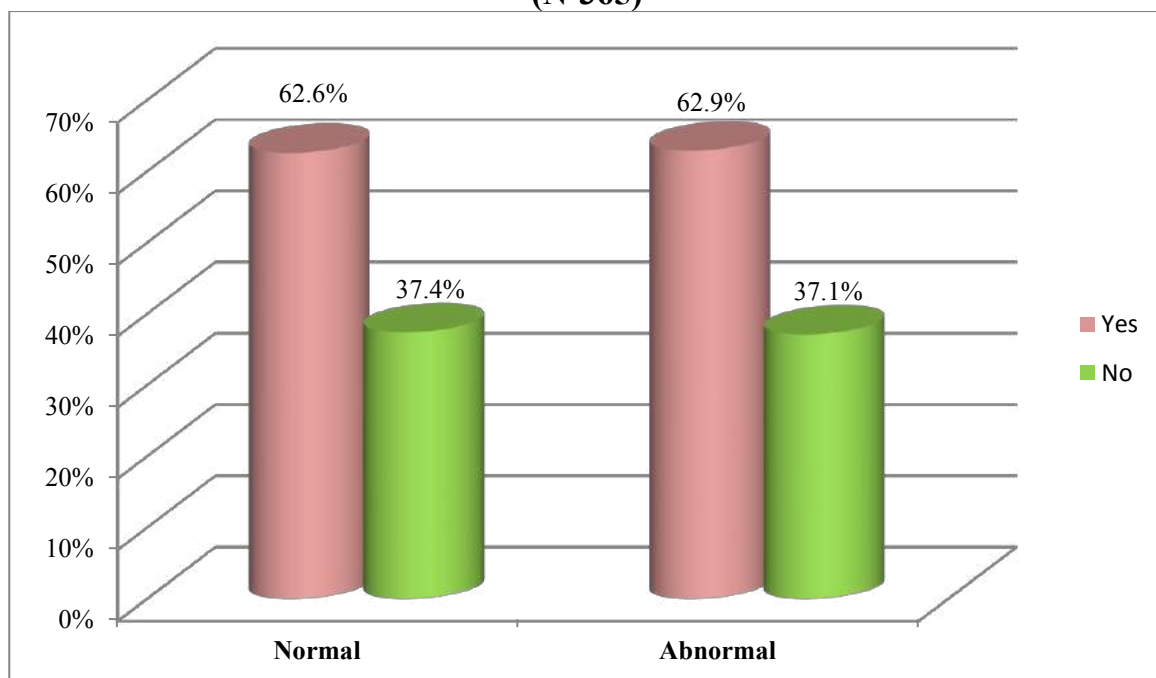
**Figure 23: Distribution of Metabolic Syndrome as per waist circumference (N-365)**



**Table 24: Distribution of Metabolic Syndrome as per the triglyceride value (N-365)**

Variable	Metabolic Syndrome	
	Yes (229)	No (136)
<b>Triglyceride Value</b>		
Normal (< 150 mg/dl)	134	80
Abnormal ( $\geq$ 150 mg/dl)	95	56

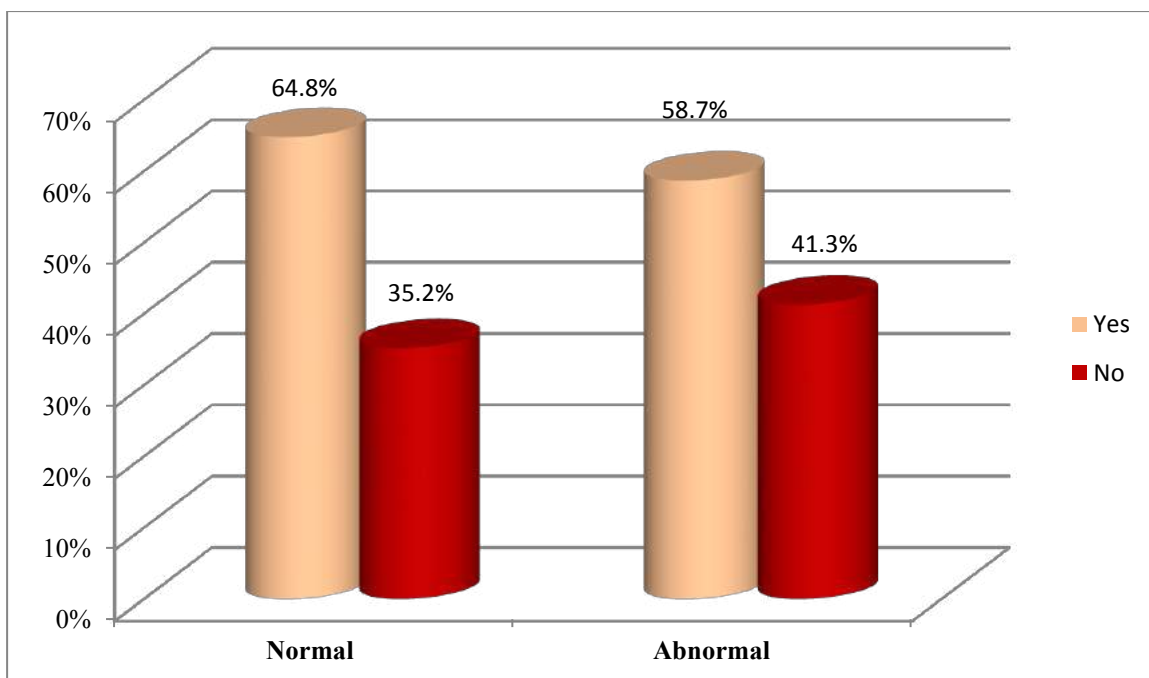
**Figure 24: Distribution of Metabolic Syndrome as per the triglyceride value (N-365)**



**Table 25: Distribution of Metabolic Syndrome as per the HDL value (N-365)**

Variable	Metabolic Syndrome	
	Yes (229)	No (136)
<b>HDL Value</b>		
Normal (Male < 40 mg/dl and Female < 50 mg/dl)	155	84
Abnormal (Male $\geq$ 40 mg/dl and Female $\geq$ 50 mg/dl)	74	52

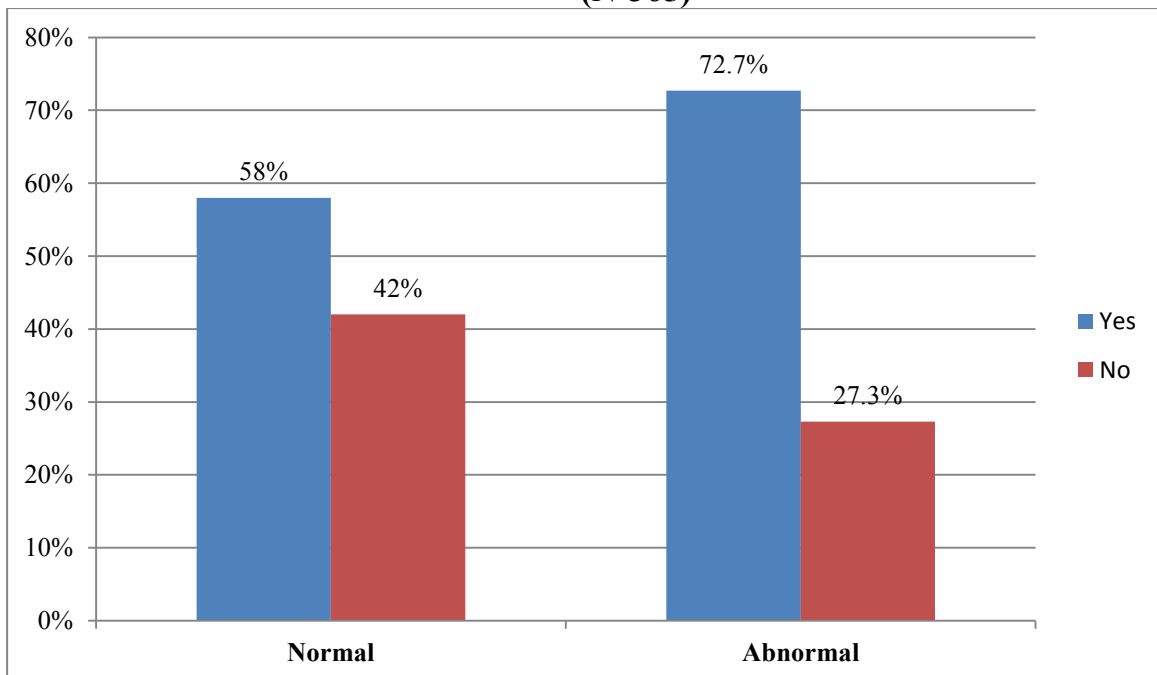
**Figure 25: Distribution of Metabolic Syndrome as per the HDL value (N-365)**



**Table 26: Distribution of Metabolic Syndrome as per the Fasting Blood Sugar value (N-365)**

Variable	Metabolic Syndrome	
	Yes (229)	No (136)
<b>Fasting Blood Sugar Value</b>		
Normal (< 100 mg/dl)	141	103
Abnormal ( $\geq$ 100 mg/dl)	88	33

**Figure 26: Distribution of Metabolic Syndrome as per the Fasting Blood Sugar value (N-365)**



**Table 27: Compliance to drugs and lifestyle modification among those with and without metabolic syndrome (N-365)**

S No	Variable	Metabolic syndrome		P value	Odds ratio (95% CI)
		Yes (229)	No (136)		
<b>1.</b>	<b>Compliance to hypertensive drugs</b>				
	<b>No</b>	201	50	<b>&lt; 0.0001*</b>	12.34 (7.28-20.91)
	<b>Yes</b>	28	86		
<b>2.</b>	<b>Compliance to lifestyle modification</b>				
	<b>No</b>	188	55	<b>&lt; 0.0001*</b>	6.75 (4.17-10.92)
	<b>Yes</b>	41	81		
<b>* p value &lt; 0.05 is statistically significant and p value &lt; 0.01 is highly significant statistically</b>					

In this study, patients with low compliance to hypertension medication were considered as non-compliant and the odds of being non-compliant to hypertension drugs was 12.34 times higher among those with metabolic syndrome when compared to those without metabolic syndrome (p value < 0.0001). Also, patients who does not follow lifestyle modification or follow them rarely were considered as being non-compliant and the odds of being non-compliant to lifestyle modifications was 6.75 times higher among those with metabolic syndrome when compared to those without metabolic syndrome (p value < 0.0001)



## Themes derived

We have conducted interviews with the study participants for the detailed analysis of the factors contributing to poor compliance of treatment. From the patient interviews, reasons for non compliance have been categorized under the following themes:

1. Patients related factors
2. Healthcare system and provider related factors
3. Medication related factors
4. Disease related factors
5. Social and environmental factors

**Figure 27. Themes derived from the in-depth interviews**



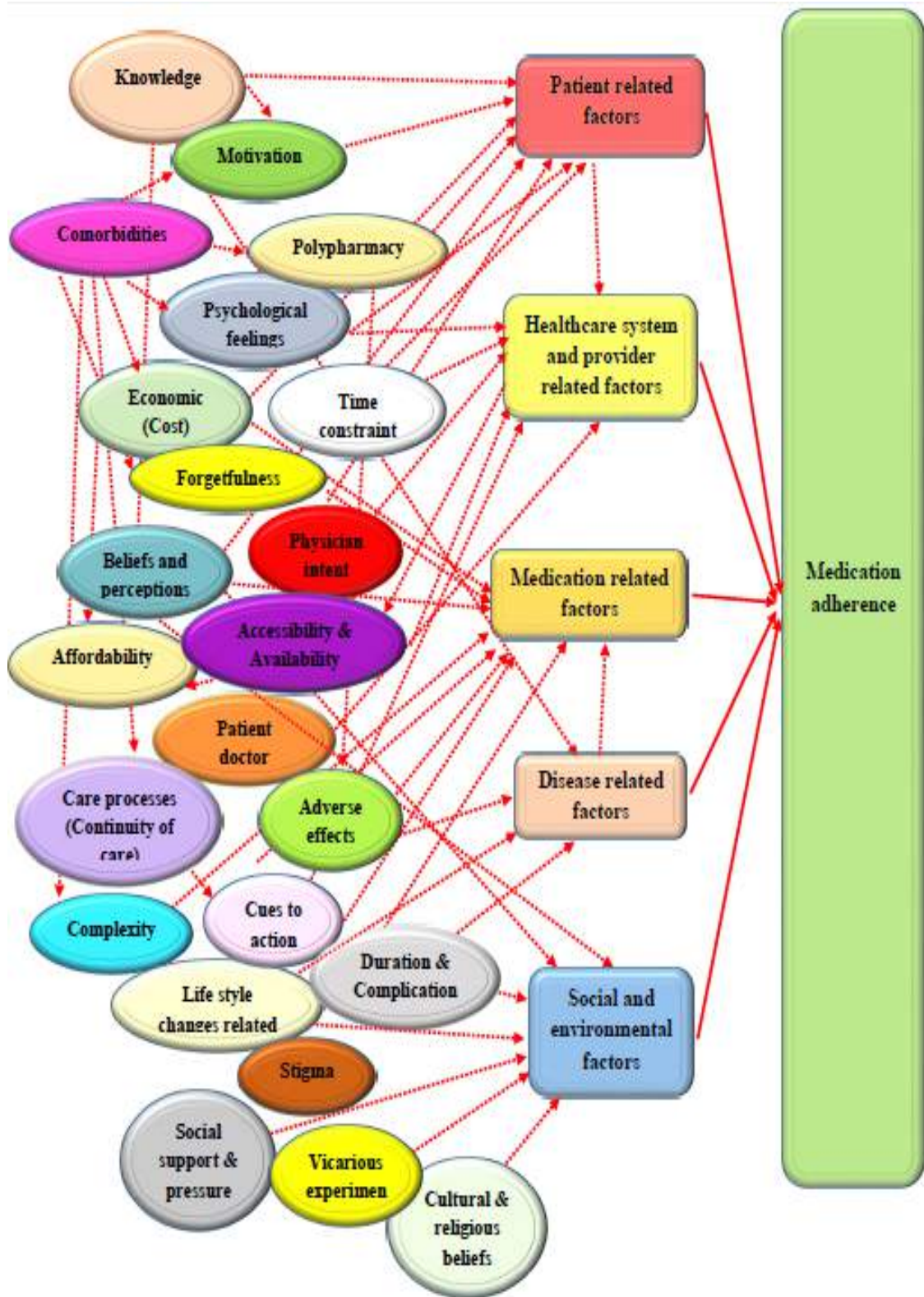


Figure 28. Conceptual framework (Barriers to medication adherence)

## **Theme 1. Patient related factors**

Patients are the centre of any healthcare management and only the patient centric health care management succeeds. Ultimately it is the patient compliance which makes our treatment to reach the patient at the end. Patient education dramatically improves the compliance. Knowledge about the disease and its effects makes the patient to think about its influence on day to day life activities of the patient. If the lifestyle modifications, the methods to adopt them and their beneficial effects are explained in patient's language it provides a motivation for the patient to adhere to the treatment suggested by us. Patient related factors are expense incurred at each visit which is augmented by long distance to travel to hospital, lack of accompanying persons and most importantly lack of that day's wages contribute to patient non compliance.

## **Theme 2. Healthcare system and provider related factors**

### **Affordability**

As we are dealing with chronic diseases the drugs which we prescribe has to be taken by the patients for lifetime, whose dosage may progressively increase and additional molecules may be added based upon the patient's new onset comorbidities. Affordability factor is the main reason as the cost for medications increase and the earnings decrease progressively as the patient ages. Here in our state all the medications are available free of cost and are distributed fortnightly or monthly.

### **Accessibility and availability**

Hospital accessibility is the most important factor for drug compliance. Not only the distance of hospital from home, availability of transportation services, availability of

accompanying persons and the cost of transportation also contribute towards the accessibility and availability of health care.

### **Care processes (Continuity of care, follow-up and visit duration)**

Patient care process contributes towards the effectiveness and reach of our advice given. In some hospitals health care provider and patient ratio are according to the recommended guidelines while in some others the ratio is far less compared to the standard guidelines. In the latter cases, time spent with each patient drastically reduces which reduces the patient confidence and compliance to treatment. Long term measures are needed to improve the health man power infrastructure.

### **Patient attender interaction:**

Joint family concept is diminishing nowadays. Due to westernization and urbanization more and more families are becoming nuclear. Most of the elderly population are isolated and don't have accompanying persons to hospital. Attender education and awareness are also important factors in compliance of treatment, especially in cases of elderly people.

## **Theme 3. Medication related factors**

### **Complexity of the drugs and polypharmacy**

Most of the hypertensive patients in our country have co existent diabetes and all of them are on multiple drugs with varying frequency. Old age patients with poor visual acuity find it difficult for them to identify the dose of the drug. Also it is practically difficult for them to remember about the frequency and the number of pills to be taken. This contributes towards wrong drug dosage ultimately leading to poor disease control.

### **Lifestyle changes related**

Lifestyle modifications form the core pillar of treatment in hypertension. Initially with lifestyle modifications patient may feel better which leads to the wrong practice of stopping drugs abruptly. Also lifestyle modifications like daily aerobic exercise are not followed by most of patients due to poor discipline. Some of them need long working hours in office which make them impossible to spare time for exercise.

### **Adverse effects**

Adverse effects of drugs especially the postural hypotension is a daring side effect which makes patient to stop the drug abruptly which again precipitates the disease specific complications.

### **Theme 4. Social and environmental factors**

Still there are communities in our society who do not access health care facilities due to superstition beliefs. Social stigmata, lack of social support and social pressure contribute towards poor hospital accessibility and poor compliance.

### **Theme 5. Disease related factors**

Diseases like hypertension and diabetes exhibit iceberg phenomenon where in only later stages of diseases exhibit symptoms. By the the time patient access' the health care facility end organ damage had already occurred. This long asymptomatic latency contributes towards lack of addressing the health care issues at the earliest. To overcome this issue in particular, screening programmes at the community level are of great value.

# *Discussion*

## DISCUSSION

Here in our study we have sampled 365 participants presented to our hypertension OPD. After getting their consent through a structured questionnaire details have been recorded. For universal standardization adherence to medication was enquired by Morisky Medication Adherence Questionnaire. Perceived stress scale scoring system was used to categorise the stress experienced by the participants. IPAQ questionnaire was used to categorise the participants according to their weekly physical activity. Also their compliance to lifestyle modification and follow up adherence were also recorded.

We have seen high prevalence of metabolic syndrome 62.8% in hypertensive patients. These people are prone to accelerated vascular diseases which drastically reduces their productivity and life expectancy, Importantly, mean fasting blood glucose value is 98.8 which signifies most of the participants are on the verge of diabetes mellitus. Also mean waist circumference of the study population is 91.9 which signifies the high prevalence of obesity.

In all those persons with metabolic syndrome we have educated the patients regarding life style modifications and pharmacotherapy. Psychiatric counselling is also given to those who needed them. The most important lacunae identified during our study is lack of involvement of family members in treatment plan. During the one year course of study we have seen drug adherence rate increased dramatically if any family member is involved, especially the spouse.

Although most of our study participants are manual labourers with moderate physical activity (88.8%) prevalence of obesity is high. it is due the intake of high glycemic index foods especially the fried rice and parotta. These items are readily

available in the shops near their work and contribute towards obesity and impaired

glucose tolerance. To avoid this home cooked foods are the healthy economical alternative.

Eventhough the study population is relatively young most of them forgot to take medicines regularly. Keeping a reminder with alarm in the cellphones greatly improves their compliance. Also prescribing single combination pill greatly reduces the aversion towards drug intake.

For those with poor compliance to regular aerobic exercises due to poor discipline forming a group with peers in the same locality greatly helps. It provides peer motivation for performing aerobic exercises especially in the morning.

Smoking and Alcoholism are widely prevalent among our study population. Not only the money spent on the substrates is lost but also the productivity of life on the next day is also lost leading to reduction in monthly income and also spending of already saved money. Most of these substance abuse are concealed from family. Even when they try to quit it is impossible or very difficult for them to stay away from these substances. This is mainly because lack of knowledge regarding the proper ways to quit them. These people have the fear of stigmatization on revealing their substance abuse habits to family and the vicious cycle continues on. Education of the family members on the substance abuse greatly reduces the stigma on it. Involvement of the family members on alcohol and smoking quitting greatly helps. Whenever the person gets demotivated it is the family members especially the spouse comes in rescue. So education and involvement of the family members is the critical bottle neck point in rehabilitation.



Inertia to approach health care facility remains in most of our study population. Surprisingly, all of our study participants are of age less than 45 years. We have advised the study participants to involve their family members in their treatment plan. Inclusion of a family member especially the spouse greatly increases the compliance.

Multimodal approach to the treatment of Metabolic syndrome brings a great success. It is a win win situation for both patients and the treating physician. Patient education and involvement of the family members is the cornerstone in treatment of the disease especially the chronic ones.

# *Conclusion*

## CONCLUSION

Hypertension is the foremost silent killer prevalent in the world. Combined with metabolic syndrome they accelerate the endotheliopathy leading to drastic consequences on the patients. Contrary to our belief, prevalence of metabolic syndrome in hypertensive adult population is very high. This condition goes highly unnoticed until a major thrombotic event occurs. Early identification by screening does a great deal in improving the patient's productive life and reducing the health expenditure.

As per our interviews with the patients, unawareness on the disease, its complications and available treatment options contribute to constitute the major reason for non-compliance. Patient education forms a key role in treatment of any disease especially the chronic diseases which require lifelong treatment. One of the key factors which drastically improved the compliance to lifelong therapy is spouse education and their involvement in treatment planning.

Affordability factors are already addressed by our government in the form of providing free health services and free drugs. Also, the Mobile Medical Unit and Makkalai Thedi Maruthuvam schemes are contributing towards screening and treatment of chronic diseases at the community level.

A multimodal approach involving the planning at government level, healthcare provider level along with patient education and involvement of the family in treating these chronic illnesses proves to be a great success in greatly improving the health and financial aspects of the client's life.

# *Limitations*

## **LIMITATIONS**

- Study is restricted to small geographical area.
- Vascular complications are not documented in the study.
- Secondary causes of hypertension are not documented in this study.
- Follow up period is less than two years for the study participants, which is a small period as long term followup is necessary to evaluate effects of lifestyle modifications in and drug therapy in these patients.

# *Bibliography*

## BIBLIOGRAPHY

1. ESC Hypertension guidelines 2018
2. Clark CE, Taylor RS, Shore AC, Ukoumunne OC, Campbell JL. Association of a difference in systolic blood pressure between arms with vascular disease and mortality: a systematic review and meta-analysis. *Lancet* 2012;379:905–914.
3. Grassi G, Seravalle G, Trevano FQ, Dell'oro R, Bolla G, Cuspidi C, Arenare F, Mancia G. Neurogenic abnormalities in masked hypertension. *Hypertension* 2007;50:537–542.
4. Huang Y, Huang W, Mai W, Cai X, An D, Liu Z, Huang H, Zeng J, Hu Y, Xu D. White-coat hypertension is a risk factor for cardiovascular diseases and total mortality. *J Hypertens* 2017;35:677–688.
5. Briasoulis A, Androulakis E, Palla M, Papageorgiou N, Tousoulis D. White-coat hypertension and cardiovascular events: a meta-analysis. *J Hypertens* 2016;34:593–599
6. Mancia G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, Bohm M, Christiaens T, Cifkova R, De Backer G, Dominiczak A, Galderisi M, Grobbee DE, Jaarsma T, Kirchhof P, Kjeldsen SE, Laurent S, Manolis AJ, Nilsson PM, Ruilope LM, Schmieder RE, Sirnes PA, Sleight P, Viigimaa M, Waeber B, Zannad F, Redon J, Dominiczak A, Narkiewicz K, Nilsson PM, Burnier M, Viigimaa M, Ambrosioni E, Caulfield M, Coca A, Olsen MH, Schmieder RE, Tsioufis C, van de Borne P, Zamorano JL, Achenbach S, Baumgartner H, Bax JJ, Bueno H, Dean V, Deaton C, Erol C, Fagard R, Ferrari R, Hasdai D, Hoes AW, Kirchhof P, Knuuti J, Kolh P, Lancellotti P, Linhart A, Nihoyannopoulos P, Piepoli MF, Ponikowski P, Sirnes PA, Tamargo JL, Tendera M, Torbicki A, Wijns W, Windecker S, Clement DL,

- Coca A, Gillebert TC, Tendera M, Rosei EA, Ambrosioni E, Anker SD, Bauersachs J, Hitij JB, Caulfield M, De Buyzere M, De Geest S, Derumeaux GA, Erdine S, Farsang C, Funck-Brentano C, Gerc V, Germano G, Gielen S, Haller H, Hoes AW, Jordan J, Kahan T, Komajda M, Lovic D, Mahrholdt H, Olsen MH, Ostergren J, Parati G, Perk J, Polonia J, Popescu BA, Reiner Z, Ryden L, Sirenko Y, Stanton A, Struijker-Boudier H, Tsioufis C, van de Borne P, Vlachopoulos C, Volpe M, Wood DA. 2013 ESH/ESC guidelines for the management of arterial hypertension: the Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *Eur Heart J* 2013;34:2159–2219
7. Myers MG. A short history of automated office blood pressure - 15 years to SPRINT. *J Clin Hypertens (Greenwich)* 2016;18:721–724.
  8. Parati G, Pomidossi G, Casadei R, Mancia G. Lack of alerting reactions to intermittent cuff inflations during noninvasive blood pressure monitoring. *Hypertension* 1985;7:597–601.
  9. Parati G, Stergiou GS, Asmar R, Bilo G, de Leeuw P, Imai Y, Kario K, Lurbe E, Manolis A, Mengden T, O'Brien E, Ohkubo T, Padfield P, Palatini P, Pickering T, Redon J, Revera M, Ruilope LM, Shennan A, Staessen JA, Tisler A, Waeber B, Zanchetti A, Mancia G. European Society of Hypertension guidelines for blood pressure monitoring at home: a summary report of the Second International Consensus Conference on Home Blood Pressure Monitoring. *J Hypertens* 2008;26:1505–1526



10. Gaborieau V, Delarche N, Gosse P. Ambulatory blood pressure monitoring versus self-measurement of blood pressure at home: correlation with target organ damage. *J Hypertens* 2008;26:1919–1927.
11. Clement DL, De Buyzere ML, De Bacquer DA, de Leeuw PW, Duprez DA, Fagard RH, Gheeraert PJ, Missault LH, Braun JJ, Six RO, Van Der Niepen P, O'Brien E, Office versus Ambulatory Pressure Study Investigators. Prognostic value of ambulatory blood-pressure recordings in patients with treated hypertension. *N Engl J Med* 2003;348:2407–2415.
12. Sega R, Facchetti R, Bombelli M, Cesana G, Corrao G, Grassi G, Mancia G. Prognostic value of ambulatory and home blood pressures compared with office blood pressure in the general population: follow-up results from the Pressioni Arteriose Monitorate e Loro Associazioni (PAMELA) study. *Circulation* 2005;111:1777–1783.
13. Banegas JR, Ruilope LM, de la Sierra A, Vinyoles E, Gorostidi M, de la Cruz JJ, Ruiz-Hurtado G, Segura J, Rodriguez-Artalejo F, Williams B. Relationship between clinic and ambulatory blood-pressure measurements and mortality. *N Engl J Med* 2018;378:1509–1520.
14. Investigators ABC-H, Roush GC, Fagard RH, Salles GF, Pierdomenico SD, Reboldi G, Verdecchia P, Eguchi K, Kario K, Hoshide S, Polonia J, de la Sierra A, Hermida RC, Dolan E, Zamalloa H. Prognostic impact from clinic, daytime, and night-time systolic blood pressure in nine cohorts of 13,844 patients with hypertension. *J Hypertens* 2014;32:2332–2340; discussion 2340.
15. Fagard RH, Celis H, Thijs L, Staessen JA, Clement DL, De Buyzere ML, De Bacquer DA. Daytime and nighttime blood pressure as predictors of death and

- cause-specific cardiovascular events in hypertension. *Hypertension* 2008;51: 55–61.
16. Parati G, Ochoa JE, Bilo G, Agarwal R, Covic A, Dekker FW, Fliser D, Heine GH, Jager KJ, Gargani L, Kanbay M, Mallamaci F, Massy Z, Ortiz A, Picano E, Rossignol P, Sarafidis P, Sicari R, Vanholder R, Wiecek A, London G, Zoccali C, European Renal and Cardiovascular Medicine Working Group of the European Renal Association-European Dialysis and Transplant Association. Hypertension in chronic kidney disease part 2: role of ambulatory and home blood pressure monitoring for assessing alterations in blood pressure variability and blood pressure profiles. *Hypertension* 2016;67:1102–1110.
17. Piper MA, Evans CV, Burda BU, Margolis KL, O'Connor E, Whitlock EP. Diagnostic and predictive accuracy of blood pressure screening methods with consideration of rescreening intervals: a systematic review for the U.S. Preventive Services Task Force. *Ann Intern Med* 2015;162:192–204.
18. Omboni S, Parati G, Palatini P, Vanasia A, Muiesan ML, Cuspidi C, Mancia G. Reproducibility and clinical value of nocturnal hypotension: prospective evidence from the SAMPLE study. *Study on Ambulatory Monitoring of Pressure and Lisinopril Evaluation. J Hypertens* 1998;16:733–738.
19. Parati G, Stergiou G, O'Brien E, Asmar R, Beilin L, Bilo G, Clement D, de la Sierra A, de Leeuw P, Dolan E, Fagard R, Graves J, Head GA, Imai Y, Kario K, Lurbe E, Mallion JM, Mancia G, Mengden T, Myers M, Ogedegbe G, Ohkubo T, Omboni S, Palatini P, Redon J, Ruilope LM, Shennan A, Staessen JA, vanMontfrans G, Verdecchia P, Waeber B, Wang J, Zanchetti A, Zhang Y, European Society of Hypertension Working Group on Blood Pressure Monitoring

- and Cardiovascular Variability. European Society of Hypertension practice guidelines for ambulatory blood pressure monitoring. *J Hypertens* 2014;32:1359–1366
20. Vinyoles E, Felip A, Pujol E, de la Sierra A, Dura R, del Rey RH, Sobrino J, Gorostidi M, de la Figuera M, Segura J, Banegas JR, Ruilope LM. Clinical characteristics of isolated clinic
  21. Ganong's textbook of physiology 26<sup>th</sup> edition
  22. Rimoldi SF, Scherrer U, Messerli FH. Secondary arterial hypertension: when, who, and how to screen? *Eur Heart J* 2014;35:1245–1254.
  23. van den Born BJ, Lip GYH, Brguljan-Hitij J, Cremer A, Segura J, Morales E, Mahfoud F, Amraoui F, Persu A, Kahan T, Rosei EA, de Simone G, Gosse P, Williams B. ESC Council on hypertension position document on the management of hypertensive emergencies. *Eur Heart J Cardiovasc Pharmacotherapy* 2018; doi:10.1093/ehjcvp/pvy032.
  24. Vaughan CJ, Delanty N. Hypertensive emergencies. *Lancet* 2000;356:411–417.
  25. Chester EM, Agamanolis DP, Banker BQ, Victor M. Hypertensive encephalopathy: a clinicopathologic study of 20 cases. *Neurology* 1978;28:928–939.
  26. van den Born BJ, Koopmans RP, Groeneveld JO, van Montfrans GA. Ethnic disparities in the incidence, presentation and complications of malignant hypertension. *J Hypertens* 2006;24:2299–2304.
  27. Cremer A, Amraoui F, Lip GY, Morales E, Rubin S, Segura J, Van den Born BJ, Gosse P. From malignant hypertension to hypertension-MOD: a modern definition for an old but still dangerous emergency. *J Hum Hypertens* 2016;30:463–466.

28. Pinna G, Pascale C, Fornengo P, Arras S, Piras C, Panzarasa P, Carmosino G, Franza O, Semeraro V, Lenti S, Pietrelli S, Panzone S, Bracco C, Fiorini R, Rastelli G, Bergandi D, Zampaglione B, Musso R, Marengo C, Santoro G, Zamboni S, Traversa B, Barattini M, Bruno G. Hospital admissions for hypertensive crisis in the emergency departments: a large multicenter Italian study. *PLoS One* 2014;9:e93542.
29. van den Born BJ, Lowenberg EC, van der Hoeven NV, de Laat B, Meijers JC, Levi M, van Montfrans GA. Endothelial dysfunction, platelet activation, thrombogenesis and fibrinolysis in patients with hypertensive crisis. *J Hypertens* 2011;29:922–927.
30. Grassi D, O’Flaherty M, Pellizzari M, Bendersky M, Rodriguez P, Turri D, Forcada P, Ferdinand KC, Kotliar C. Hypertensive urgencies in the emergency department: evaluating blood pressure response to rest and to antihypertensive drugs with different profiles. *J Clin Hypertens (Greenwich)* 2008;10:662–667.
31. Perez MI, Musini VM. Pharmacological interventions for hypertensive emergencies: a Cochrane systematic review. *J Hum Hypertens* 2008;22:596–607.
32. Lane DA, Lip GY, Beevers DG. Improving survival of malignant hypertension patients over 40 years. *Am J Hypertens* 2009;22:1199–1204.
33. Amraoui F, Van Der Hoeven NV, Van Valkengoed IG, Vogt L, Van Den Born BJ. Mortality and cardiovascular risk in patients with a history of malignant hypertension: a case-control study. *J Clin Hypertens (Greenwich)* 2014;16:122–126.
34. Gonzalez R, Morales E, Segura J, Ruilope LM, Praga M. Long-term renal survival in malignant hypertension. *Nephrol Dial Transplant* 2010;25:3266–3272

35. Devereux RB, Alderman MH. Role of preclinical cardiovascular disease in the evolution from risk factor exposure to development of morbid events. *Circulation* 1993;88:1444–1455.
36. Cordero A, Morillas P, Bertomeu-Gonzalez V, Quiles J, Mazon P, Guindo J, Soria F, Llacer A, Lekuona I, Gonzalez-Juanatey JR, Bertomeu V, Prevalence of Peripheral Arterial Disease in Patients with Acute Coronary Syndrome Investigators. Clustering of target organ damage increases mortality after acute coronary syndromes in patients with arterial hypertension. *J Hum Hypertens* 2011;25:600–607.
37. Greve SV, Blicher MK, Sehestedt T, Gram-Kampmann EM, Rasmussen S, Vishram JK, Olsen MH. Effective risk stratification in patients with moderate cardiovascular risk using albuminuria and atherosclerotic plaques in the carotid arteries. *J Hypertens* 2015;33:1563–1570.
38. de Simone G, Devereux RB, Izzo R, Girfoglio D, Lee ET, Howard BV, Roman MJ. Lack of reduction of left ventricular mass in treated hypertension: the strong heart study. *J Am Heart Assoc* 2013;2:e000144.
39. Lonnebakken MT, Izzo R, Mancusi C, Gerds E, Losi MA, Canciello G, Giugliano G, De Luca N, Trimarco B, de Simone G. Left ventricular hypertrophy regression during antihypertensive treatment in an outpatient clinic (the Campania Salute Network). *J Am Heart Assoc* 2017;6:e004152.
40. Bacharova L, Schocken D, Estes EH, Strauss D. The role of ECG in the diagnosis of left ventricular hypertrophy. *Curr Cardiol Rev* 2014;10:257–261
41. Pahor M, Guralnik JM, Ambrosius WT, Blair S, Bonds DE, Church TS, Espeland MA, Fielding RA, Gill TM, Groessl EJ, King AC, Kritchevsky SB, Manini TM,

- McDermott MM, Miller ME, Newman AB, Rejeski WJ, Sink KM, Williamson JD. Effect of structured physical activity on prevention of major mobility disability in older adults: the LIFE study randomized clinical trial. *JAMA* 2014;311:2387–2396
42. Okin PM, Devereux RB, Jern S, Kjeldsen SE, Julius S, Nieminen MS, Snapinn S, Harris KE, Aurup P, Edelman JM, Wedel H, Lindholm LH, Dahlof B, LIFE Study Investigators. Regression of electrocardiographic left ventricular hypertrophy during antihypertensive treatment and the prediction of major cardiovascular events. *JAMA* 2004;292:2343–2349.
43. Okin PM, Oikarinen L, Viitasalo M, Toivonen L, Kjeldsen SE, Nieminen MS, Edelman JM, Dahlof B, Devereux RB, LIFE Study Investigators. Prognostic value of changes in the electrocardiographic strain pattern during antihypertensive treatment: the Losartan Intervention for End-Point Reduction in Hypertension Study (LIFE). *Circulation* 2009;119:1883–1891.
44. de Simone G, Izzo R, Chinali M, De Marco M, Casalnuovo G, Rozza F, Girfoglio D, Iovino GL, Trimarco B, De Luca N. Does information on systolic and diastolic function improve prediction of a cardiovascular event by left ventricular hypertrophy in arterial hypertension? *Hypertension* 2010;56:99–104.
45. Bombelli M, Facchetti R, Cuspidi C, Villa P, Dozio D, Brambilla G, Grassi G, Mancia G. Prognostic significance of left atrial enlargement in a general population: results of the PAMELA study. *Hypertension* 2014;64:1205–1211
46. de Simone G, Izzo R, Aurigemma GP, De Marco M, Rozza F, Trimarco V, Stabile E, De Luca N, Trimarco B. Cardiovascular risk in relation to a new classification of hypertensive left ventricular geometric abnormalities. *J Hypertens* 2015;33:745–754; discussion 754.

47. Marwick TH, Gillebert TC, Aurigemma G, Chirinos J, Derumeaux G, Galderisi M, Gottdiener J, Haluska B, Ofili E, Segers P, Senior R, Tapp RJ, Zamorano JL. Recommendations on the use of echocardiography in adult hypertension: a report from the European Association of Cardiovascular Imaging (EACVI) and the American Society of Echocardiography (ASE). *Eur Heart J Cardiovasc Imaging* 2015;16:577–605.
48. Vlachopoulos C, Xaplanteris P, Aboyans V, Brodmann M, Cifkova R, Cosentino F, De Carlo M, Gallino A, Landmesser U, Laurent S, Lekakis J, Mikhailidis DP, Naka KK, Protogerou AD, Rizzoni D, Schmidt-Trucksass A, Van Bortel L, Weber T, Yamashina A, Zimlichman R, Boutouyrie P, Cockcroft J, O'Rourke M, Park JB, Schillaci G, Sillesen H, Townsend RR. The role of vascular biomarkers for primary and secondary prevention. A position paper from the European Society of Cardiology Working Group on peripheral circulation: endorsed by the Association for Research into Arterial Structure and Physiology (ARTERY) Society. *Atherosclerosis* 2015;241:507–532.
49. Touboul PJ, Hennerici MG, Meairs S, Adams H, Amarenco P, Desvarieux M, Ebrahim S, Fatar M, Hernandez Hernandez R, Kownator S, Prati P, Rundek T, Taylor A, Bornstein N, Csiba L, Vicaut E, Woo KS, Zannad F, Advisory Board of the 3rd Watching the Risk Symposium 2004, 13th European Stroke Conference. Mannheim intima-media thickness consensus. *Cerebrovasc Dis* 2004;18:346–349
50. Van Bortel LM, Laurent S, Boutouyrie P, Chowienczyk P, Cruickshank JK, De Backer T, Filipovsky J, Huybrechts S, Mattace-Raso FU, Protogerou AD, Schillaci G, Segers P, Vermeersch S, Weber T, Artery Society, European Society of Hypertension Working Group on Vascular Structure and Function, European

- Network for Noninvasive Investigation of Large Arteries. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity. *J Hypertens* 2012;30:445–448.
51. Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, Pannier B, Vlachopoulos C, Wilkinson I, Struijker-Boudier H, European Network for Non-invasive Investigation of Large Arteries. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J* 2006;27:2588–2605
52. Schmidt M, Mansfield KE, Bhaskaran K, Nitsch D, Sørensen HT, Smeeth L, Tomlinson LA. Serum creatinine elevation after renin-angiotensin system blockade and long term cardiorenal risks: cohort study. *BMJ* 2017;356:j791
53. Clase CM, Barzilay J, Gao P, Smyth A, Schmieder RE, Tobe S, Teo KK, Yusuf S, Mann JF. Acute change in glomerular filtration rate with inhibition of the reninangiotensin system does not predict subsequent renal and cardiovascular outcomes. *Kidney Int* 2017;91:683–69
54. Breslin DJ, Gifford RW Jr, Fairbairn JF II, Kearns TP. Prognostic importance of ophthalmoscopic findings in essential hypertension. *JAMA* 1966;195:335–338
55. Longstreth WT Jr, Manolio TA, Arnold A, Burke GL, Bryan N, Jungreis CA, Enright PL, O’Leary D, Fried L. Clinical correlates of white matter findings on cranial magnetic resonance imaging of 3301 elderly people. The Cardiovascular Health Study. *Stroke* 1996;27:1274–1282.
56. Vermeer SE, Longstreth WT Jr, Koudstaal PJ. Silent brain infarcts: a systematic review. *Lancet Neurol* 2007;6:611–619.



57. Kearney-Schwartz A, Rossignol P, Bracard S, Felblinger J, Fay R, Boivin JM, Lecompte T, Lacolley P, Benetos A, Zannad F. Vascular structure and function is correlated to cognitive performance and white matter hyperintensities in older hypertensive patients with subjective memory complaints. *Stroke* 2009;40:1229–1236.
58. Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL, Cooney MT, Corra U, Cosyns B, Deaton C, Graham I, Hall MS, Hobbs FDR, Lochen ML, Lollgen H, Marques-Vidal P, Perk J, Prescott E, Redon J, Richter DJ, Sattar N, Smulders Y, Tiberi M, van der Worp HB, van Dis I, Verschuren WMM, Binno S, ESC Scientific Document Group. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). *Eur Heart J* 2016;37:2315–2381.
59. Stevens VJ, Obarzanek E, Cook NR, Lee IM, Appel LJ, Smith West D, Milas NC, Mattfeldt-Beman M, Belden L, Bragg C, Millstone M, Raczynski J, Brewer A, Singh B, Cohen J, Trials for the Hypertension Prevention Research G. Longterm weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, phase II. *Ann Intern Med* 2001;134:1–11
60. Whelton PK, Appel LJ, Espeland MA, Applegate WB, Ettinger WH Jr, Kostis JB, Kumanyika S, Lacy CR, Johnson KC, Folmar S, Cutler JA. Sodium reduction and weight loss in the treatment of hypertension in older persons: a randomized

- controlled trial of nonpharmacologic interventions in the elderly (TONE). TONE Collaborative Research Group. *JAMA* 1998;279:839–846.
61. Elliott P, Stamler J, Nichols R, Dyer AR, Stamler R, Kesteloot H, Marmot M. Intersalt revisited: further analyses of 24 hour sodium excretion and blood pressure within and across populations. Intersalt Cooperative Research Group. *BMJ* 1996;312:1249–1253. 248. He FJ, Li J, Macgregor GA. Effect of longer-term modest salt reduction on blood pressure. *Cochrane Database Syst Rev* 2013;4:CD004937.
62. Suckling RJ, He FJ, Markandu ND, MacGregor GA. Modest salt reduction lowers blood pressure and albumin excretion in impaired glucose tolerance and type 2 diabetes mellitus: a randomized double-blind trial. *Hypertension* 2016;67:1189–1195.
63. Graudal NA, Hubeck-Graudal T, Jurgens G. Effects of low-sodium diet vs. high-sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Cochrane Review). *Am J Hypertens* 2012;25:1–15. 251. He FJ, MacGregor GA. How far should salt intake be reduced? *Hypertension* 2003;42:1093–1099.
64. Bibbins-Domingo K, Chertow GM, Coxson PG, Moran A, Lightwood JM, Pletcher MJ, Goldman L. Projected effect of dietary salt reductions on future cardiovascular disease. *N Engl J Med* 2010;362:590–599.
65. He FJ, Burnier M, Macgregor GA. Nutrition in cardiovascular disease: salt in hypertension and heart failure. *Eur Heart J* 2011;32:3073–3080.
66. He FJ, MacGregor GA. Salt reduction lowers cardiovascular risk: meta-analysis of outcome trials. *Lancet* 2011;378:380–382.

67. Taylor RS, Ashton KE, Moxham T, Hooper L, Ebrahim S. Reduced dietary salt for the prevention of cardiovascular disease: a meta-analysis of randomized controlled trials (Cochrane review). *Am J Hypertens* 2011;24:843–853.
68. Cushman WC, Cutler JA, Hanna E, Bingham SF, Follmann D, Harford T, Dubbert P, Allender PS, Dufour M, Collins JF, Walsh SM, Kirk GF, Burg M, Felicetta JV, Hamilton BP, Katz LA, Perry HM Jr, Willenbring ML, Lakshman R, Hamburger RJ. Prevention and Treatment of Hypertension Study (PATHS): effects of an alcohol treatment program on blood pressure. *Arch Intern Med* 1998;158:1197–1207.
69. Dickinson HO, Mason JM, Nicolson DJ, Campbell F, Beyer FR, Cook JV, Williams B, Ford GA. Lifestyle interventions to reduce raised blood pressure: a systematic review of randomized controlled trials. *J Hypertens* 2006;24:215–233.
70. Mente A, de Koning L, Shannon HS, Anand SS. A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease. *Arch Intern Med* 2009;169:659–669.
71. Sofi F, Abbate R, Gensini GF, Casini A. Accruing evidence on benefits of adherence to the Mediterranean diet on health: an update
72. Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity-induced hypertension: interaction of neurohumoral and renal mechanisms. *Circ Res* 2015;116:991–1006. 271. Neter JE, Stam BE, Kok FJ, Grobbee DE, Geleijnse JM. Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension* 2003;42:878–884.
73. Prospective Studies Collaboration, Whitlock G, Lewington S, Sherliker P, Clarke R, Emberson J, Halsey J, Qizilbash N, Collins R, Peto R. Body-mass index and

- cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *Lancet* 2009;373:1083–1096.
74. Flegal KM, Kit BK, Orpana H, Graubard BI. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *JAMA* 2013;309:71–82.
75. Thomopoulos C, Parati G, Zanchetti A. Effects of blood-pressure-lowering treatment on outcome incidence. 12. Effects in individuals with high-normal and normal blood pressure: overview and meta-analyses of randomized trials. *J Hypertension* 2017;35:2150–2160
76. Alberti KG et al: Harmonizing the metabolic syndrome: A joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation* 120:1640, 2009.
77. Brown AE, Walker M: Genetics of insulin resistance and the metabolic syndrome. *Curr Cardiol Rep* 18:75, 2016.
78. Eckel RH et al: The metabolic syndrome. *Lancet* 365:1415, 2005. Genser L et al: Obesity, type 2 diabetes, and the metabolic syndrome: Pathophysiologic relationships and guidelines for surgical intervention. *Surg Clin North Am* 96:681, 2016.
79. Lechner K et al: High-risk atherosclerosis and metabolic phenotype: The roles of ectopic adiposity, atherogenic dyslipidemia, and inflammation. *Metab Syndr Relat Disord* 18:176, 2020.

80. Lim S, Eckel RH: Pharmacological treatment and therapeutic perspective of metabolic syndrome. *Rev Endocr Metab Disord* 15:329, 2014. Neeland IJ et al: Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: A position statement. *Lancet Diabetes Endocrinol* 7:715, 2019.
81. Apurva Sawant, Ranjit Mankeshwar, Swarup Shah, et al., "Prevalence of Metabolic Syndrome in Urban India," *Cholesterol*, vol. 2011, Article ID 920983, 7 pages, 2011. doi:10.1155/2011/92098
82. Mancia G. The association of hypertension and diabetes: prevalence, cardiovascular risk and protection by blood pressure reduction. *Acta Diabetol* 2005;42:S17-S25
83. Bakris GL. Current perspectives on hypertension and metabolic syndrome. *J Manag Care Pharm* 2007;13:S3-S5.
84. Pickering T G .The effects of environmental and lifestyle factors on Blood pressure and intermediary role of sympathetic nervous system. *J Hum Hypertens* 11 (suppl 1 ): S9-S18,1997.
85. Arcidiacono B, Iiritano S, Nocera A et al. Insulin resistance and cancer risk: an overview of the pathogenetic mechanisms. *Exp Diabetes Res* 2012;2012:789174.
86. Gallagher EJ, LeRoith D. Epidemiology and molecular mechanisms tying obesity, diabetes, and the metabolic syndrome with cancer. *Diabetes Care* 2013;36(Suppl. 2): S233–S239.
87. Yamaoka-Tojo M, Tojo T, Takahira N et al. Elevated circulating levels of an incretin hormone, glucagon-like peptide-1, are associated with metabolic components in high-risk patients with cardiovascular disease. *Cardiovasc Diabetol* 2010; 9: 17.

88. Reaven G. Metabolic syndrome: pathophysiology and implications for management of cardiovascular disease. *Circulation* 2002; 106: 286–288.
89. Jeppesen J, Hansen TW, Rasmussen S et al. Insulin resistance, the metabolic syndrome, and risk of incident cardiovascular disease: a population-based study. *J Am CollCardiol* 2007; 49: 2112–2119
90. Duvnjak L, Bulum T, Metelko Z. Hypertension and the meta-bolic syndrome. *Diabetol Croat* 2008; 37: 83–89.
91. Reynolds K, Wildman RP. Update on the metabolic syndrome: hypertension. *CurrHypertens Rep* 2009; 11: 150–155.
92. Grundy SM, Cleeman JI, Daniels SR et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation* 2005; 112: 2735–2752.
93. Sookoian S, Pirola C. Metabolic syndrome: from the genetics to the pathophysiology. *CurrHypertens Rep* 2011; 13: 149–157.
94. Stepan CM, Lazar MA. Resistin and obesity-associated insulin resistance. *Trends EndocrinolMetab.* 2002;13:18 –23.
95. Stancakova A, Laakso M. Genetics of metabolic syndrome. *Rev EndocrMetabDisord* 2014. DOI 10.1007/s11154-014-9293-9.

# *Annexures*

**INFORMED CONSENT**

NAME :

AGE :

GENDER :

ADDRESS :

CONTACT :

PRINCIPAL INVESTIGATOR :

GUIDE :

The details of the study have been provided to me in writing in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I fully consent to participate in the above study.

SIGNATURE OF PARTICIPANT

SIGNATURE OF INVESTIGATOR

PLACE:

DATE:



## ஆராய்ச்சிஒப்புதல்படிவம்

பெயர்:

வயது: பாலினம்: ஆண் / பெண்

பங்குபெறுபவர் அடையாளஎண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

இந்த சோதனையில் நான் கலந்து கொண்டு ரத்தப்பரிசோதனைக்கு சம்மதிக்கிறேன்.

எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்துகொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழுசம்மதத்துடன் இந்த ஆராய்ச்சிக்கு என்னை பரிசோதிக்க சம்மதிக்கிறேன்.

பங்கேற்பாளர்கையொப்பம் :

ஆராய்ச்சியாளர்கையொப்பம்:

இடம் :

நாள் :

## ஆராய்ச்சி தகவல்தாள்

இதனால் தங்களது ஆய்வறிக்கையோ, அன்றாட செயல்பாடுகளோ பாதிக்கப்படாது என்று தெரிவித்துக்கொள்கிறோம். ஆராய்ச்சியின் முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ தங்கள் பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின்போது அல்லது அதன் முடிவில் அறிவிக்கப்படும் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்தநேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

பங்கேற்பாளர்

ஆராய்ச்சியாளர்

கையொப்பம்

கையொப்பம்

இடம் :

நாள் :

**GOVERNMENT STANLEY MEDICAL COLLEGE AND HOSPITAL**

**DEPARTMENT OF GENERAL MEDICINE**

**PROFORMA**

1. RESPONDENT ID CODE:

2. AGE:

3. SEX:

4. HOSPITAL NO:

5. ADDRESS:

6. MARIETAL STATUS:

- Married
- Unmarried
- Divorced
- Widow/widower

7. EDUCATIONAL STATUS:

- No formal education
- Primary education
- Secondary education
- Higher secondary education
- Graduate and above

8. OCCUPATION:

- Unemployed
- Employed
- Homemaker

- Retired

9. RESIDING IN:

- Rural area
- Urban area

10. DISTANCE OF RESIDENCE FROM HOSPITAL:

- <5 kms
- 5 – 10 kms
- >10kms

11. MONTHLY INCOME:

- <5000 Rs
- 5000-10000 Rs
- >10,000 Rs

12. WERE YOU PRESCRIBED ANY MEDICATION FOR YOUR HEALTH  
CONDITION?

- Yes
- No

13. IF YES, HOW MANY KINDS OF MEDICATION WERE YOU PRESCRIBED?

- Oral tablets only
- Injections only
- Combined (both)

MORISKY MEDICATION ADHERANCE QUESTIONNAIRE

1. Do you sometimes forget to take your medicine?

- Yes
  - No
- 2. People sometimes miss taking medication for reasons other than forgetting.  
Thinking over the past 2 weeks, were there any days when you did not take your medication?
  - Yes
  - No
- 3. Have you ever cut back or stopped taking your medication without telling your doctor because you felt worse when you took it?
  - Yes
  - No
- 4. When you travel or leave home, do you sometimes forget to bring along your medicine?
  - Yes
  - No
- 5. Did you take your all your medicines yesterday?
  - Yes
  - No
- 6. When you feel like your symptoms are under control, do you sometimes stop taking your medication?
  - Yes
  - No
- 7. Taking medication every day is a real inconvenience for some people. Do you

ever feel hassled about sticking to your treatment plan?

- Yes
- No

8. How often do you have difficulty remembering to take all your medicine?

- Never/ rarely (score 0)
- Once in a while
- Sometimes
- Usually
- All the time

Scoring:

Yes =0; no =1

For Q.no 5 alone, Yes = 1; No = 0

Total score:

<6 – low adherence

6 to 8 = medium adherence

8 = high adherence

PERCEIVED STRESS SCALE (during the last month).

1. In the last month, how often have you been upset because of something that happened unexpectedly? 0 1 2 3 4

2. In the last month, how often have you felt that you were unable to control the important things in your life? 0 1 2 3 4

3. In the last month, how often have you felt nervous and “stressed”? 0 1 2 3 4
4. In the last month, how often have you felt confident about your ability to handle your personal problems? 0 1 2 3 4
5. In the last month, how often have you felt that things were going your way? 0 1 2 3 4
6. In the last month, how often have you found that you could not cope with all the things that you had to do? 0 1 2 3 4
7. In the last month, how often have you been able to control irritations in your life? 0 1 2 3 4
8. In the last month, how often have you felt that you were on top of things? 0 1 2 3 4
9. In the last month, how often have you been angered because of things that were outside of your control? 0 1 2 3 4
10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them? 0 1 2 3 4

ADHERANCE TO .PHYSICAL ACTIVITY (During last 7 days, for at least 10 mins at a time)

1. How many days did you do vigorous physical activities? (if No, GO TO 3)
2. How much time did you usually spend doing them on one of those days? Hrs: Min:
3. How many days did you do moderate physical activities? (if No, GO TO 5)
4. How much time did you usually spend doing them on one of those days? Hrs: Min:
5. How many days did you walk? (if No, GO TO 7)
6. How much time did you usually spend walking on one of those days? Hrs:  
Min:

7. How much time did you spend sitting on a week day?      Hrs:    Min:

ADHERANCE TO DIETARY MODIFICATION:

1. Do you follow the advice regarding dietary modification given by your physician or dietician?
  - Daily
  - Frequently
  - Rarely
  - Never
2. If not, why?
  - It is difficult to follow the dietary schedule given to me due to lack of discipline
  - I was not given dietary advice at all
  - I was given some oral dietary advice but I forgot the details as I was not given in writing
  - I think it does not make any change to my health condition

ADHERANCE TO LIFESTYLE MODIFICATION

1. Are you a smoker?
  - Current smoker(past 30 days)
  - Past smoker
  - Never smoked
2. Are u an alcohol consumer?
  - Current alcohol consumer(30 days)
  - Past alcohol consumer



- Never consumed

### ADHERANCE TO FOLLOWUP

1. How often do you visit a hospital for periodic medical checkup?
  - As advised by your doctor
  - Less frequently than advised by your doctor
  - More frequently than advised by your doctor
2. If less than that advise by your doctor, why?
  - Long distance from hospital
  - Lack of transportation services
  - High cost incurred in each visit
  - Non availability of persons to accompany to the hospital
  - Due to your busy work schedule
  - Because I think it is not necessary to visit so frequently as I feel better now

### ANTHROPOMETRY

HEIGHT

WEIGHT

BMI

Waist circumference:            cms

Hip circumference    :            cms

BLOOD PRESSURE

LABORATORY TEST REPORTS:

HDL cholesterol levels:

TGL levels

FBS

RBS

























