STUDY ON METABOLIC SYNDROME IN NEWLY DETECTED HYPERTENSIVE INDIVIDUALS AT GOVERNMENT VELLORE MEDICAL COLLEGE AND HOSPITAL, VELLORE

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I, **DR.LAVANYA.M** solemnly declare that this dissertation titled **"STUDY ON METABOLIC SYNDROME IN NEWLY DETECTED HYPERTENSIVE INDIVIDUALS"** is a bonafide work done by me in Department of General Medicine, Government Vellore Medical College and Hospital, Vellore under the guidance and supervision of **Prof. Dr. J.Philomena M.D.,** Guide and Head of Department, General Medicine.

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LIST OF ABBREVIATIONS USED

TNF- Tumour Necrosis Factor

IL- Interleukine

NCEP ATP- National Cholesterol Education Programme Adult Treatment Panel

LDL- Low Density Lipoprotein

QTL- Quantitative Trait Loci

IASO- International Association for the Study of Obesity

OR- Odds Ratio

CI – Confidence Interval

LDL- Low Density Lipoprotein

TGL- Triglyceride

FA- Fatty acids

HDL- High Density Lipoprotein

VLDL- Very Low Density Lipoprotein

TC-Total Cholesterol

NHANES- National Health and Nutrition Examination Survey

GLP-1 – Glucagon Like Peptide 1

DNA- Deoxyribo Nucleic Acid

RAAS- Renin Angiotensin Aldosterone System

ACE- Angiotensin Converting Enzyme

Na^+ - Sodium

K^+ - Potassium

BP- Blood Pressure

NO- Nitric Oxide

ET- Endothelin

ANP- Atrial Natriuretic Peptide

BNP- Brain Natriuretic Peptide

CNP- C-type Natriuretic Peptide

DNP-Dendroaspis Natriuretic Peptide

CGRP- Calcitonin Gene Related Peptide

t-PA- tissue Plasminogen Activator

MRFIT- Multiple Risk Factor Intervention Trial

SBP- Systolic Blood Pressure

DBP- Diastolic Blood Pressure

JNC- Joint National Committee

ESRD- End Stage Renal Disease

BUN- Blood Urea Nitrogen

ECG- Electro Cardio Gram

CHD- Coronary Heart Disease

BMI- Body Mass Index

BA- Bile Acids

LPL- Lipo Protein Lipase

DHA- DecosaHexaenoic Acid

EPA- EicosaPenatanoic Acid

CVA- Cerebro Vascular Accident

CAD- Coronary Artery Disease

MTO- Metabolic equivalent of Task

ANOVA- Analysis Of Variance

WHR- Waist Hip Ratio

FBS- Fasting Blood Sugar

ABSTRACT

BACKGROUND

Metabolic syndrome includes hypertension, obesity, insulin resistance and dyslipidemia. Metabolic syndrome helps in early identification of the people at risk of developing diabetes and cardiovascular diseases. The prevalence of metabolic syndrome varies from 50%-80% in various parts of the world. Thus early recognition of metabolic syndrome is needed.

AIM

1. To study the prevalence of metabolic syndrome in newly diagnosed hypertensive individuals.
2. To study the prevalence and pattern of lipid profile abnormalities in hypertensive individuals.

To study the clinical, demographic and social parameters influence on lipid profile abnormalities.

METHODS

In this cross sectional study, 150 newly detected hypertensive individuals were included in the study. All participants were explained about the study and informed consent was obtained. They were investigated for fasting blood glucose, lipid profile including total cholesterol, triglycerides, high density lipoprotein . Anthropometric measurements were made. Statistical

analysis was made using SPSS 16 software . The difference between various parameters were considered statistically significant if the p value was <0.05.

RESULTS

The study population of 150 newly detected hypertensive individuals, there are 78 (52%) males and 72 (48%) females. The mean age of the study population is 55.68 years.

The prevalence of metabolic syndrome calculated according to NCEP ATP III criteria is 59.33% that is 89 patients out of 150 newly detected hypertensive people. There is no statistical difference in the prevalence of metabolic syndrome on the basis of gender, age. Statistical significant difference in the prevalence of metabolic syndrome among smokers compared to non-smokers. The people with sedentary lifestyle had significantly higher prevalence. The prevalence of metabolic syndrome was significantly higher in people in stage 2 hypertension. The prevalence of each component of metabolic syndrome in male is found to be elevated triglycerides (80.76%), abnormal waist circumference (46.15%) , low HDL (43.58%), impaired fasting blood glucose (26.92%) and in females the same component's prevalence are 73.61%, 72.22%, 12.5%, 15.27% respectively.The prevalence of each component in patients with metabolic syndrome in our study shows that in male, elevated TGL (97.67%)is the most common component being seen. It is followed by abnormal waist circumference (79.06%), low HDL (76.74%), impaired blood glucose (39.53%). Among females with metabolic syndrome the prevalence of

components are low HDL (97.8%), elevated TGL (93.47%), abnormal waist circumference (89.13%), impaired blood glucose (17.39%).The prevalence of lipid profile abnormalities in hypertensive patients is elevated TC 43.33%, elevated TGL 76.8%, elevated LDL 28.66% and low HDL 44.4%. The prevalence of elevated total cholesterol was significantly higher in patients with age ≥50 years.The prevalence of low HDL among male and female is 43.58% and 12.5%. The difference between the two is statistically significant.The mean values of lipid profile abnormalities are compared between sedentary and non-sedentary hypertensive people and it is significantly higher in sedentary group.There is significant difference in the lipid profile values of obese people compared to non-obese individuals.

CONCLUSION

The prevalence of metabolic syndrome is high in newly detected hypertensive individuals. People with sedentary lifestyle, smokers, stage 2 hypertensive people had significantly higher prevalence of metabolic syndrome. The dyslipidemia was seen significantly higher in people with age ≥ 50 years, sedentary people and obese people.

KEY WORDS

Metabolic syndrome, hypertension, dyslipidemia, obesity

TABLE OF CONTENTS

METABOLIC SYNDROME IN NEWLY DETECTED HYPERTENSIVE INDIVIDUALS

INTRODUCTION

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[1]. These diseases in turn lead to adverse outcomes causing 1.6 times increase in mortality. In 1975 the term "metabolic syndrome" was given by Haller and Hanefeld[2]. Various terms used to denote metabolic syndrome are 'dysmetabolic syndrome', 'hypertriglyceridemic waist', 'insulin resistance syndrome', 'obesity syndrome' and 'syndrome X'. Obesity and resistance to insulin are found to be mainly caused by absence of physical work, sedentary lifestyle and poor dietary habits like fatty foods.

Metabolic syndrome includes resistance to insulin, elevated blood pressure, elevated lipid profile and obesity. All components of metabolic syndrome are mostly preceded by obesity. Atleast 3 out of five components is necessary to make a diagnosis of metabolic syndrome. Ethnic and national specificity is required for waist circumference.

Genetics, ageing, state of inflammation and changes in hormone may also have a causal effect, but the role of these may vary depending on ethnic group. Certain risk factors that are non-classic like c-reactive protein, abnormal oxidized low-density lipoprotein-cholesterol and adiponectin are very much related to metabolic syndrome.

From a practical clinical perspective and also from a clinical standpoint people at high metabolic risk are identified by using the metabolic syndrome criteria. A high level of low-density lipoprotein-cholesterol which is a traditional risk factor may be absent and so the individuals at high risk might be overlooked if these metabolic syndrome criteria are not utilized.

As metabolic syndrome being the important factor leading to two major diseases, cardiovascular diseases and type 2 diabetes, there is an immense need on social, moral and on medical perspective for early detection of people with metabolic syndrome. Thus the onset of diseases like diabetes , cardiovascular disease can be prevented very well by identifying metabolic syndrome earlier . These people can be advised and subjected to lifestyle modifications and started on appropriate treatment .

Thus metabolic syndrome is helpful in rapid identification of people at high metabolic risk. This study is conducted to find the prevalence of metabolic syndrome in newly diagnosed hypertensive individuals and the factors influencing lipid profile abnormalities in hypertensive individuals.

AIM OF STUDY

1. To study the prevalence of metabolic syndrome in newly diagnosed hypertensive individuals.

2. To study the prevalence and pattern of lipid profile abnormalities in hypertensive individuals.

3. To study the clinical, demographic and social parameters influence on lipid profile abnormalities.

REVIEW OF LITERATURE

METABOLIC SYNDROME

Metabolic syndrome is associated with an imbalance between energy intake and energy storing capacity. This imbalance results in ectopic lipid deposition in visceral fat, skeletal muscle (insulin resistance), liver (non-alcoholic steatohepatitis), pancreatic beta cells and vessel wall or macrophages (coronary artery disease)[3]. The metabolic syndrome may occur in obese and non-obese people.

Lipid storage occurs in visceral reservoirs, in the people who lack subcutaneous fat as in non-obese patients. Spillage of lipids to muscle, blood vessels, pancreas and liver occurs after the filling of these reservoirs. Staging of chronic diseases are set based on this. Metabolic syndrome acts as a predictor of the chance of developing type 2 diabetes and cardiovascular disease in case of presence of visceral obesity.

The people with metabolic syndrome, who are not obese are also expected to face the risk of cardiovascular disease and other associations of metabolic syndrome because of the fat being deposited in the viscera. Certain factors being secreted by the visceral fat known as ‘adipocytokines’ are responsible for the development of metabolic syndrome. they are resistin, (TNF) tumour necrosis factor, (IL) interleukins and leptin.

GENETICS OF METABOLIC SYNDROME

There is difficulty in identifying the genetic component of metabolic syndrome because of few factors. They are varying lifestyle factors, absence of a single defining criterion for metabolic syndrome, complexity of metabolic syndrome[4]. From linkage analysis, genome wide association studies and candidate gene approach, metabolic syndrome having a genetic component has been identified.

FAMILY AND TWIN STUDIES

Regarding genetics of metabolic syndrome , further more details have been provided by numerous family and twin studies. As per NCEP:ATPIII criteria, the heritability every single component, was found out. It was 20% for elevated blood pressure, 16-60% with dyslipidemia/hyperglycemia and 44% with obesity[5]. The metabolic syndrome as such from studies conducted among 89 Caribbean-Hispanic families showed a heritability of 24% ($p = 0.006$) in 203 subjects. Italian individuals of 293 were subjected to a similar study. The heritability of metabolic syndrome was found to be 27% ($p = 0.002$) as per NCEP ATP III criteria. 54% and 10% was the heritability of low high-density lipoprotein and elevated blood glucose. The maximum heritability was found to be 31% ($p <0.001$) for high low density cholesterol (LDL), central obesity and elevated blood pressure[6].

LINKAGE STUDIES

Many studies have been conducted on different combination of components

and on individual components of metabolic syndrome than on metabolic syndrome as such since it is a complex disorder. Numerous studies have been conducted to find the genes that are associated with the development of metabolic syndrome. It is done by using linkage studies. This led to the discovery of a quantitative trait loci (QTL). It was found to be present in the chromosome 3 on studying 507 families from U.S[7]. Another study conducted showed that there is an association between the chromosomes 10, 19 and blood sugar, blood pressure, low high density lipoprotein, triglyceride , increased body and viscera fat. Association was also found with chromosome 1[8].the association between the chromosome and the genes varied between different ethnic groups[9].

GENOME-WIDE ASSOCIATION STUDIES

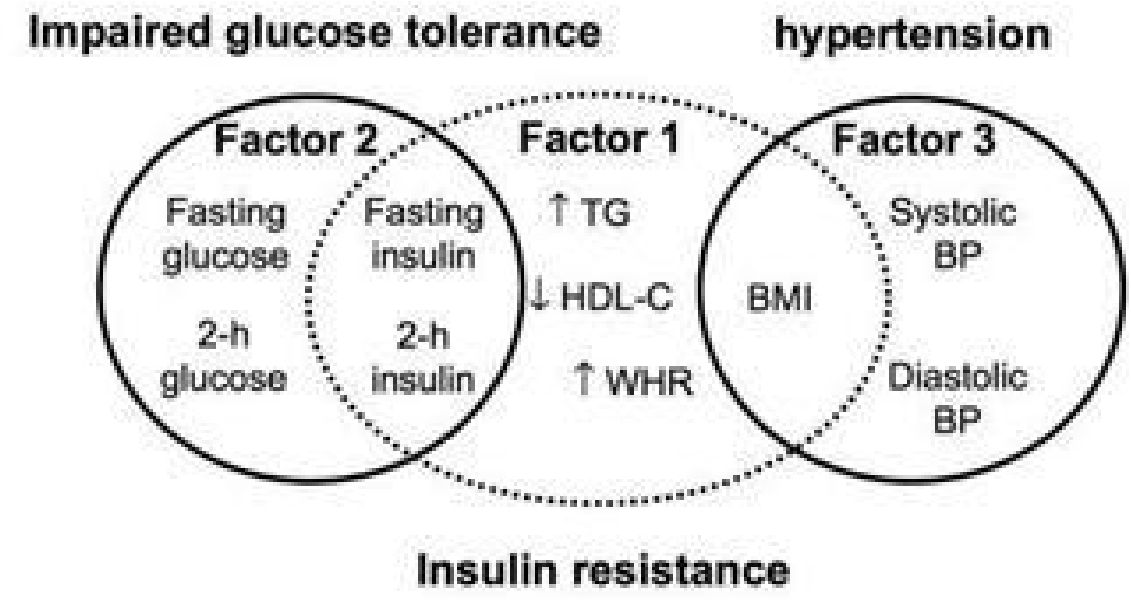
Further many studies were conducted to on genome wide association studies and its relation to metabolic syndrome. Type 2 diabetes and its relation to the gene known as (FTO) obesity associated protein gene was found by single nucleotide polymorphism by the International Association for the study of obesity (IASO). From the study evidences were found that there is a relation between the gene and the development of diabetes. It was seen that the people who had the homozygous status for the gene were heavy and had increased risk of becoming obese[10]. Thus a linkage between these genes that is FTO and others was found to be present with obesity by its effects on body mass index. This association was seen in European[11-15] as well as

Asian people[16]. But still more studies and researches are need to confirm this relation between the gene and body mass index.

The components of metabolic syndrome have been found to related many single nucleotide polymorphism as per many studies. These genes are found to be associated with central adiposity, resistance to insulin and metabolism of lipids. Yet may studies are needed to establish this linkage to metabolic syndrome.

REASON BEHIND THE OCCURRENCE OF COMPONENTS TOGETHER

Metabolic syndrome development is induced by interaction of factors that occurs together. Increased weight, especially central obesity leads to all other components to arise. The factors that occurs mostly together are high blood pressure, abnormal lipid profile, high blood sugar level. Central obesity is considered as an important factor leading to high values of blood glucose by causing resistance to insulin, abnormal lipid profile, high blood pressure and high blood sugar levels[17].



BMI, body mass index; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides; WHR, waist-to-hip ratio.

Figure 1

The cause for occurrence of these factors together was studied by Meigs et al[17]. The concept behind happening of all components altogether in individuals leading to metabolic syndrome was studied as a cohort study. Factor analysis proved that 3 things that reason out the grouping together of various components. The first thing is the one which leads to the onset of resistance to insulin. This comprises of lipid profile abnormalities, central obesity. The next thing is the one that causes blood sugar impairment and development of diabetes by causing the loss of function of pancreas. The last one is responsible for causing elevation in blood pressure. Thus the presence of all components altogether in metabolic syndrome is influenced by many factors that act individually. These components are mostly normal physiological factors but they lead to the occurrence of metabolic syndrome[17].

The pathogenesis for diabetes occurrence was studied in 1087 people who are not diabetics. The study was done by Hanley et al[18]. The analysis was done to know whether the elevated blood sugar is caused not only by metabolic causes but also by many inflammatory factors. It known as 'Insulin Resistance Atherosclerosis Study'. The things that were analyzed are sensitivity to insulin and factors that causes inflammation.

The study results found that there are 3 things that are important,

1) "metabolic" – sensitivity to insulin, central obesity like abnormal body mass index, plasminogen activator factor, high values of blood glucose.

2) “inflammation” – sensitivity to insulin, circumference of waist, fibrinogen, body mass index

3) “blood pressure” – the prediction of development of diabetes was studied after a following for few years.[18]

	Risk of incident diabetes		
Univariate models	**OR**	**95% CI**	
		from	to
Metabolic factor	2.26	1.82	2.80
Inflammation factor	1.57	1.32	1.87
Blood pressure factor	1.44	1.20	1.73

TABLE 1- The predictor of later occurrence of diabetes is metabolic syndrome.

p = 0.0001

The factors that lead to cardiovascular diseases in people who are not known diabetics are studied[18]. The studies proved that these factors are responsible for grouping together of various components of metabolic syndrome. They are sensitivity to insulin, central obesity factors like body mass index, circumference of waist. They were several metabolic factors and also things that cause inflammation.

OBESITY

According to many studies there is a drastic increase in people who are obese, and it is seen all over the world. Obesity refers to the accumulation of fat in the body that occurs beyond normal. It is measured by many parameters like measuring the circumference of waist or calculating the body mass index. Now we have come to know that among the components of metabolic syndrome obesity is important one and for establishing a diagnosis of metabolic syndrome[19].

It was estimated that the people who are obese increases rapidly and a study by Finucane et al[21] there were more than one billion obese people with a body mass index of above 25kg/m^2 . And this comprises of people who are obese and also who are overweight. This population has been reached from a one billion population of obese people from 2005.thus it was also concluded that this would lead to a obese population of more than 1.5 billion by 2015.[20]

The people who are obese or abnormal body mass index in America was found to be around 150 million of adults. This is as reported by the American heart association[22]. Thus the facts of increasing number of people with obesity shows that metabolic syndrome is a factor that has to be given prime importance to prevent the on

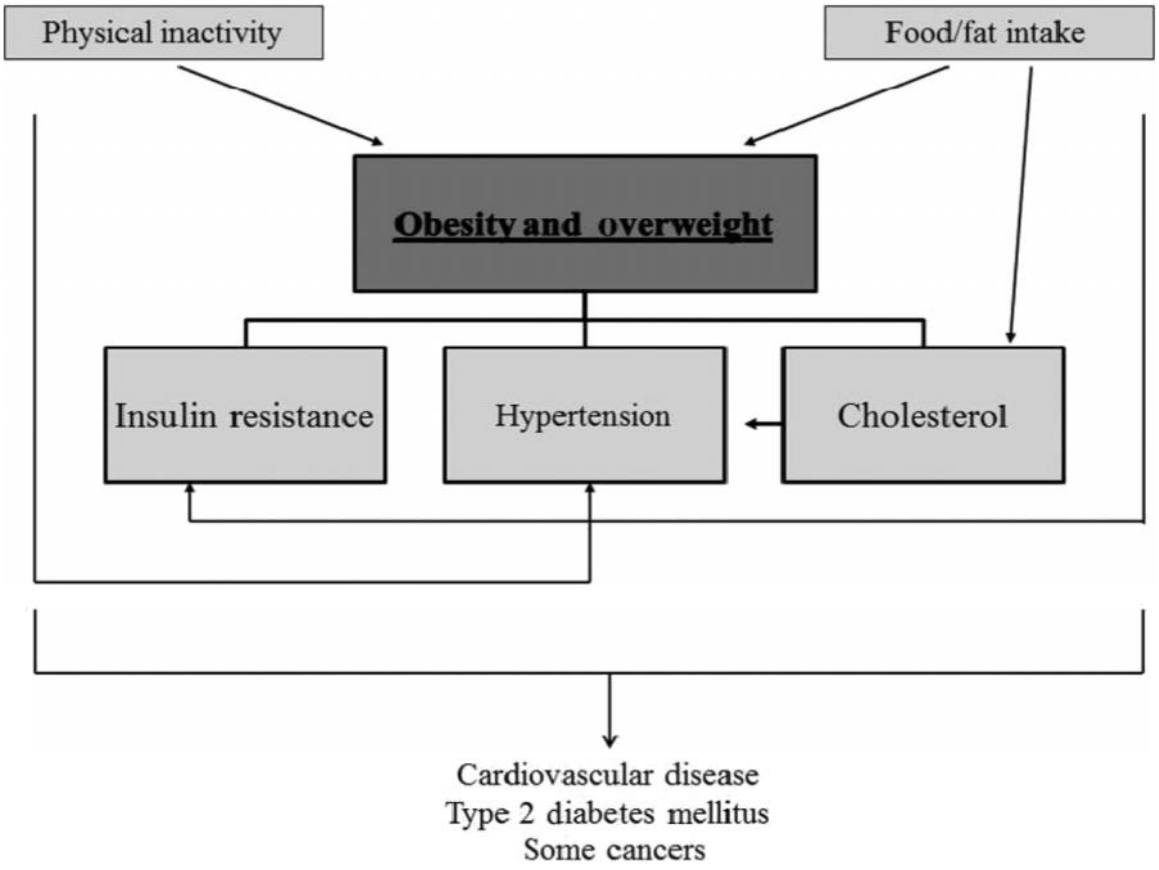


Figure 2. The above figure depicts the role of all components of metabolic syndrome. Central adiposity is the main component that leads to the arise of other components like elevated blood pressure, abnormal lipid profile, resistance to insulin, all these are found to be the components of metabolic syndrome. The figure shows that obesity and excess of weight is caused by increased consumption of food containing fat and inactive sedentary lifestyle. Excess fat intake leads to abnormal lipid profile and resistance to insulin. High blood pressure, increase in lipid profile values, resistance to insulin are caused by central adiposity.

INSULIN RESISTANCE

Syndrome X is term used to describe the occurring together of factors of metabolic syndrome. It is coined by Reaven at 1988. By 2030 it is estimated that around 300 million people in the world would develop diabetes .Though obesity is the important factor, it has been found that several genetic and environmental factors leads to the diabetes development and resistance to insulin[23]. In 2000 , it was found that around 200 million were having diabetes[24] and around 40% of the population was found to have insulin resistance[25]. Like obesity, insulin resistance is also an important factor leading to cardiovascular diseases. Pancreatic beta cell dysfunction is caused by high level of fatty acids[24]. High blood pressure, increased weight, high blood sugar, lipid profile abnormalities are found be caused by the factor insulin resistance[25].

DYSLIPIDEMIA

Dyslipidemia is considered to be another important component of metabolic syndrome along with the other factors like central adiposity and resistance to insulin. It includes many parts like elevated low-density lipoprotein (LDL), elevated triglycerides (TGL), fatty acids (FA), apolipoprotein (apo B)[27]. Albrink studied the link between the components like high blood pressure, elevated triglycerides, abnormal weight. Later many studies were conducted by him to study the relation between metabolic syndrome and lipid profile abnormalities[26]. The association

between atherosclerosis and resistance to insulin was established by Insulin Resistance Atherosclerosis Study after many years. It was found that the fatty acids not only plays a crucial role in causing resistance to insulin in people who are not obese but also it leads to elevation in the level of TGL causing hypertriglyceridemia[26].

Abnormal production of apo B, increased production of very low density lipoprotein, reduced metabolism of apo B and accelerated breakdown of HDL are factors causing dyslipidemia in people with metabolic syndrome. In metabolic syndrome patients, dyslipidemia may be caused by a combination of the overproduction of very-low-density lipoprotein, over-production of ApoB, decreased breakdown of ApoB and increased catabolism of HDL cholesterol. These changes are attributed to insulin resistance. All of these may be a consequence of IR [27]. Thus the most significantly seen components of dyslipidemia in patients with metabolic syndrome are elevated levels of low density lipoprotein, low HDL, high fasting and postprandial TGL[29]. (Fig. 3).

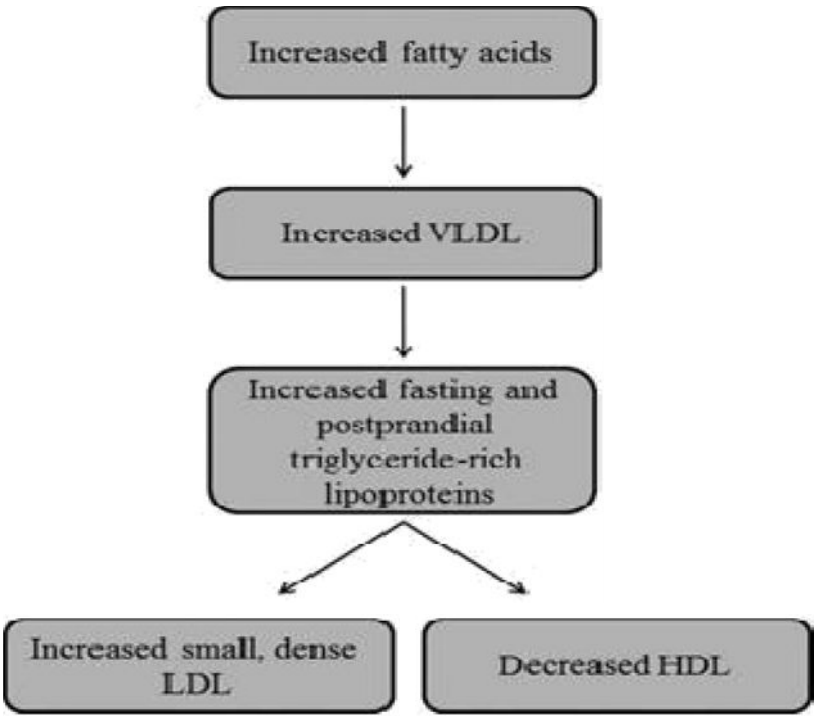


Figure 3 The occurrence of dyslipidemia from the state of resistance to insulin and obesity . Raised FA leads to increase in level of VLDL which inturn raises the TGL levels and thus it increases the levels of small dense LDL and low level of HDL.

HYPERTENSION

Studies conducted at many sites have shown that approximately 85% of people with metabolic syndrome have high level of blood pressure that is hypertension as the main driving factor for other components[30]. Most of the time the diagnosis of hypertension is not made early. This leads to the development of serious conditions like renal failure and heart diseases[31]. Almost 50% of people with elevated

blood pressure are found to be resistant to insulin[26]. Obesity and resistance to insulin is found to be the important cause for the high blood pressure[31]. The altered response to insulin is seen in people with insulin resistance. Normally, vasodilation occurs on introduction of insulin inside the blood stream due to the release of nitric oxide. But in people with insulin resistance, this response is not seen[26] and rather there occurs activation of renin angiotensin aldosterone system by the state of hyperinsulinemia, that causes high blood pressure and constriction of blood vessels[30].

A study conducted in U.S. showed that the frequency of high blood pressure is more high than other components in males with metabolic syndrome (41%) whereas in female the frequency was in the following order, Abnormal waist circumference in 52%, Low high density lipoprotein in 43% and then high blood pressure in 37%. It was conducted in adults of age above twenty years in 1999-2000 by NHANES[32].

METABOLIC SYNDROME

There are criteria for defining metabolic syndrome. They are modified NCEP-ATP III and IDF criteria.

INTERNATIONAL DIABETES FEDERATION CRITERIA

People were said to have metabolic syndrome according to IDF criteria, if the patient had central obesity that is abnormal waist circumference that is more than or

equal to 94 cm in male and more than or equal to 80 cm in female- along with other any 2 factors

(1) high TGL (≥150 mg/dl);

(2) low HDLC (<40 mg/dl in males and <50 mg/dl in females);

(3) high blood pressure (systolic BP ≥130 or diastolic BP ≥85 mmHg) or on drugs for hypertension;

(4) elevated fasting blood glucose (≥100 mg/dl)

MODIFIED NCEP-ATP III DEFINITION

When three or more of the components were present the person is said to have metabolic syndrome.

(1) central obesity (waist circumference >90 cm in males and >80 cm in females Asian-Indian criteria);

(2) high TGL (TG ≥150 mg/dl);

(3) low HDLc (<40 mg/dl in males and <50 mg/dl in females);

(4) elevated BP (≥130/85 mmHg);

(5) impaired fasting blood glucose (≥100 mg/dl)

METABOLIC SYNDROME AND ITS EFFECTS

Studies have enough evidence to say that people with metabolic syndrome are having increased risk of developing cardiovascular diseases and diabetes. The risk is 3 fold for heart diseases and 5 fold for developing diabetes. Though there are evidences to take it as risk factor , the relative risk for each person cannot be individualized so far for the heart diseases and diabetes[33].

HEART DISEASES

The cardiovascular diseases have become the leading cause of death in the developed countries as well as in the developing countries. So as early diagnosis of metabolic syndrome is warranted , since atleast the risk of developing cardiovascular diseases in future could be known[34]. The metabolic syndrome not only as a whole increases a person's risk to develop heart diseases but every single factor of metabolic syndrome is found to have an effect and act as a risk factor [35].

The metabolic syndrome's prevalence was studied in 2493 people, by using two criteria. It was found that according to NCEP ATP III criteria the prevalence was 16% and according to IDF criteria it was 21%. The study mainly analyzed the association between resistance to insulin, heart disease and metabolic syndrome. Follow up was done for a period of around 10 years, then it was found that 233 participants of the study died

from heart diseases leading to coronary artery diseases, stroke. The cardiovascular disease's incidence was studied and it was 16.6% as per NCEP ATP III definition and 14.6% as per IDF definition. Thus it was concluded that NCEP ATP III is better criteria with respect to predicting the cardiovascular disease than IDF criteria. Resistance to insulin acts as a risk factor to heart disease both as an individual factor as well as in combination with metabolic syndrome[36]. The predicting power of NCEP ATP III was found to be more in terms of cardiovascular diseases.

DIABETES MELLITUS

Diabetes mellitus is also a mixture of many metabolic conditions like abnormal lipid profile, overweight , elevated blood glucose and resistance to insulin similar to that if metabolic syndroeme[38]. The onset of diabetes is preceded by the factors like elevated lipid profile, resistance to insulin, high levels of insulin and abnormal weight in almost 85% of people. This condition is seen in almost 150 million people and might double by 2020[37].

Along with insulin the role of the hormone glucagon is being studied. It shows that the pancreatic beta cell failure is first preceded by dysfunction of alpha cells of the pancreas seen in case of people with diabetes mellitus[39]. The dysfunction of alpha cell is seen both in type 1 and type 2 diabetes. This loss of function leads either more secretion or reduction in the suppression of alpha cells in the diabetic patients. It was found that the

level of glucagon is high in people with diabetes leading to increased blood glucose and dysregulation of glucose metabolism[40]. Further studies have showed glucagon like peptide-1 (GLP-1) is also increased in these diabetic patients. Glucagon like peptide-1 causing glucagon and insulin regulation is an integrin that is being secreted in intestine. The level of glucagon like peptide-1 in people was studied in people with and without metabolic syndrome. it was found that the levels were higher in people with metabolic syndrome. The difference was statistically significant. It also had relation to every single factor of metabolic syndrome. These patients are more prone to develop heart diseases[41].

CANCER

There are studies which show that there exists a relation between the metabolic syndrome and the incidence of cancer. But there is no clear evidence to substantiate it. The cancer prevalence is found to increase worldwide. Almost 20% of the cancer death are attributed to obesity[42] .

RELATION BETWEEN CANCER AND OBESITY

Studies conducted so far has shown that association lies between the obesity and malignancies. Though the mechanism is not known clearly, the association is seen between malignancies of pancreas , kidney, ovary, breast , endometrium and esophagus[43,44]. Resistance to insulin that is seen in obese individuals leads to oxidative

stress and also increases the insulin like growth factor-1[44]. This is a possible mechanism that has been suggested for the occurrence of malignancies.

ASSOCIATION WITH INSULIN RESISTANCE

The mechanism for the development of malignancy in patient with insulin resistance has been studied. The increase in levels of insulin in the blood due to resistance leads to increase in blood glucose levels and increase in synthesis of glucose. The insulin also induces mitosis which inturn is affected by the reactive oxygen species that is produced in excess in people with high levels of blood glucose and high levels of fatty acids. Mutation occurs in the (DNA) deoxyribonucleic acid and leads to development of cancer. The high levels of insulin leads to reduction in sex hormone binding globulin and increase in insulin like growth factor. All the changes leads to carcinogenesis. These factors also leads to sex steroid secretion from the ovaries that leads to cancer in endometrium and breast[46].

MALIGNANCY AND LIPID ABNORMALITY

The low high density lipoprotein and high levels of low density lipoprotein is associated with increase in the odds of developing cancer. Studies have shown that around 2.3% drop in the chances of malignancy development if the level of high density lipoprotein increases by one mg/dl. The malignancy that could occur are gastrointestinal, prostrate and breast malignancies[47].

HYPERTENSION

PATHOPHYSIOLOGY OF HYPERTENSION

ENVIRONMENTAL AND PSYCOSOCIAL STRESS

Increase in blood pressure was observed in over the lifespan of people who settled in stressful urban region from traditional, calm rural environment[48]. Westernized people with low socio economic status have higher BP[49]. Type A or Coronary prone behavior pattern is linked with hypertension and risk of cardiovascular disease[50]. Anger arousing experiments have long been known to raise BP and increase vasoconstriction[51,52]. Other psychological factors related to increase in BP are power motivation (desire to dominate others), depression[53,54], hopelessness and pessimism . The effect of persistent hopelessness was equivalent to that of smoking 2.5 packs of cigarettes daily [55,56] . The stress buffers like social support, aerobic exercises and stress reduction interventions like bio feedback , relaxation training and cognitive behavioral therapies have been shown to lower B.P. with mixed success [57].

RENIN-ANGIOTENSIN SYSTEM (RAS)

In humans, rennin is coded for by a single gene in chromosome-1. It is secreted by juxta- glomerular cells in response to decrease perfusion. The rennin converts angiotensinogen to angiotensin I which is converted to angiotensin II by ACE.

Angiotensin II is thought to be responsible for most of the physiologic and pathophysiologic effects of RAS. Evidences are accumulating that ACE polymorphism due to insertion or deletion may be responsible at least a part to the pathology of essential hypertension[58-60].

ALDOSTERONE AND MINERALOCORTICOIDS.

Apart from the physiological role in retaining Na + and excreting K + in epithelial cell, Aldosterone has other less explored functions like vasoconstriction by acting on vascular smooth muscle, in brain to stimulate salt appetite and direct action on cardiac myocytes which may contribute to essential hypertension.

INSULIN RESISTANCE AND HYPERINSULINEMIA.

Though the relation between hyperinsulinemia and hypertension is well established the mechanisms by which it rises the BP remains doubtful and complex. The possible mechanisms include.

1. Insulin mediated salt retention [61,62].
2. Insulin activation of sympathetic nervous system [63,64].
3. Proliferative effects of insulin[65].
4. Non enzymatic glycation of interstitial tissues in arterial walls [66].
5. Insulin induced endothelial dysfunction leading to arterial stiffness.

REMODELLING OF RESISTANT ARTERIES IN HYPERTENSION.

Abnormalities of endothelial cells, smooth muscle cells, adhesion molecules and extra cellular matrix in vasculature may contribute to structural, mechanical and functional changes that reduce lumen diameter of small arteries (400 – 100 micron).There are two types of remodeling.

1. Eutrophic remodeling in which the cross sectional area of lumen is maintained
2. Hypertrophic remodeling in which the cross sectional area of lumen is increased.

Of these the Eutrophic remodeling occurs in essential hypertension.

ENDOTHELIUM IN HYPERTENSION

The two major endothelium derived vasoactive substances are Nitric oxide (NO) and Endothelin (ET).Nitric oxide is released from endothelium in response to shear stress and exerts vasodilating and antiproliferative effects on smooth muscle cells. It also inhibits the thrombocyte aggregation and leucocyte adhesion. The whole body Nitric oxide production in patients with essential hypertension is diminished under basal conditions[67,68]. Oxidative stress plays an important role in the pathogenesis of hypertension by oxidation of Nitric oxide to peroxynimine by superoxide anion thereby effectively reducing the bioavailability of Nitric oxide [69,70].

Endothelin exerts its major vascular effects, vasoconstriction and cell proliferation through ET A receptors on vascular smooth muscle cells. In contrast ET B receptor mediate vasodilation via release of NO and prostacyclin. Apart from the effects on blood vessels it also causes sodium retention and Renin release in kidneys , Aldosterone release from adrenals and hypertrophy and fibrosis of heart musculature.These effects may contribute to essential hypertension in human[71-73].

NATRIURETIC AND VASODILATORY PEPTIDES

The natriuretic peptides including ANP , BNP , CNP , DNP and urodilantin performs multiple functions like natriuresis, vasodilation , anti- proliferative effects , vascular remodeling and modulation of nor adrenergic and RAAS . In addition to these, the vasodilatory peptides like calcitonin gene related peptide (CGRP), substance P and Adrenomedullin regulates cardiovascular function in normal state and in hypertension.

THE KALLIKREIN – KININ SYSTEM

The kininogenic enzymes act on kininogen to form kinins. The kinins act via two receptors . The B1 receptor mediates inflammation , pain and fibrosis, the B2 receptor mediates the depressor , natriuretic , antitrophic and fibrinolytic functions via mediators like Eicasanoids , EDHF (endothelium derived hyperpolarizing factors) , NO, t-PA etc.

CONCEPT OF VASCULAR STIFFENING

A decreased distensibility of aorta and other large arteries or the loss of the windkessel function is known to be a cause for hypertension. This vascular Stiffening develops from complex interaction between structural and celluar elements of vessel wall . The structural components include 2 prominent scaffolding proteins , collagen and elastin . Dysregulation of the balance between the production and degradation of collagen and decreased production of elastin which contribute to vascular stiffness [74]. These vascular alterations are influenced by intrinsic factors [75] like hemodynamic forces and extrinsic factors like hormones, salt, glucose and lipids [76]. In addition to the structural components , cellular components also play a role in the pathogenesis of vascular stiffening . They are signals from endothelial cells like NO and Endothelin -1 and the vascular smooth muscle tone. The Vascular smooth muscle tone is modified by mechanostimulation and by paracrine mediators like Angiotensin II , ET -1 , oxidative stress and nitric oxide[77] .

DEFININIG HYPERTENSION

The multiple risk factor intervention trial (MRFIT) demonstrated a continuous and graded influence of both systolic and diastolic blood pressure of coronary heart disease mortality extending down to systolic blood pressures of 120 mm Hg. No definite level of blood pressure is there to define blood pressure.

Clinically "Hypertension may be defined as that level of Blood pressure at which the institution of therapy reduces Blood pressure related morbidity and mortality". The JNC VII classifies hypertension as follows

	SBP (mm Hg)		DBP (mm Hg)
Normal	<120	And	<80
Pre HT	120-139	Or	80-89
Stage 1	140-159	Or	90-99
Stage 2	>/ 160	Or	>/100
ISH	>/140	And	<90

TYPES OF HYPERTENSION

Based on the evaluation of patients, 80-95% of people with high blood pressure are said to have "Essential Hypertension (also called as Primary or Idiopathic Hypertension) . In the remaining 5-20 % , a specific underlying disorder causing the

elevation of blood pressure can be identified. These group is called " SECONDARY HYPERTENSION".Essential Hypertension tends to be familial and is likely to be the consequences of an interaction between environmental and genetic factors.

PATHOLOGIC CONSEQUENCES OF HYPERTENSION

Hypertension is a risk factor for atherosclerosis . It is an independent predisposing factor for heart failure,coronary artery disease, stroke, renal disease and peripheral arterial disease.

HEART

Hypertensive heart disease is the result of structural and functional adaptations of heart leading to left ventricular hypertrophy, diastoloic dysfunction, congestive heart failure, coronary heart disease, microvascular disease and cardiac arrythmias.

BRAIN

Hypertension is an important risk factor for brain infarction and haemorrhage . Hypertension is also associated with an impaired cognition in aging population . In malignant hypertension there is loss of cerebral auto regulation leading to hyperperfusion causing encephalopathy .

KIDNEY

Hypertension is an important risk factor for renal injury and ESRD. The atherosclerotic hypertension related vascular lesion in kidney primary affect the pre glomerular arterioles , resulting in ischemic changes in glomeruli and post glomerular structures . The injury may also be due to direct damage to the glomerular capillaries due to hyperperfusion . The pathology progress to glomerulosclerosis and eventually to gradual atrophy of tubules. The renal lesion associated with malignant hypertension consists of fibrinoid necrosis of afferent arterioles and sometimes the necrosis of glomerular tuft.

ARTERIAL DAMAGE

Hypertension is caused by changes in the blood vessels and the high blood pressure inturn leads to damage to the blood vessels. These damages leads to retinopathy, intermittent claudication.

PATIENT APPROACH & EVALUATION

Once the patient comes first the causes of hypertension are analyzed and the secondary causes of hypertension need to be screened. Then the factors that might lead to heart diseases to be screened. Most of the hypertensive patients have no specific symptoms referable to elevated BP . Only people with severe hypertension are known to have headache. Headache generally occurs only in patients with severe hypertension .

Patients mostly complain of morning, occipital headache. Dizziness, easy fatigability, palpitation and impotence may also be the presenting complaints of the patients.

MEASUREMENT OF BP

The person for whom the blood pressure is measured is made to sit in a room at comfortable room temperature as well as in a calm setting. The blood pressure measuring cuff has to be atleast 40% of the arm circumference . The arm in which the cuff is tied is to be placed at the level of heart. After inflating the cuff, it has to be deflated at a rate of 2mm of mercury/ second. Korotkoff sounds are used to measure the blood pressure. The tapping sound of the korotkoff sounds is taken as the systolic blood pressure . The point when the regular korotkoff sound is heard for last time is taken as the diastolic pressure.

Nowadays the oscillometric method is used for measuring the blood pressure. They measure the blood pressure every fifteen to thirty minutes. They are automated ambulatory machines. As per recommendations of JNC VII , the usage of ambulatory blood pressure monitoring for people with episodic hypertension, resistant hypertension and symptomatic hypertension. Thus ambulatory measurement is not used for all patients.

EXAMINATION OF THE PATIENT

Anthropometric measurements are made , that is height and weight of the patient is measured. The bloodpressure measurement is done in both right and left arm with the patient in sitting posture and also in standing and lying down in supine position. In

hypertension diagnosed before the age of 30 years, lower limb BP should be checked. All the peripheral pulses should be examined for rate, rhythm , volume and character. Heart rate should be recorded . Examination of the neck is done for thyroid gland . Fundus is also examined for signs of retinopathy. Auscultation is done for bruit over carotids,femorals and renal arteries is done. Cardiac auscultation for loud S2 and S4 Gallop is done. Palpation of the precordium is done for a heaving apical impulse.

LABORATORY TESTING

Basic laboratory testing for initial evaluation

RENAL	ELECTROLYTES	METABOLIC	OTHERS
Urine microscopy Urine albumin BUN Serum creatinine	Serum sodium Serum potassium Serum calcium	FBS Lipid profile	Hematocrit ECG

The aim of anti - hypertensive management by various means is to bring down and to maintain the BP below 140/90 mmHg. In diabetic patients the BP goal is 130/80 mmHg and in nephropathy patients it is125/75 mmHg.

DYSLIPIDEMIA

Dyslipidemia is the most prevalent and important modifiable risk factor for atherosclerosis . Proper treatment reduces the risk for cardiac death, non fatal MI, stroke, and peripheral vascular disease by 25 – 50 %. Despite these benefits only 20 % of adults meet national guidelines for cholesterol control.

Dyslipidemia is defined as abnormal lipid status. Common lipid abnormalities include elevated total cholesterol, LDL cholesterol, lipoprotein (a) and triglycerides , low level of HDL cholesterol and a preponderance of small density LDL particles. These abnormalities occur alone or in combination.

PREVALANCE, RISK AND SCREENING

Approximately 50% of US adults have an elevated total cholesterol. In a vast majority of the patients with atherosclerotic vascular disease there is some form of dyslipidemia even though their total cholesterol is normal. Elevated total cholesterol , LDL c and low HDL c levels are major modifiable risk factors for coronary heart disease and other forms of atherosclerotic vascular disease. ATP III recommends a routine lipoprotein analysis for all adults aged 20 years. Screening should be repeated every 5 years. Since dyslipidemia is an asymptomatic condition, early recognition and treatment improves prognosis.

EFFECT OF THERAPY

Several large randomized , placebo controlled trials of statin therapy have shown reduction in cardio vascular morbidity and mortality. A meta-analysis of more than 330 interventional trials has shown that for every 1 % total cholesterol, mortality is reduced by 1.5 % and for each 1 % reduction in LDL c and for each 1 % increase in HDL c, the risk for cardiovascular events is reduced by 2 % and 3 % respectively [78].A number of studies with serial angiography also show that the increase in stenosis was 1-3 % less per year in aggressively treated patients than in placebo. The lower level of LDL c maximum benefit extends to as low as 50 -70 mg/dl for high risk patients.

LDL CHOLESTEROL GOALS

The LDLc goals are individualized depending on their CHD risk

MAJOR RISK FACTORS THAT MODIFY LDL GOALS [79]

The first step in estimating the LDL c goal is counting the number of risk factors

Cigarette smoking	Age (male > 45 yrs ; women > 55 yrs)
Hypertension	Obesity (BMI > 30 kg/m2)
Diabetes mellitus	Physical inactivity
HDL_c <40 mg/dl	Atherogenic diet
Cardiovascular disease in family with early onset	Emerging risks

The next step in calculation of the 10 year risk of coronary heart disease using the Framingham heart study risk score. Based on these, individuals are classified in 5 groups and their LDL c goals are set.

VERY HIGH RISK GROUP

- LDL c goal < 70 mg % [80]
- 10 year risk for major coronary event
- 20 % Established CHD
- CHD equivalents
- Diabetes mellitus
- Multiple cardiovascular risk factors

HIGH RISK

- LDL c goal < 100 mg % ,
- optional goal < 70 mg %
- Coronary heart disease
- Coronary heart disease risk equivalents
- 10 year risk > 20 %

≥ 2 risk factors

MODERATELY HIGH RISK

- LDLc goal < 130mg%,
- optional< 100 mg %
- 10 year risk 10- 20 %

≥ 2 risk factors

MODERATE RISK

- LDL c goal < 130 mg %
- 10 year risk < 10 %

≥ 2 risk factors

LOWER RISK

- LDL c goal < 160 mg %

0-1 risk factors

NON HDL CHOLESTEROL [81]

Non – HDL c is calculated by subtracting HDL c from TC.The primary goal of therapy for persons with dyslipidemia is LDL-c lowering . A secondary goal of non - HDL c is set by ATP III guidelines.

High / Very high risk - < 130 mg/dl.(optional < 100 mg / dl)

Moderately high risk - < 160 mg/dl.(optional < 130 mg / dl)

Moderate risk - < 160 mg / dl

Low risk - < 190 mg / dl

TREATMENT OF DYSLIPIDEMIA

THEREPEUTIC LIFE STYLE CHANGE [82-84].

This includes Diet, physical activity, weight reduction and smoking cessation . In many individuals this could reduce total cholesterol by < 10%.

DRUGS

DRUGS	MECHANISM	LIPID LOWERING
Statins	HMG CoA reductase inhibitors	LDL c : 18 – 55 % HDL c : 5 – 15 % TG : 7 – 30 %
Ezetimibe	Decrease cholesterol absorption	LDL c : 18 – 20 % HDL c : 1 – 5 % TG : 5 – 11 %
Niacin	Decrease production and release of VLDL	LDL c : 5 – 25 % HDL c : 15 – 35 % TG : 20 – 50 %
Bile acid sequesterants	Prevents enterohepatic circulation of BA	LDL c : 15 – 30 % HDL c : 3 – 5 % TG : unaffected
Fibrates	Increase LPL activity	LDL c : 5 – 20 % HDL c : 10 – 35 % TG : 20 – 50 %
Omega 3 fatty acids	DHA and EPA reduce VLDL production	LDL c : 44 % incr HDL c : 9 % TG : 45 %

HIGH BLOOD PRESSURE AND ABNORMAL LIPID PROFILE -

The various factors of metabolic syndrome have found to be interlinked that is one factor leads to the other and have a crucial role in other diseases onset. Similarly, the abnormal lipid profile is also related to the development of increase in blood pressure in many patients. So studies are conducted not only to study their relation but also to know the effects of abnormal lipid profile on blood pressure. The effect of lipid lowering agents on blood pressure is also being studied.

ROLE OF LIPIDS IN THE PATHOGENESIS OF HYPERTENSION

The mechanism by which the lipid profile abnormalities could lead to increase in blood pressure is being studied. This shows that the endothelial lining of the blood vessels are damaged by the lipid abnormalities and increase in blood pressure is also caused by the dysfunction in the vasomotor activity. This endothelial dysfunction is mainly caused by the atherogenic lipid profile abnormalities[85]. Endothelial damage causes impairment in the expression of endothelin-1 and endothelin A&B receptor expression[86]. The vasodilatory effect of nitric oxide and its production is also impaired. These changes leads in increase in the blood pressure.

The relation between hypertension and dyslipidemia is studied and shows that there is more increased expression of angiotensin-I. This is established by Nickenig and

Harrison[87,88] . And also over activity of sympathetic system is seen patients with resistance to insulin and lipid profile abnormalities[89].

CLINICAL TRIALS IN PATIENTS WITH HT & DYSLIPIDEMIA

ASCOT-LLA TRIAL[90]

(Anglo –Scandinavian cardiac outcome Trial-Lipid lowering arm)

In the above study there were two groups, one who received atorvastatin 10mg and other who received placebo.10,305 men and women aged 40-79 years with high blood pressure and with more than or equal to three risk factors were distributed randomly in the two groups. Non fatal myocardial infarction, cardiovascular diseases, death are taken as the primary end point of the study.

The Atorvastatin arm of the trial was stopped prematurely at 3.3 years due to a significant 36% reduction in the primary end point. Benefits were apparent in the first year . Atorvastatin also reduced fatal or non fatal stroke by 27%, total cardiovascular events by 21% and total coronary events by 29 % . At 1 year Atorvastatin reduced total and LDL cholesterol by 24% and 35% respectively.

ALLHAT – LLT [91]

(Anti hypertensive and lipid lowering treatment to prevent heart attack trial) 10,355 men and women aged $\geq$55 years with stage 1 and 2 hypertension , $\geq$ 2 additional

coronary heart disease risk factor and LDL c 120 -189 mg/dl (100 – 129 mg/dl in patients with CHD) were randomized to Pravastatin 40 mg/dl or usual care . Mean follow up for 4.8 years.

The results showed Pravastatin did not significantly reduce all cause mortality (primary end point) or coronary Heart disease events (relative risk 0.91 p=0.16). Lack of benefit is attributed to substantial (30 %) use of statins in the placebo group , resulting in a modest 17 % differential in total cholesterol between Pravastatin and usual case groups.

PHYSICIAN HEALTH STUDY [92]

It was a study that was conducted among the physicians to estimate the primary prevention of coronary heart disease and malignancy by aspirin and placebo beta carotene. It was conducted as a double blinded trial and also randomized and controlled trail. Physicians of 40-84 years of age were included in the study. Then the individuals who did not have coronary heart disease, ,malignancy , high blood pressure were taken and the lipid profile of them were tested. They are the total cholesterol, high density lipoprotein , non HDL and the ratio between the total cholesterol and high density lipoprotein was calculated. Later on following these people over 14 years showed that persons with high levels of these parameters developed hypertension whereas the persons with low range of these parameters did not develop hypertension that significantly.

Reduction in the risk of developing hypertension was seen in patients with higher value of high density lipoprotein which was 32%. Thus this study conducted in healthy physicians showed that lipid profile abnormalities lead to the development of high blood pressure.

LIPID PROFILE IN HYPERTENSIVE NIGERIANS [93]

Lipid profile studied in 150 hypertensive patients aged 30 – 59 and 30 years and socio economic status matched normotensive controls using standard laboratory techniques . Of the hypertensive patients 54% were females and 46% were males.Hypertensive patients have significantly higher lipid profiles except for HDLc which did not showed any difference in the two groups.

LIPID PROFILE OF HYPERTENSIVE PATIENTS IN SPAIN [94]

In a Spanish hospital a study was conducted in people of mean age of forty five years. The people were divided into two groups with 23 healthy people and 27 people with high blood pressure. The body mass index of the patient were matched.the persons were subjected to many analysis . Statistical analysis was done using the student's t test and Welch's test. The values of triglycerides, uric acid, total cholesterol, creatinine and chloride was higher in hypertensive people.

LIPID PROFILE OF HYPERTENSIVE PATIENTS IN BANGLADESH [95]

This prospective study carried out in department of bio chemistry and molecular biology , university of Rajshahi , Bangaladesh. 60 human subjects of age ranging from 33 – 60 years were studied . Among these 40 were hypertensive and 20 were normotensive . Their lipid profile results were collected and analyzed statistically. The total cholesterol (241.25 vs 182.14) , triglycerides (184.77 vs 142.73) and LDL c (154.32 vs 105.73) were significantly higher and HDL c (32.91 vs 42.88) was significantly low among hypertensive patients.

MULTICENTRIC HYPERTENSIVE POPULATION STUDY IN UAE [96]

Study conducted in 162 hypertensive and 112 normotensive matched for age , gender , & ethnicity to determine ET-1 , NO, lipid profile. Levels of VLDL and TG were significantly higher ($P < 0.01$) in hypertensive. In contrast total cholesterol ($P < 0.01$) and LDL c ($P < 0.001$) were lower among hypertensive. ET-1 and NO were significantly higher in hypertensive.

MANAGEMENT OF HYPERTENSION IN METABOLIC SYNDROME

Main line of management in patients with hypertension and metabolic syndrome is the lifestyle changes. The changes that are warranted are smoking cessation, quitting alcohol, physical activity, reducing the body weight, change in diet including salt restricted diet, calorie restricted diet. These non-pharmacological changes are alone not

enough to maintain the normal level of blood pressure[97,98].

Pharmacological therapy used to treat high blood pressure are selected in such a way they also exert some effect on the other components of metabolic syndrome. Thus the drugs that are chosen are the one which act on the rein angiotensin aldosterone system, angiotensin convertase inhibitors, (ARBs) angiotensin receptor blockers, central sympatholytic agents[99,100].

Studies conducted on these drugs have shown that the drug of first choice is generally the drugs acting on renin angiotensin system. These agents have inhibition on sympathetic system and also increase the sensitivity to insulin[98]. Drugs like imidazoline which belong to the class of central sympatholytic agents found to have many favourable effects. The drug by inhibition of sympathetic outflow to the skeletal muscles produces an increase in insulin sensitivity[101].

Thus considering the various effects of hypertension in patients with metabolic system , the hypertension is managed by targeting on many metabolic factors.

MATERIALS AND METHODS

Patients newly detected as hypertensive in hypertensive clinic, medical out patient department and ward of Government Vellore Medical College Hospital were taken. Study period is from October 2015 to September 2016.

The study design is cross sectional study.

STUDY GROUP

The study included newly detected hypertensive patients of age group 31-75 years.

PATIENTS WHO WHERE EXCLUDED FROM STUDY

1. Patients who are already known hypertensives and on drugs
2. Patients with secondary hypertension
3. Newly diagnosed hypertensive patients with one or more complications like CVA, CAD, renal, hepatic and other endocrine diseases
4. Patients with secondary cause of obesity
5. Hypertensive patients who are alcoholics
6. Patients on steroidal or other medications likely to cause elevated plasma glucose

7. Any acute illness

8. Pregnant women

9. Patients not willing to participate in the study are excluded from the study

The participants were given clear and detailed information about the study. Then informed consent was obtained from the participants. Following which the participants were subjected to an interview and analysis for exclusion criteria. Patients who were meeting the inclusion criteria and did not fit in any exclusion criteria were selected. These selected patients participated in the study.

Totally 150 newly detected hypertensive patients were included in the study. History regarding the patient's smoking, alcohol consumption, occupation , daily activities and hypertension in family members were asked in detail.

The patients socioeconomic status was assessed and classified according to modified kuppuswamy scale (2007). Metabolic equivalent of task was used to classify the patients as sedentary and non sedentary. Participants with daily physical activity of MET of < 1.5 was considered to be sedentary as per WHO.

Participants who smoke more than or equal to 5 cigarettes per day were taken as smokers.

MEASUREMENTS

WAIST CIRCUMFERENCE

The waist circumference is measured midway between the costal margin and iliac crest. And the measurement is taken after asking the patient to take two normal breaths and at the end of second breath measurement of waist circumference is taken.

HIP CIRCUMFERENCE

Hip circumference is measured with tape parallel to the floor around the widest portion of the buttocks, with the patient wearing little clothing.

Waist – hip ratio was calculated. Body mass index (BMI) was calculated by using the formula,

BMI = body weight (kg) / $[height]^2$ (metres)

OBESE

Participants with waist circumference for women ≥80 cm and men ≥90cm, waist hip ratio for women ≥0.85 and men ≥0.90, and BMI of ≥ 25 kg / m^2 were considered obese.

Blood pressure was measured with patient in sitting posture and arm at the level of heart. After 30 minutes of rest and in the right arm of the patient blood pressure was measured. For preceding six hours the patient was abstained from caffeine intake and

smoking. The blood pressure was again measured at least 24 hours later and average of the two readings was taken.

BLOOD COLLECTION

After an overnight 12 hours of fasting, venous blood sample of five ml was collected for the purpose of investigations.The method used to determine total cholesterol (TC), triglycerides (TGL) and HDL c is enzyme calorimetric method. Friedeward formula is used to calculate LDL-c and VLDL-c.

$$LDL\text{-}c = TC - (HDL + VLDL)$$

Participants with total cholesterol value ≥ 200 or triglycerides value ≥ 150 or

LDL –c value ≥ 130 or HDL-c ≤ 40 were considered to have dyslipidemia.

Metabolic syndrome is said to be present , when 3 out of 5 criteria is present according to modified NCEP ATP III.

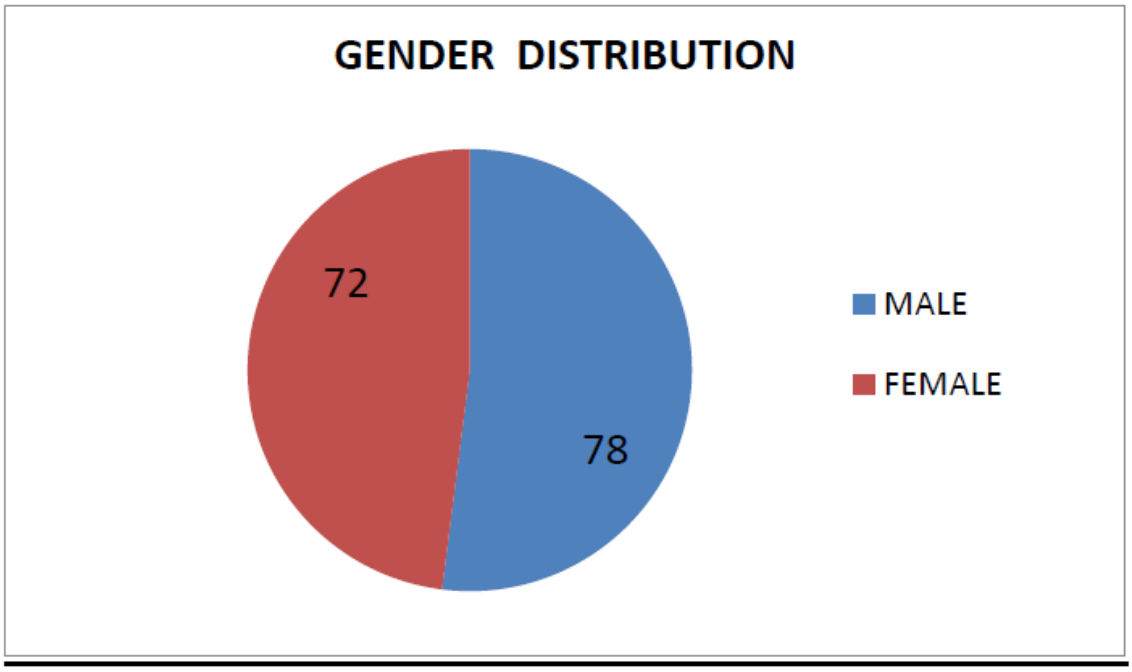
The statistical analysis is done using SPSS16 software. A p value of <0.05 is taken as significant. The influence of various factors on metabolic syndrome is studied. The statistical significance is studied using chi-square test. ANOVA is used to analyze the effect of multiple factors on metabolic syndrome. The mean values of lipid profile was calculated and the significance of difference of mean was calculated by using unpaired test.

RESULTS AND ANALYSIS

The 150 newly detected hypertensive individuals are studied and analysed in our study. There are 78 (52%) males and 72 (48%) females in our study.

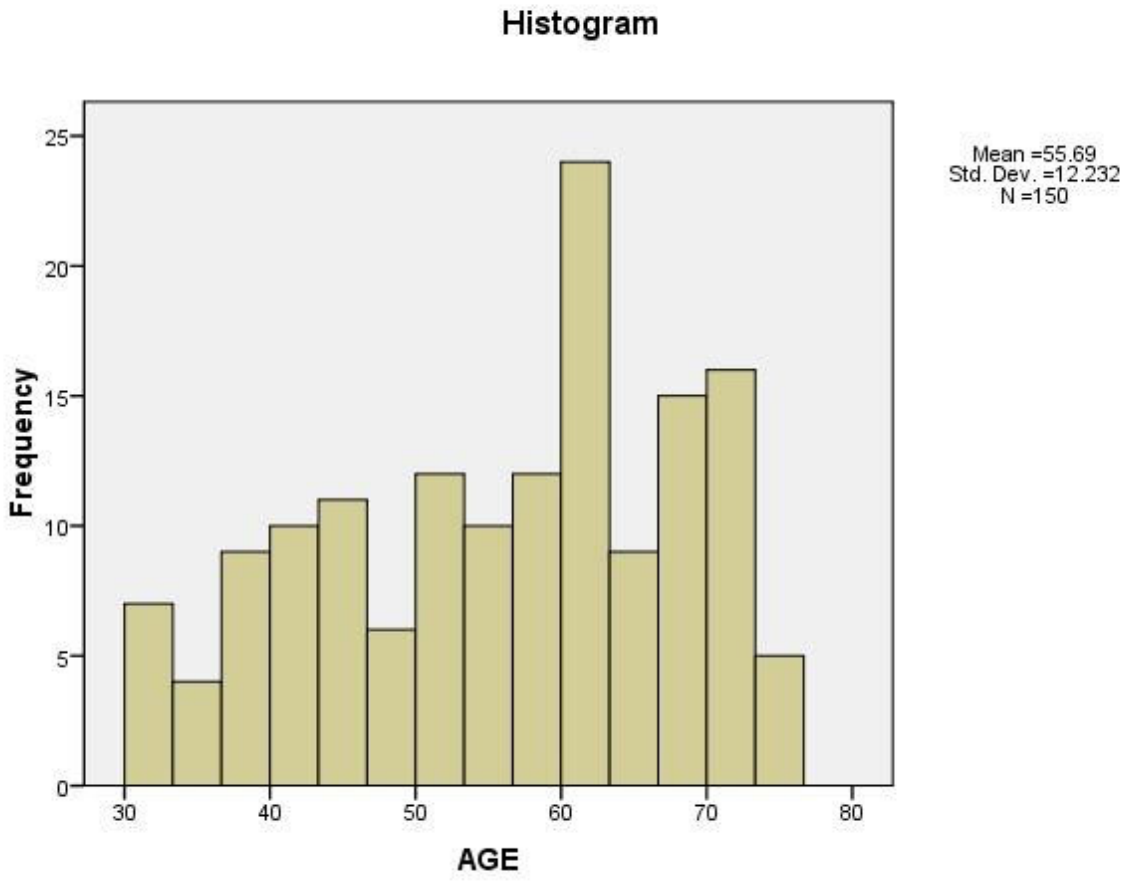
GENDER DISTRIBUTION

GENDER	NO OF PATIENTS
MALE	78
FEMALE	72



There is no statistically significant difference in gender distribution in our study.

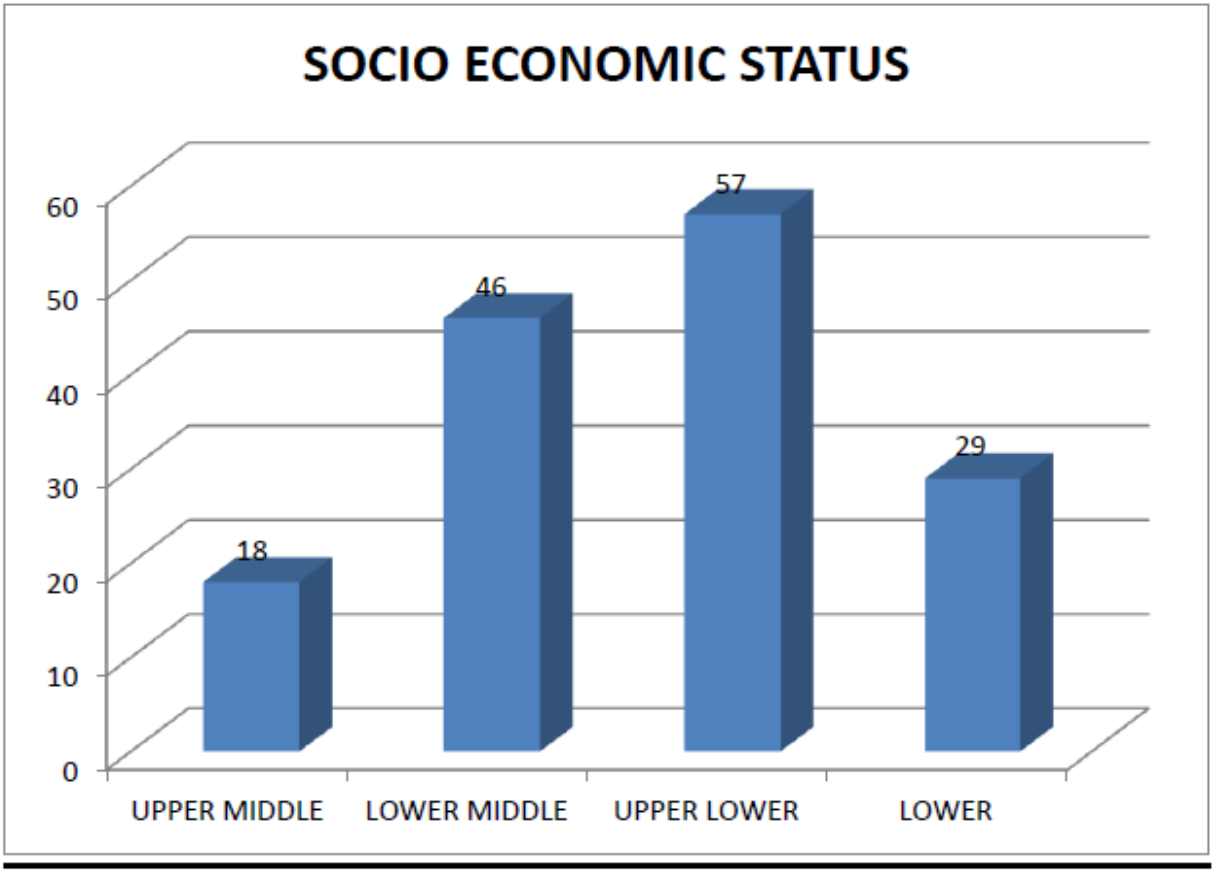
AGE DISTRIBUTION



The age distribution of our study population has more people belonging to 60-70 age group.

SOCIO ECONOMIC STATUS DISTRIBUTION

SOCIOECONOMIC STATUS	NO OF PATIENTS
UPPER MIDDLE	18
LOWER MIDDLE	46
UPPER LOWER	57
LOWER	29

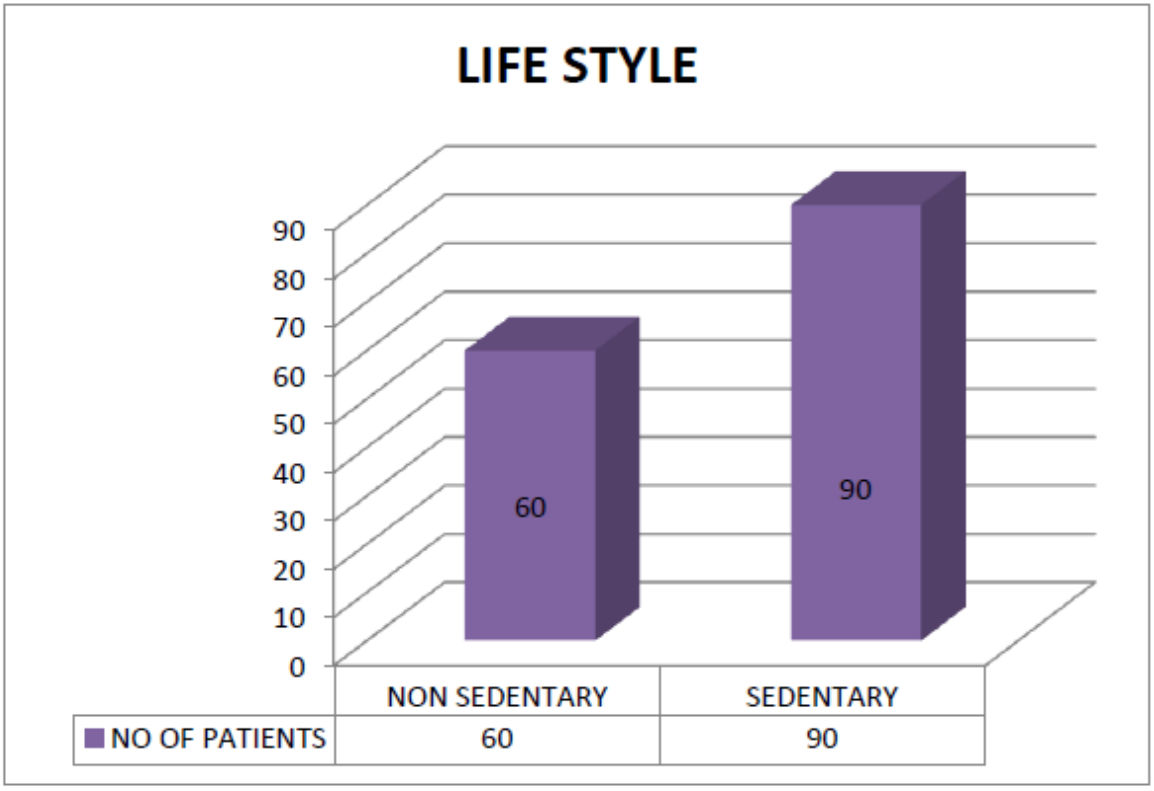


The distribution of the study population in various socioeconomic classes

according to modified kuppuswamy scale (2007) is 18 patients belong to (class 2) upper middle class, 46 patients belong to lower middle class (class 3), 57 patients belong to upper lower class (class 4) and 29 patients belong to lower class (class5). Most of the patients belong to class 4.

LIFE STYLE DISTRIBUTION

LIFE STYLE	NO OF PATIENTS
NON-SEDENTARY	60
SEDENTARY	90

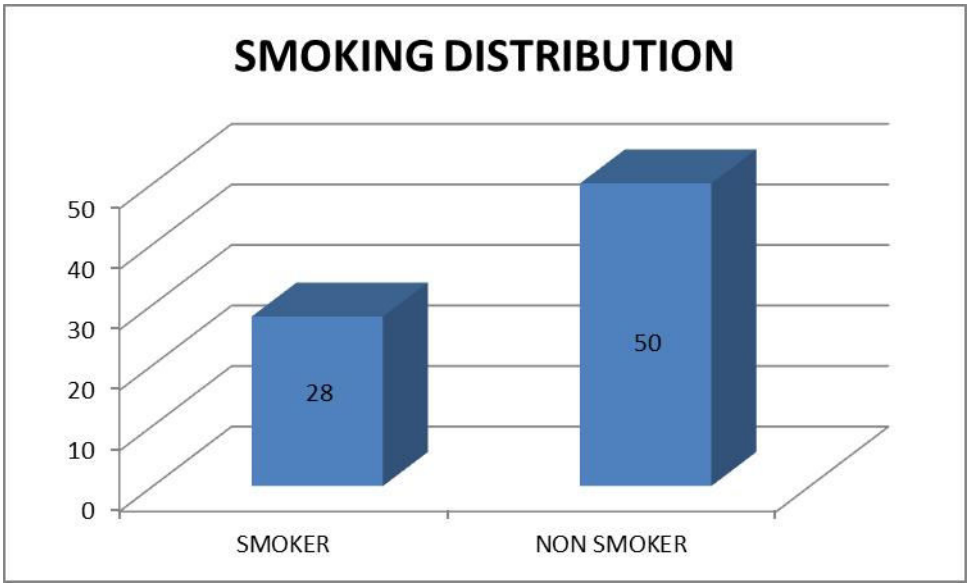


The study population is divided into two groups as sedentary and non-sedentary

people based on their daily physical activity. There are 90 patients belonging to sedentary group and 60 patients belonging to non-sedentary group.

SMOKING DISTRIBUTION

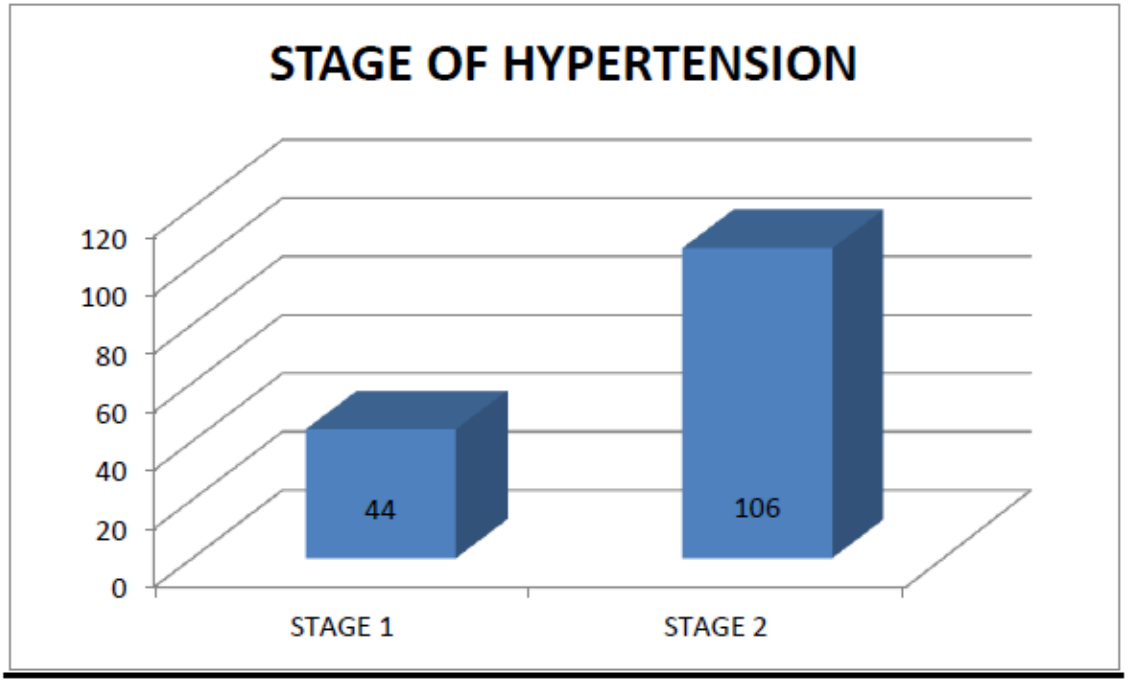
SMOKING HABITS	NO OF PATIENTS
SMOKERS MALE	28
NON SMOKERS MALE	50



The smokers and non-smokers in the study population comprises of 28 smokers and 50 non-smokers male.

STAGE OF HYPERTENSION

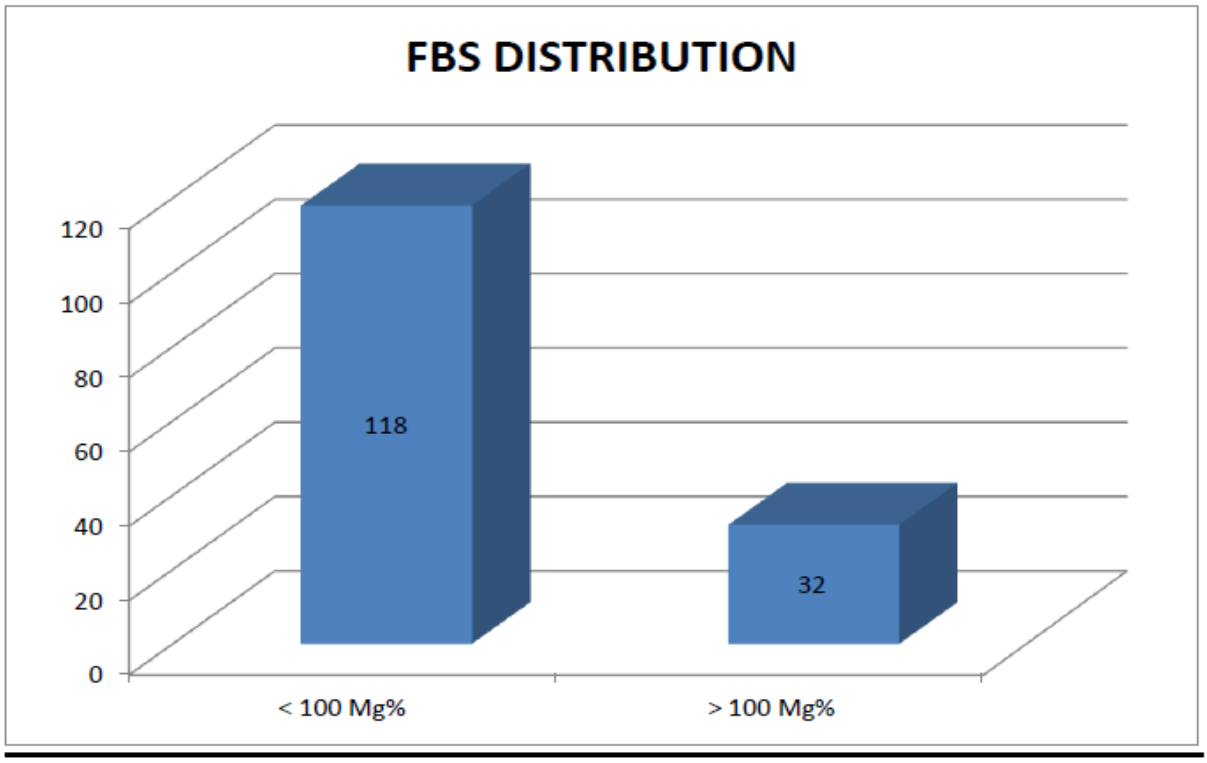
STAGE	NO OF PATIENTS
STAGE 1	44
STAGE 2	106



The hypertensive patients of our study population on grouping into stage 1 and stage 2 according to JNC 7, there are 44 patients belonging to stage 1 and 106 patients belonging to stage 2 of hypertension.

IMPAIRED FASTING BLOOD SUGAR DISTRIBUTION

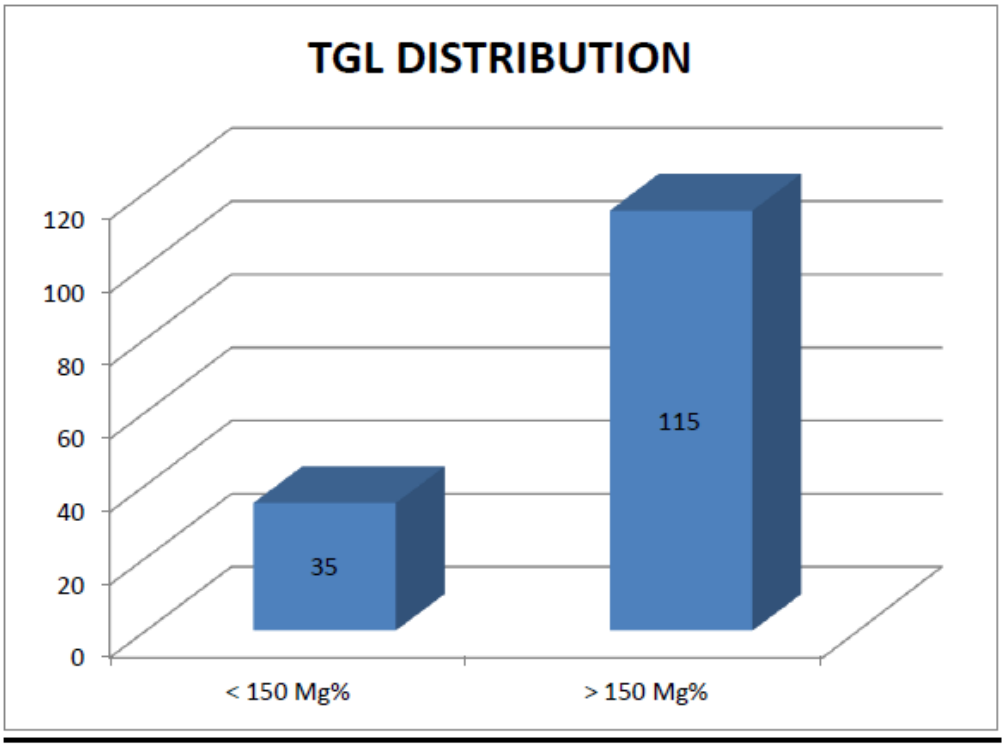
FBS	NO OF PATIENTS
< 100 Mg%	118
> 100 Mg%	32



The impaired fasting blood glucose (>100mg/dl) is found to be present in 32 patients (21.33%) and fasting blood glucose is found to be normal (<100mg /dl) in 118 patients (78.66%).

TGL DISTRIBUTION

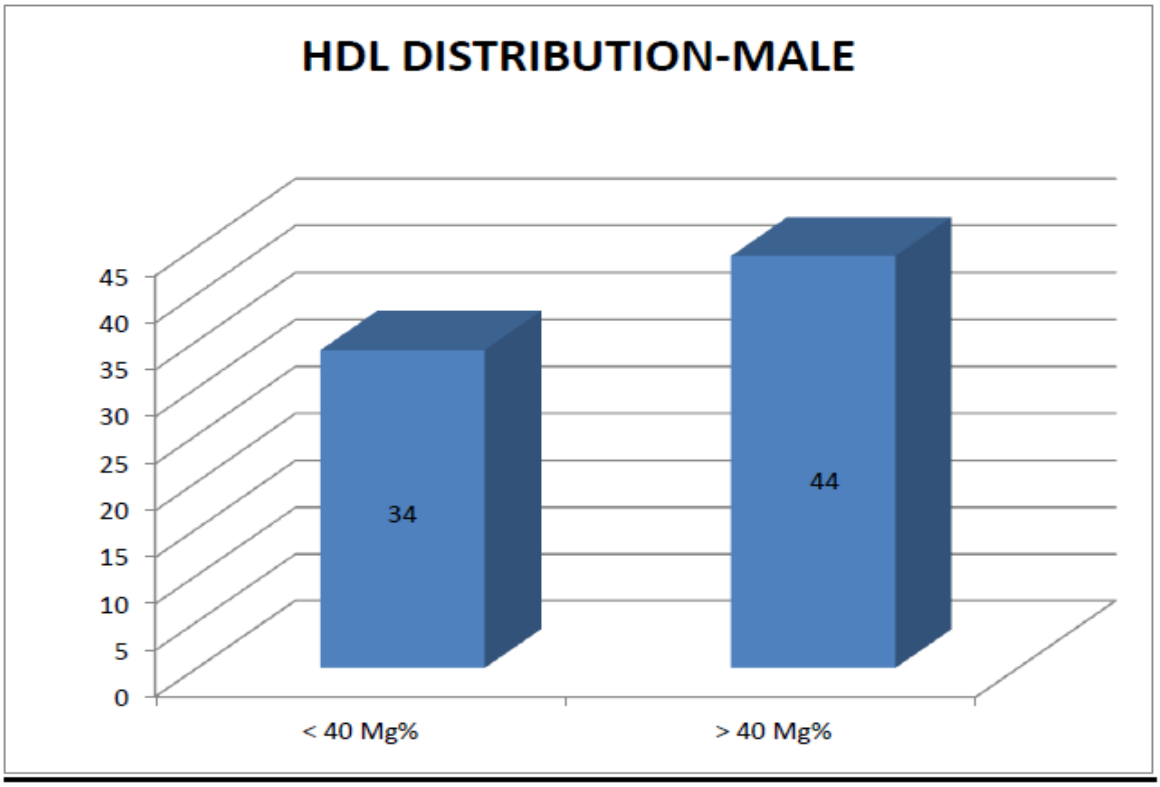
TGL	NO OF PATIENTS
< 150 Mg%	35
> 150 Mg%	115



Elevated triglycerides that is greater than 150mg/dl is seen in 115 (76.66%) patients. Normal triglyceride is seen in 35 patients (23.33%).

HDL DISTRIBUTION IN MALE PATIENTS

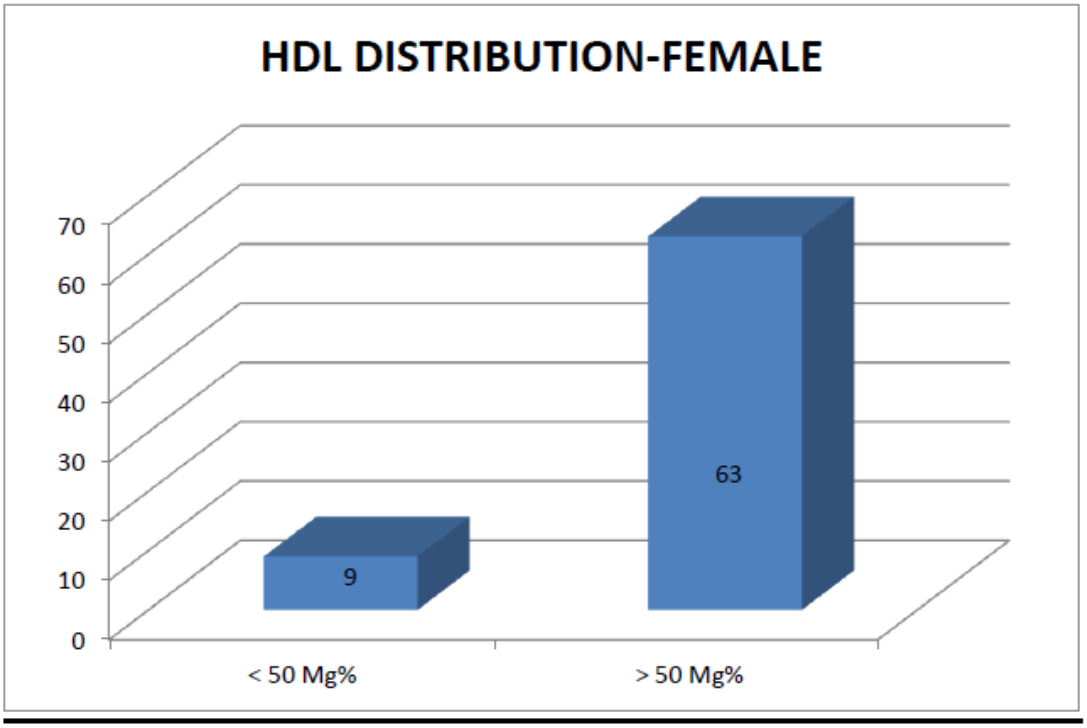
HDL	NO OF PATIENTS
< 40 Mg%	34
> 40 Mg%	44



The number of patients with low high-density lipoprotein value <40mg/dl is 34 (43.58%). The number of patients with high-density lipoprotein value above 40mg/dl is 44 (56.42%).

HDL DISTRIBUTION IN FEMALE PATIENTS

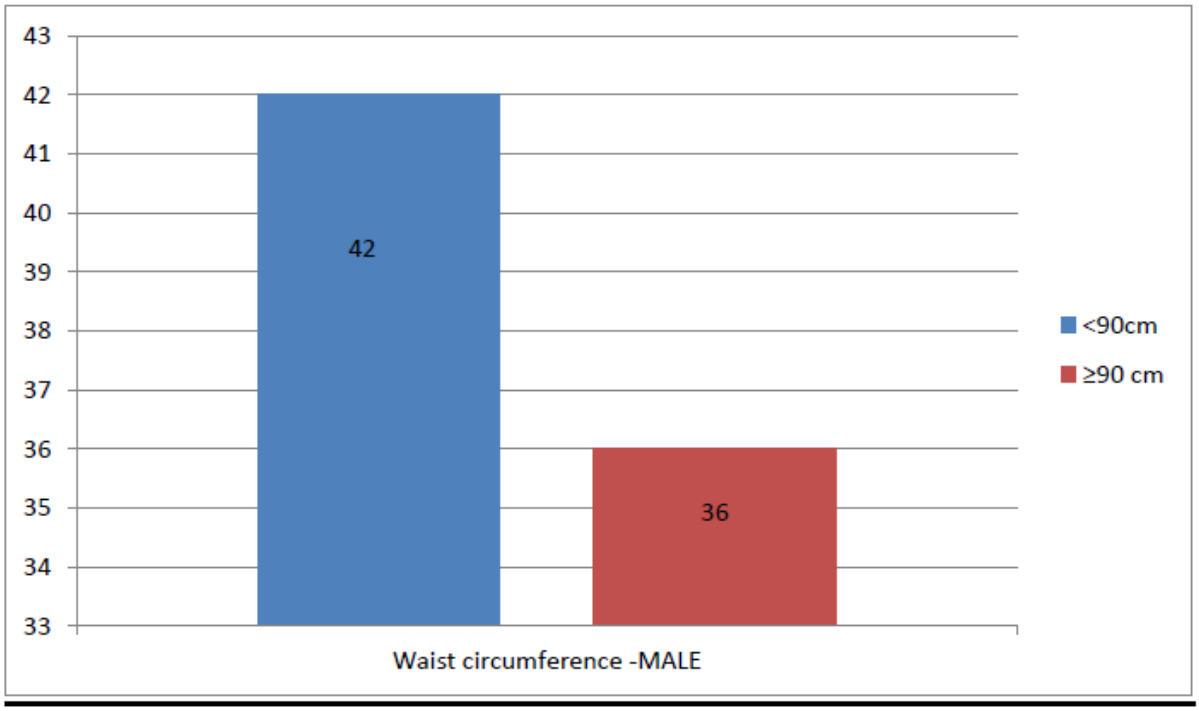
HDL	NO OF PATIENTS
< 50 Mg%	9
> 50 Mg%	63



The number of patients having high-density lipoprotein value < 50 mg/dl is 9 (12.5%). The number of patients having high-density lipoprotein value >50 mg/dl is 63(87.5%).

WAIST CIRCUMFERENCE IN MALE PATIENTS

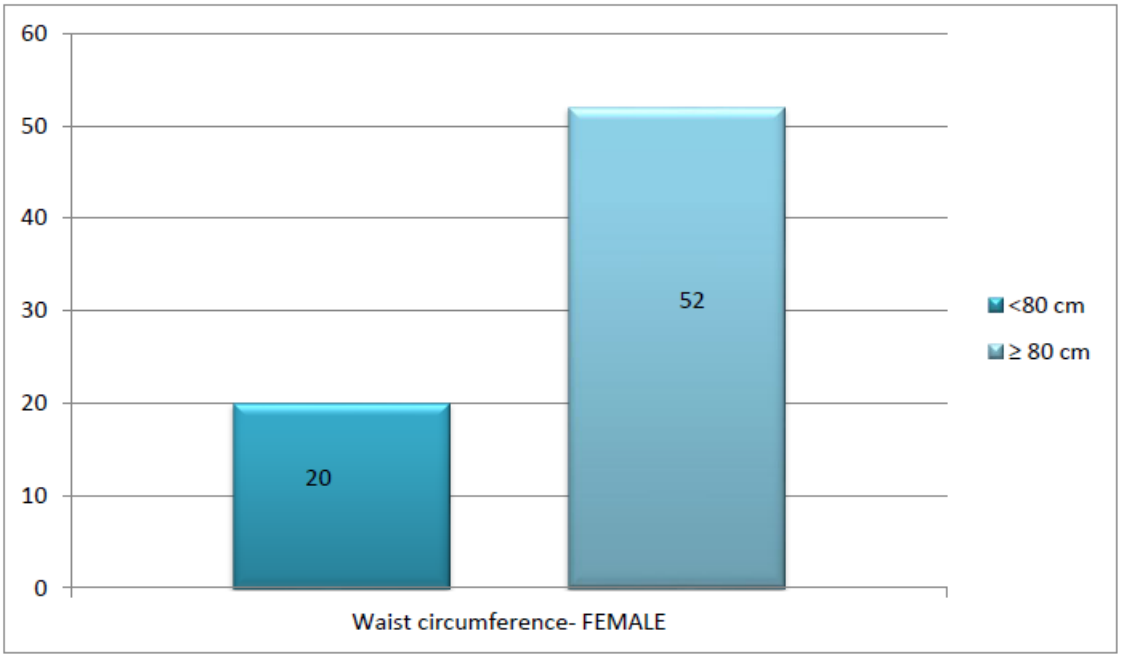
WAIST CIRCUMFERENCE	NO OF PATIENTS
<90 cm	42
≥90 cm	36



The waist circumference of ≥90cm for male is seen in 36 patients (46.15%). The waist circumference of newly detected hypertensive males in our study is 42 (53.85%).

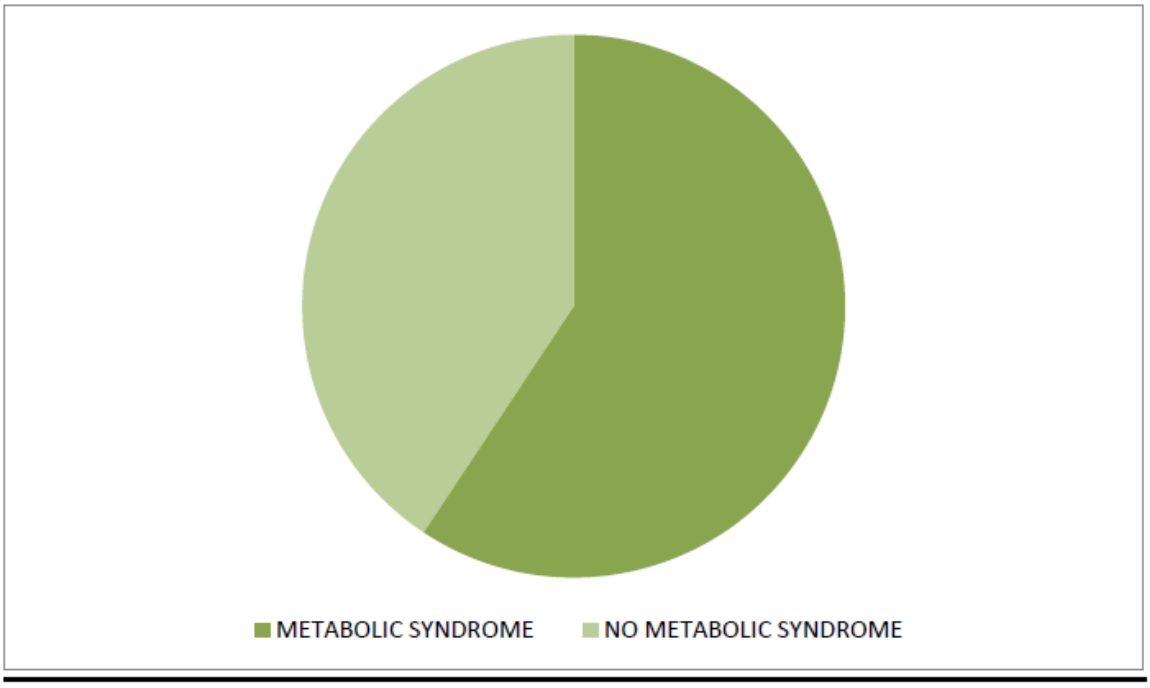
WAIST CIRCUMFERENCE IN FEMALE PATIENTS

WAIST CIRCUMFERENCE	NO OF PATIENTS
<80 cm	20
≥80 cm	52



The number of patients with waist circumference <80 cm in female in newly detected hypertensive in our study population is 20(27.78%). The number of patients with waist circumference ≥80 cm is 52 (72.22%).

PREVALENCE OF METABOLIC SYNDROME

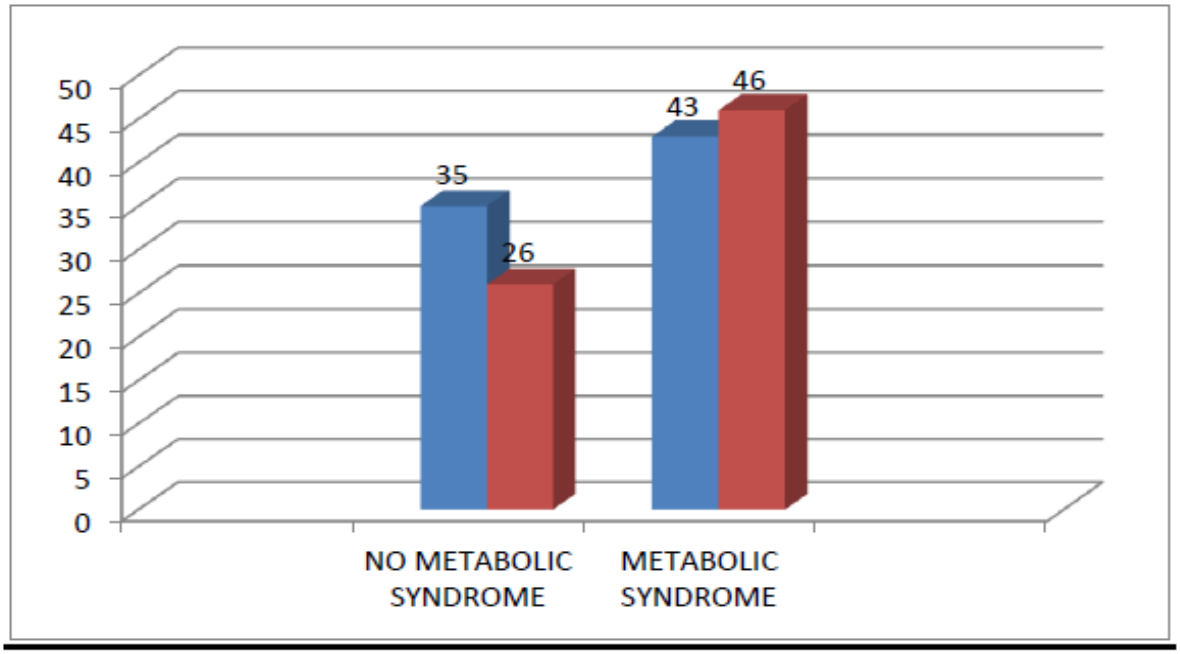


The prevalence of metabolic syndrome studied in 150 newly detected hypertensive individuals showed that 59.33% of newly detected hypertensive individuals has metabolic syndrome.

GENDER Vs METABOLIC SYNDROME

GENDER	NO METABOLIC SYNDROME	METABOLIC SYNDROME	TOTAL
MALE	35	43	78
FEMALE	26	46	72
	61	89	150

P VALUE=0.275

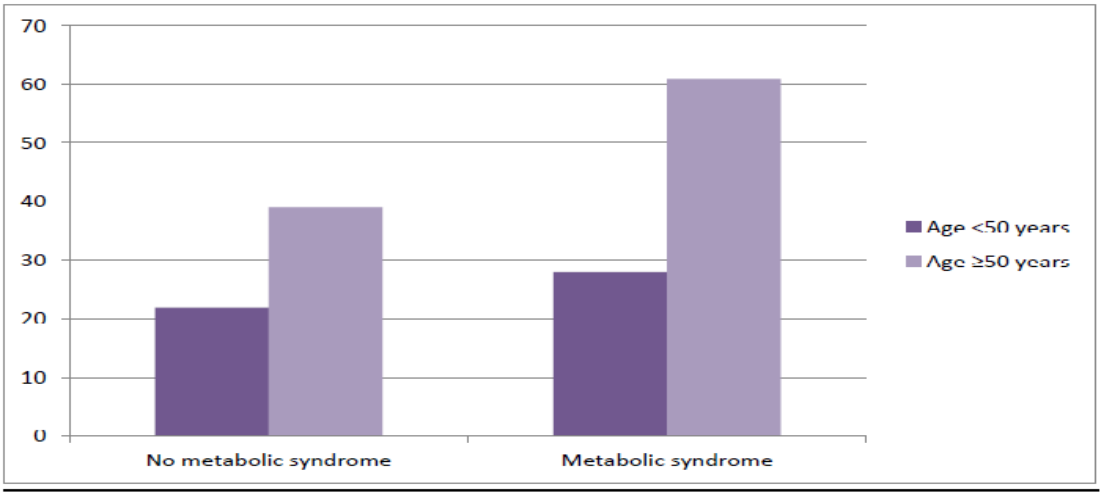


The number of patients with metabolic syndrome among male and female hypertensive patients are 43 (55.12%)and 46 (63.88%) respectively. There is no statistical significant difference in prevalence between male and female population.

AGE Vs METABOLIC SYNDROME

AGE	NO METABOLIC SYNDROME	METABOLIC SYNDROME	TOTAL
<50 YRS	22	28	50
≥50 YRS	39	61	100
TOTAL	61	89	150

P VALUE=0.557

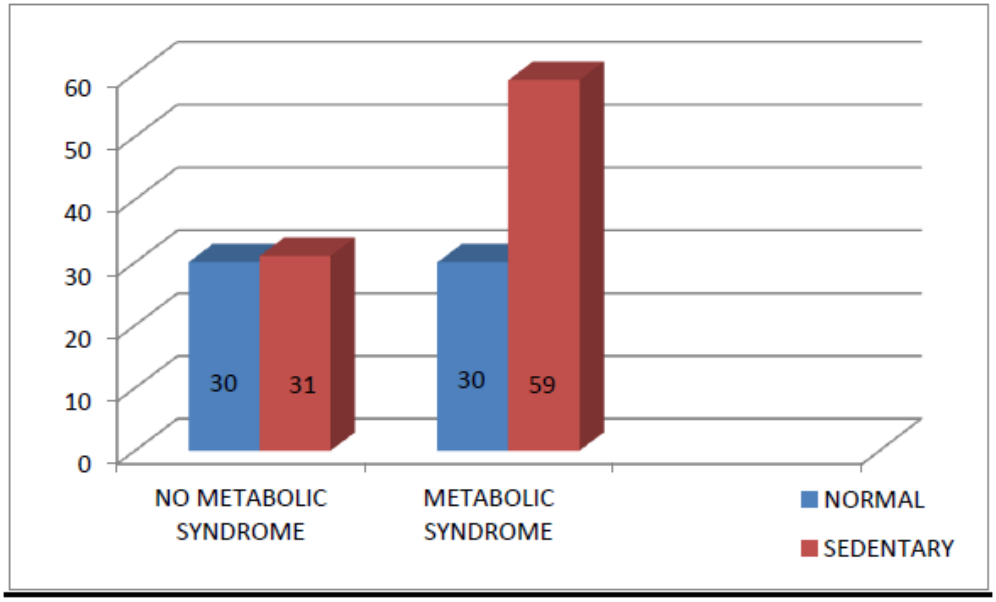


The prevalence of metabolic syndrome among age groups less than and more than 50 are studied. There are 28(56%) people with metabolic syndrome in age group less than 50 and 61 people (61%) in age group more than 50. There is no st atistical significance in the prevalence in two age groups in our study.

LIFE STYLE Vs METABOLIC SYNDROME

LIFE STYLE	NO METABOLIC SYNDROME	METABOLIC SYNDROME	TOTAL
NORMAL	30	30	60
SEDENTARY	31	59	90
	61	89	150

P VALUE=0.05

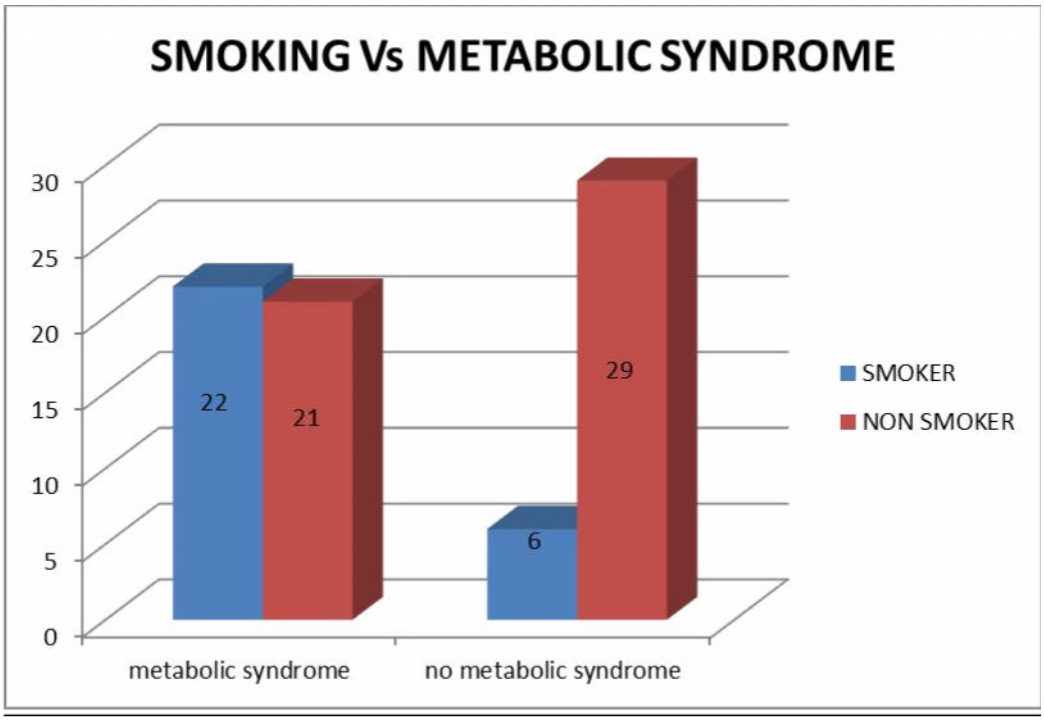


The number of patients with metabolic syndrome in sedentary group is 65.55% and non-sedentary group is 50%. Thus a high prevalence of metabolic syndrome is seen in people with sedentary lifestyle when compared to non-sedentary people. The difference is statistically significant .

SMOKING Vs METABOLIC SYNDROME

SMOKING	NO METABOLIC SYNDROME	METABOLIC SYNDROME	TOTAL
SMOKER MALE	6	22	28
NON SMOKER MALE	29	21	50
TOTAL	35	43	78

P VALUE=0.001

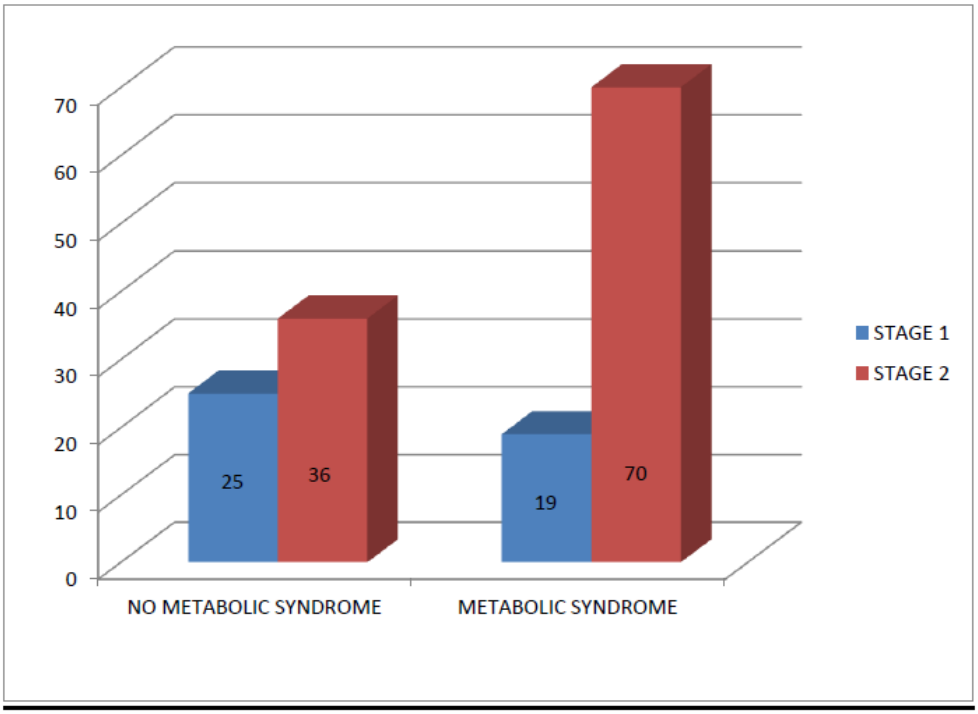


The patients with metabolic syndrome among smokers and non-smoker males are 22 (78.57%) and 21 (42%). The prevalence is high among smokers and the difference is statistically significant.

STAGE OF HYPERTENSION Vs METABOLIC SYNDROME

STAGE	NO METABOLIC SYNDROME	METABOLIC SYNDROME	TOTAL
STAGE 1	25	19	44
STAGE 2	36	70	106
TOTAL	61	89	150

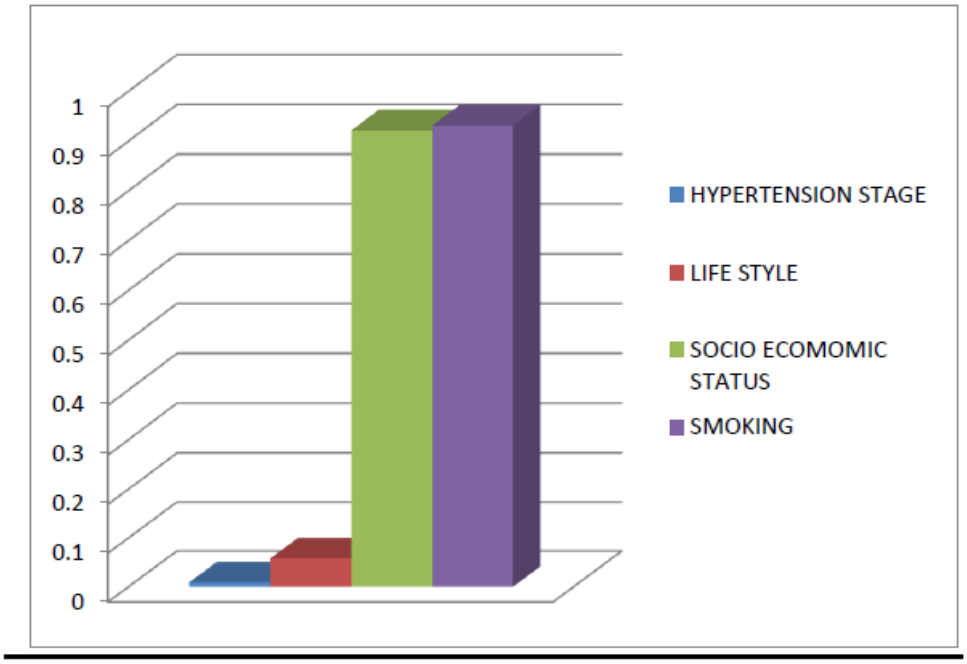
P VALUE=0.009



The prevalence of metabolic syndrome among hypertensive patients divided as stage 1 and stage 2 shows that 19 patients (43.18%) in stage 1 has metabolic syndrome and 70 (66.02%) patients in stage 2 has metabolic syndrome. The difference between the two is statistically significant.

IMPACT OF VARIOUS FACTORS ON METABOLIC SYNDROME IN OUR STUDY

FACTORS	SIGNIFICANCE (P VALUE)
HYPERTENSION STAGE	0.009
LIFE STYLE	0.058
SOCIO ECOMOMIC STATUS	0.921
SMOKING	0.931

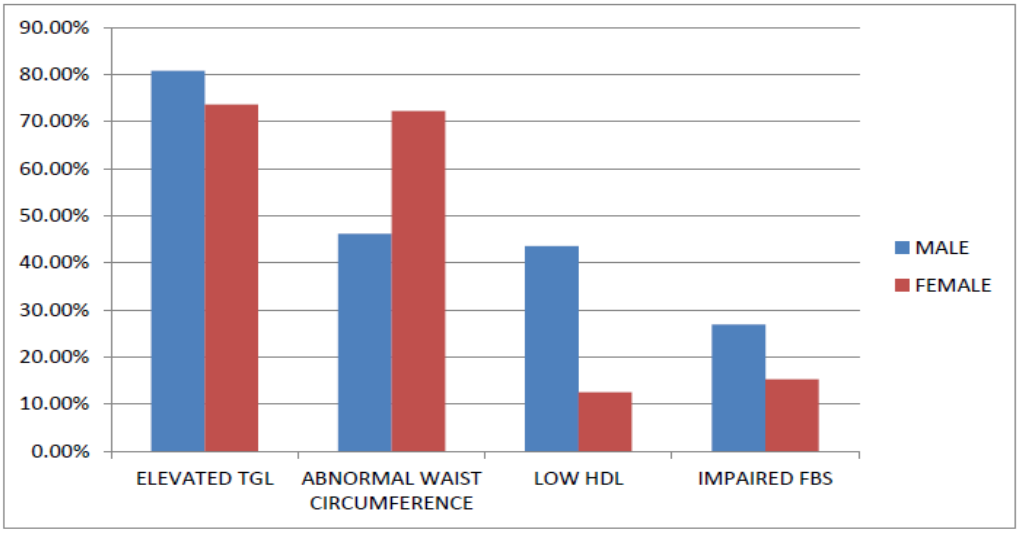


The influence of various factors on the prevalence of metabolic syndrome in our study is as follows. The stage of hypertension is the first most influential factor on prevalence of metabolic syndrome. The other factors that influence the prevalence in descending order are lifestyle, socioeconomic status and smoking.

FREQUENCY OF INDIVIDUAL COMPONENTS IN METABOLIC SYNDROME IN ALL INDIVIDUALS

COMPONENT	MALE	FEMALE
ELEVATED TGL	80.76%	73.61%
ABNORMAL WAIST CIRCUMFERENCE	46.15%	72.22%
LOW HDL	43.58%	12.5%
IMPAIRED FASTING BLOOD GLUCOSE	26.92%	15.27%

FREQUENCY OF EACH COMPONENTS IN ALL MALE AND FEMALE

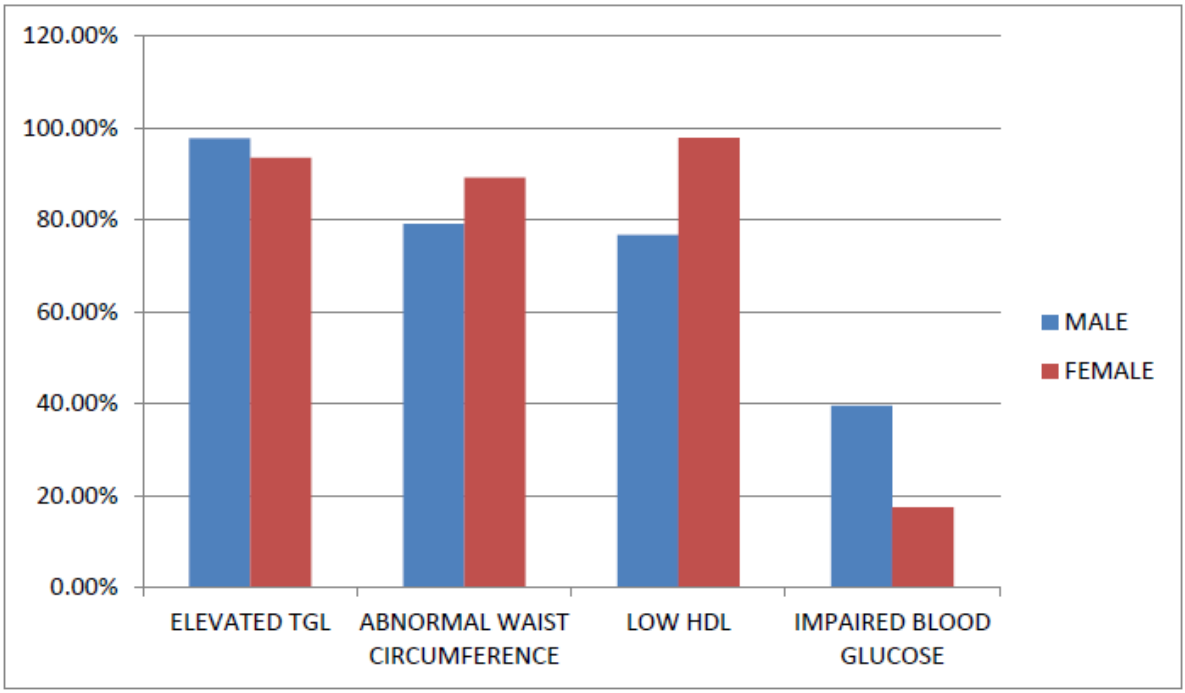


The prevalence of each component of metabolic syndrome in male is found to be elevated triglycerides (80.76%), abnormal waist circumference (46.15%) , low HDL (43.58%), impaired fasting blood glucose (26.92%) and in females the same component's prevalence are 73.61%, 72.22%, 12.5%, 15.27% respectively.

FREQUENCY OF EACH COMPONENTS IN PATIENTS WITH METABOLIC SYNDROME

COMPONENTS	MALE	FEMALE
ELEVATED TGL	97.67%	93.47%
ABNORMAL WAIST CIRCUMFERENCE	79.06%	89.13%
LOW HDL	76.74%	97.8%
IMPAIRED BLOOD GLUCOSE	39.53%	17.39%

FREQUENCY OF EACH COMPONENT IN PATIENTS WITH METABOLIC SYNDROME

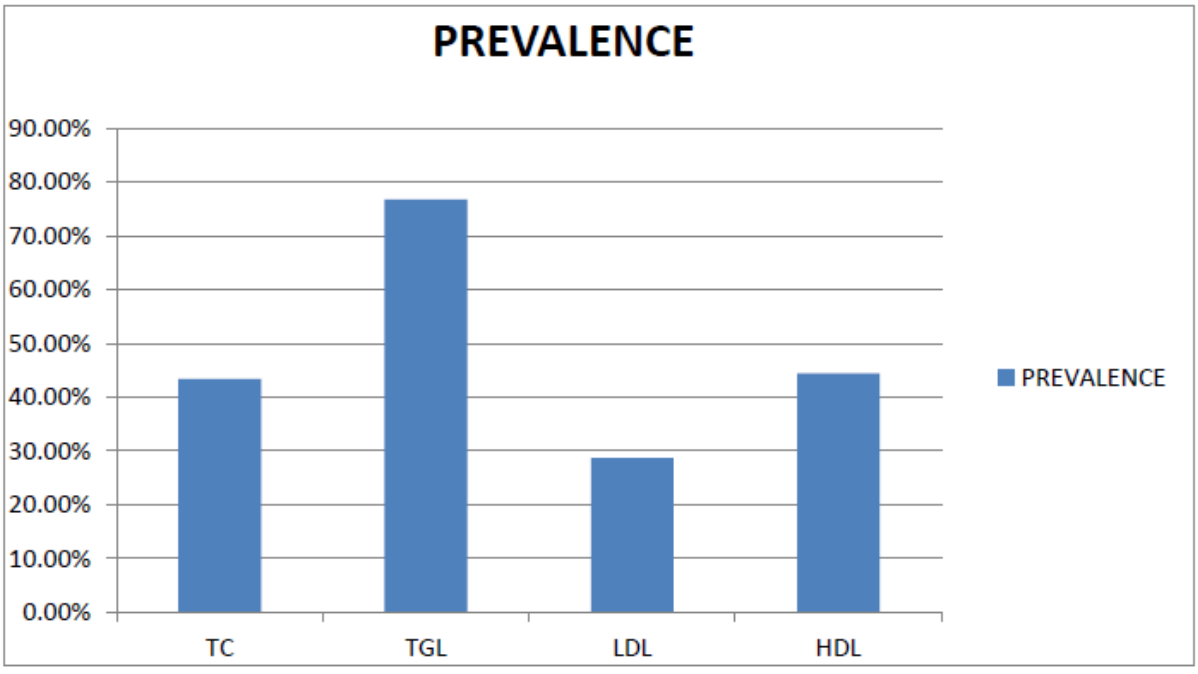


The prevalence of each component in patients with metabolic syndrome in our study shows that in male, elevated TGL (97.67%)is the most common component being

seen. It is followed by abnormal waist circumference (79.06%), low HDL (76.74%), impaired blood glucose (39.53%). Among females with metabolic syndrome the prevalence of components are in the following order-low HDL (97.8%), elevated TGL (93.47%), abnormal waist circumference (89.13%), impaired blood glucose (17.39%).

LIPID PROFILE ABNORMALITIES IN HYPERTENSIVE PATIENTS

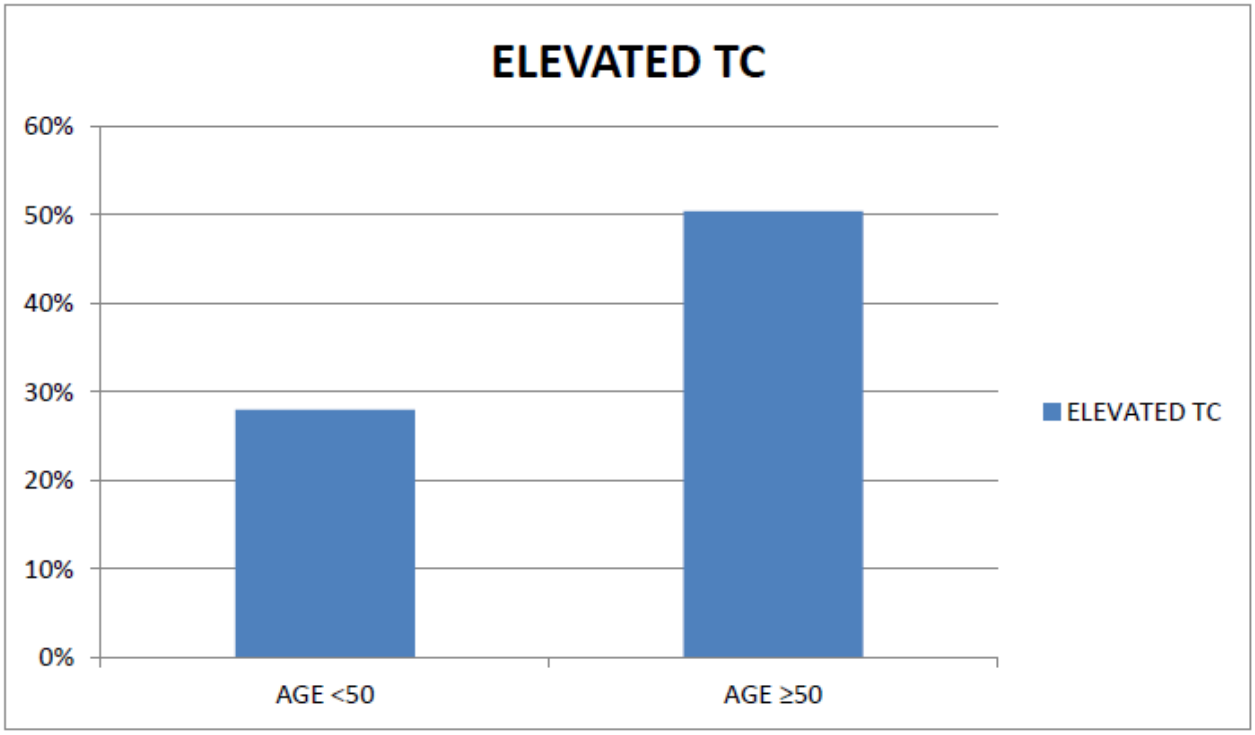
LIPID	PERCENTAGE
TC	43.33%
TGL	76.8%
LDL	28.66%
HDL	44.4%



The prevalence of lipid profile abnormalities in hypertensive patients is elevated TC 43.33%, elevated TGL 76.8%, elevated LDL 28.66% and low HDL 44.4%.

INFLUENCE OF AGE

AGE	ELEVATED TC
<50 years	28%
≥50 years	50.4%

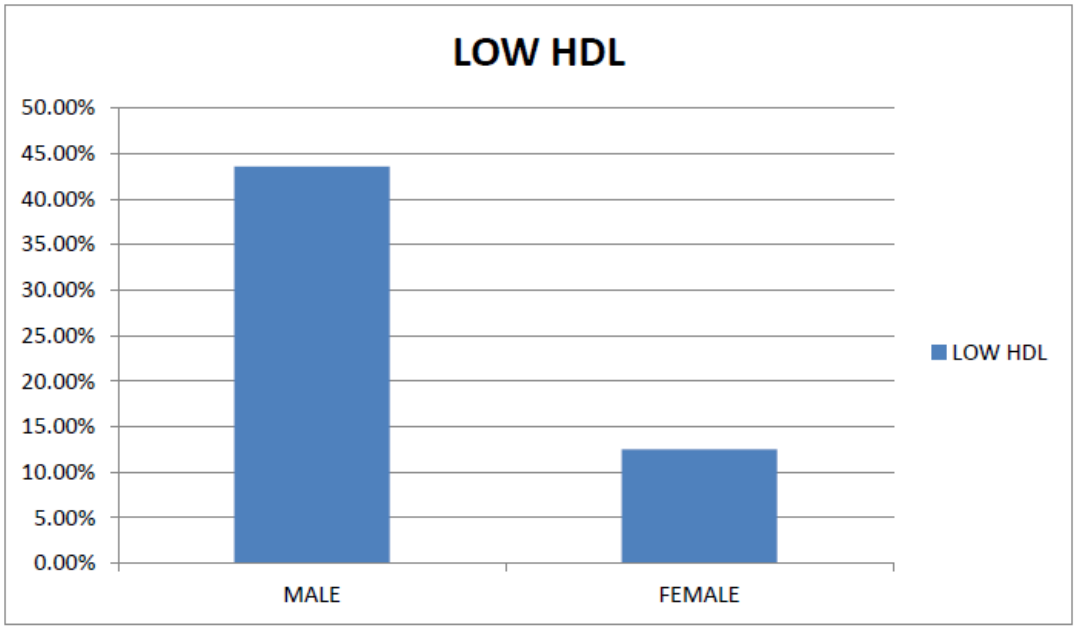


p = **0.009**

The prevalence of elevated TC in hypertensive patients belonging to age <50 years is 28% and ≥50 years is 50.4%. The difference is statistically significant.

INFLUENCE OF GENDER

GENDER	HDL
MALE	43.58%
FEMALE	12.5%

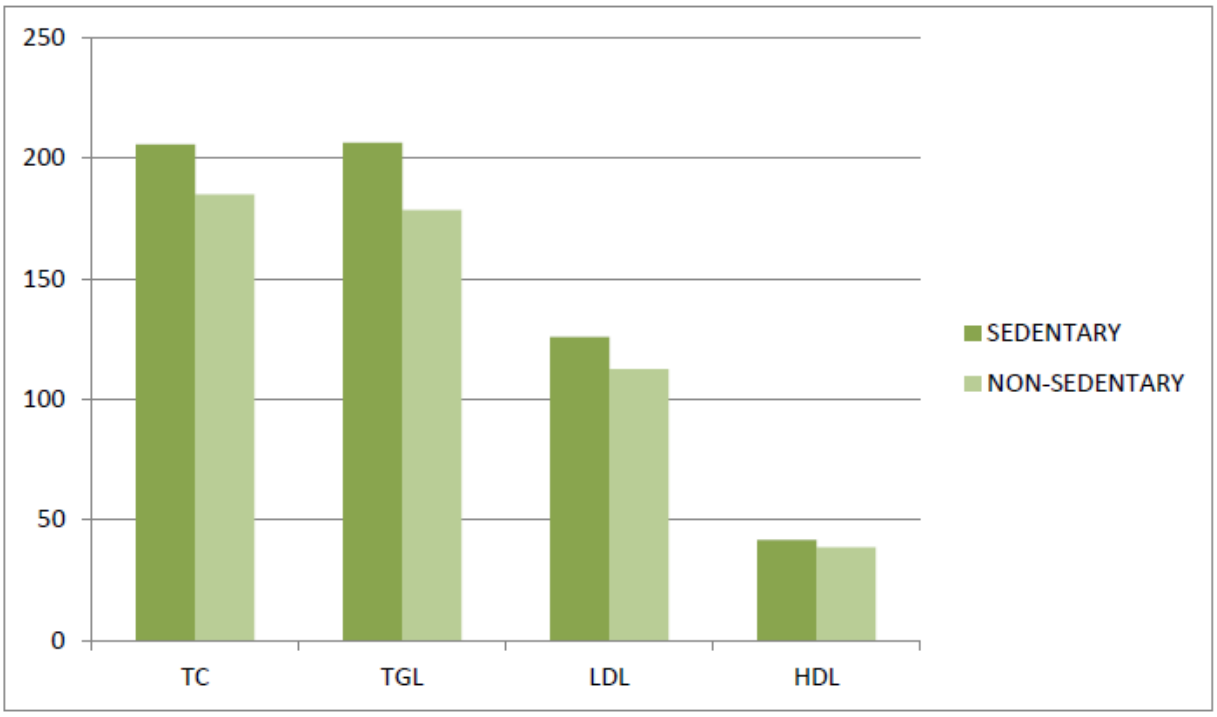


p **= 0.003**

The prevalence of low HDL among male and female is 43.58% and 12.5%. The difference between the two is statistically significant.

INFLUENCE OF LIFESTYLE

LIPID (Mean value)	SEDENTARY	NON-SEDENTARY
TC	205.77 mg/dl	185.02 mg/dl
TGL	206.34mg/dl	178.42 mg/dl
LDL	125.83mg/dl	112.57 mg/dl
HDL	41.76 mg/dl	38.58 mg/dl

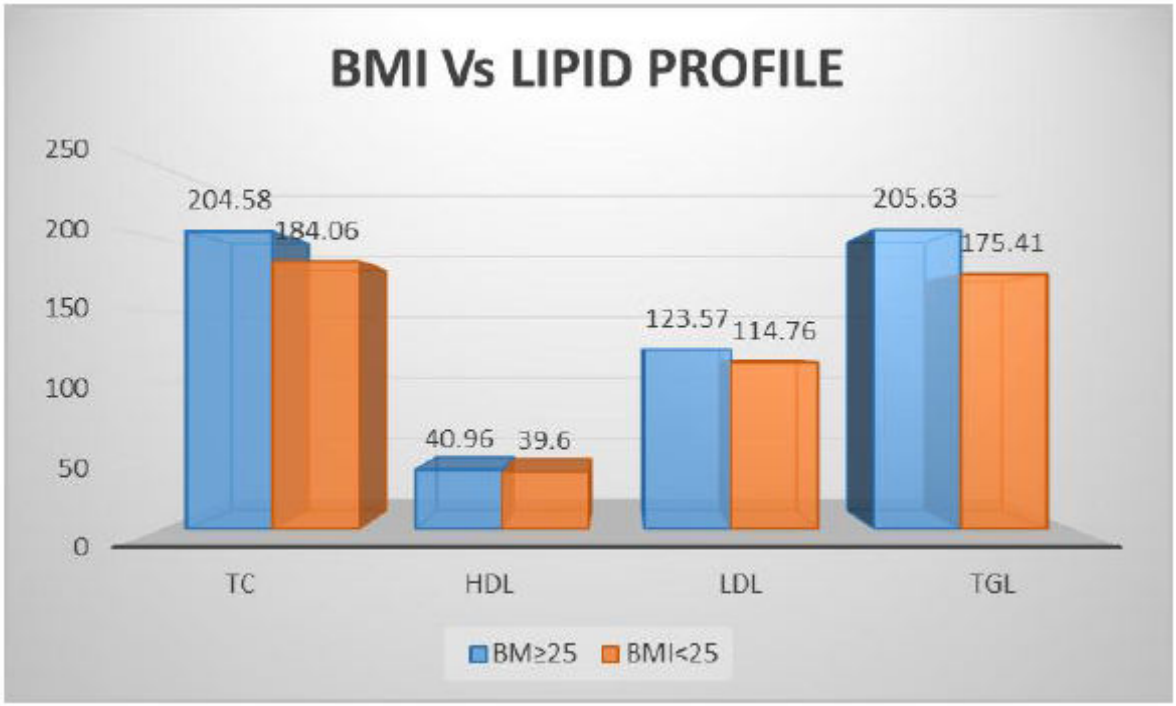


The mean values of lipid profile abnormalities are compared between sedentary and non-sedentary hypertensive people. The difference between the means TC, TGL, LDL, HDL between the two group is statistically significant with p value of 0.0001, 0.01, 0. 001,0.002 respectively.

COMPARISON OF OBESITY AND LIPID

PROFILE BMI AND LIPID PROFILE

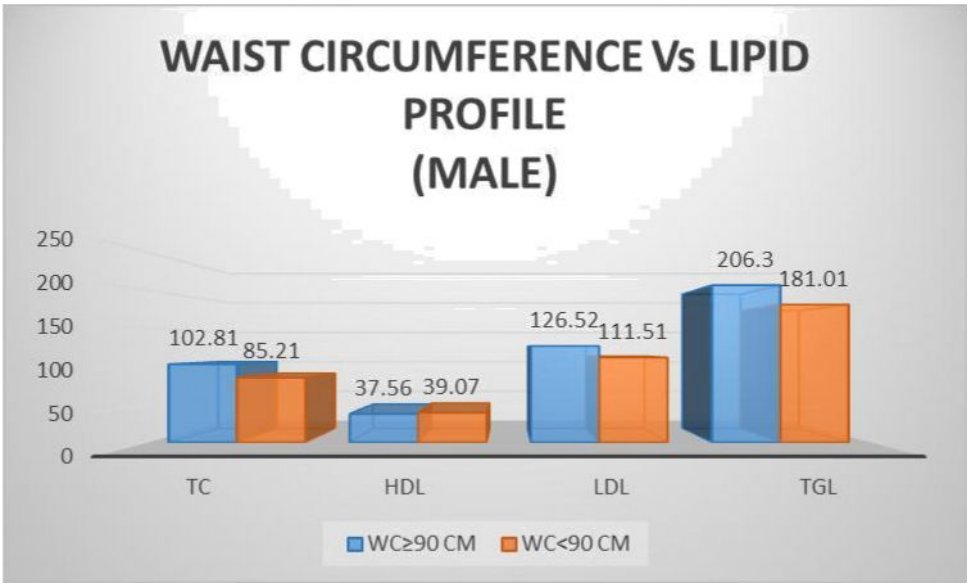
	BM≥25	BMI<25	P VALUE
TC	204.58	184.06	<0.0001
HDL	40.96	39.6	0.2503
LDL	123.57	114.76	0.0484
TGL	205.63	175.41	0.0144



The comparison of lipid profile of people with BMI <25 and ≥25 shows that there is significant difference in the values of total cholesterol, low density lipoprotein, triglycerides. The values are high in people with BMI ≥25.

WAIST CIRCUMFERENCE AND LIPID IN MALES

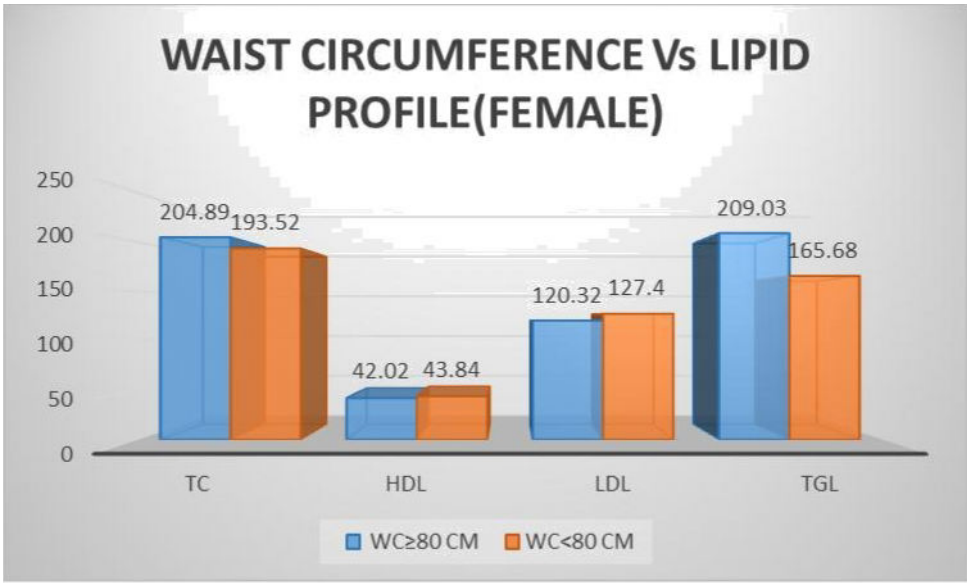
	WC≥90 CM	WC<90 CM	P VALUE
TC	102.81	85.21	<0.0001
HDL	37.56	39.07	0.2709
LDL	126.52	111.51	0.0021
TGL	206.3	181.01	0.0641



Thus on comparison of lipid profile of males with waist circumference <90 cm and ≥90 cm shows that the total cholesterol, low density lipoprotein, triglycerides are significantly high in obese people.

WAIST CIRCUMFERENCE AND LIPID IN FEMALE

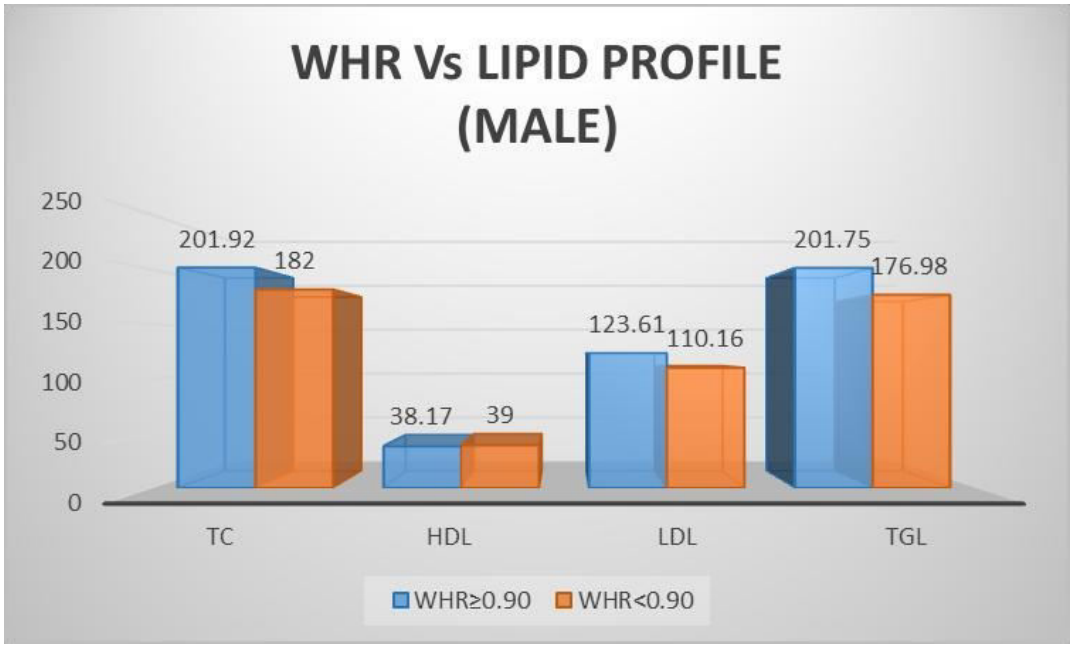
	WC≥80 CM	WC<80 CM	P VALUE
TC	204.89	193.52	0.01184
HDL	42.02	43.84	0.3054
LDL	120.32	127.4	0.3409
TGL	209.03	165.68	0.023



This shows that the female with waist circumference of ≥80 cm have significantly higher values of total cholesterol and triglycerides.

WAIST- HIP RATIO AND LIPID IN MALE

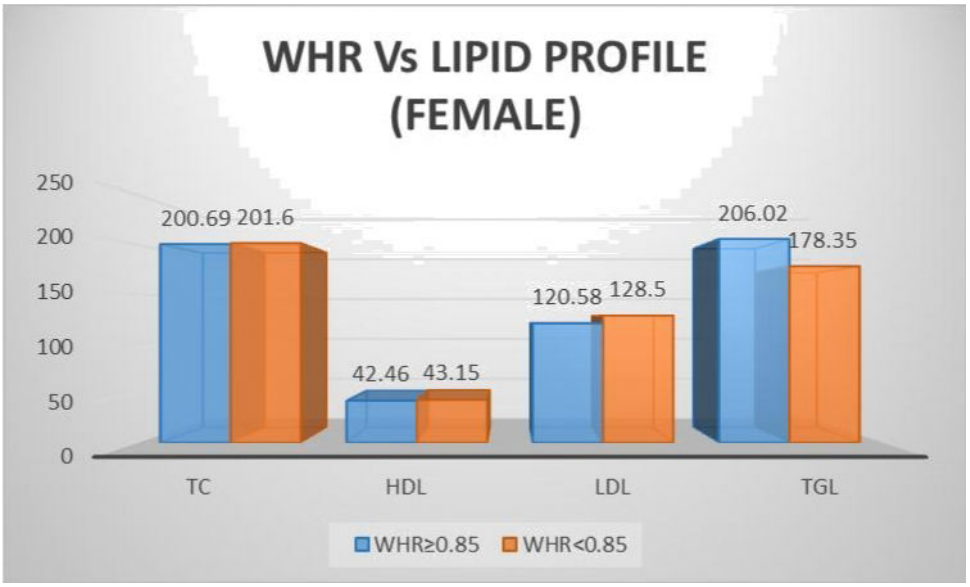
	WHR≥0.90	WHR<0.90	P VALUE
TC	201.92	182	0.0007
HDL	38.17	39	0.558
LDL	123.61	110.16	0.0077
TGL	201.75	176.98	0.0656



The lipid profile of male with waist-hip ratio of ≥0.90 that is obese group have statistically high values of total cholesterol, low density lipoprotein, triglycerides compared to male with <0.9 waist-hip ratio.

WAIST-HIP RATIO AND LIPID IN FEAMLE

	WHR≥0.85	WHR<0.85	P VALUE
TC	200.69	201.6	0.9075
HDL	42.46	43.15	0.716
LDL	120.58	128.5	0.315
TGL	206.02	178.35	0.215



The lipid profile of female with waist-hip ratio <0.85 and ≥0.85 did not show any statistical difference in the lipid values.

CONTINUOUS VARIABLES STATISTICS

S.no	FACTORS	MEAN	MEDIAN	RANGE
1	AGE	55.6866	57.5	30-75
2	HEIGHT	156.6733	156.5	138-174
3	WEIGHT	66.54	65	48-97
4	BODY MASS INDEX	27.314	26.4086	19.8-40.42
5	WAIST CIRCUMFERENCE	91.64	89	72-116
6	WAIST-HIP RATIO	0.9021	0.8916	0.76-1.078
7	SYSTOLIC BLOOD PRESSURE	163.6266	162	130-200
8	DIASTOLIC BP	95.5333	96	70-140
9	FBS	94.52	95	72-136
11	TC	197.4666	192	146-283
12	TG	195.156	180	58-495
13	LDL	120.522	117.5	64-195
14	HDL	40.486	41	24-56
15	VLDL	40.31	36.05	12-128

DISCUSSION

PREVALENCE OF METABOLIC SYNDROME

The study population of 150 newly detected hypertensive individuals, there are 78 (52%) males and 72 (48%) females. The mean age of the study population is 55.68 years. There are 29 smokers (37.179%) out of 78 males. All the females are non-smokers.

The prevalence of metabolic syndrome calculated according to NCEP ATP III criteria is 59.33% that is 89 patients out of 150 newly detected hypertensive people.

The prevalence of metabolic syndrome studied in newly detected hypertensive people conducted by Anusha Govindula, Chandrasekhar Valupadas, Sabitha Panchagiri at outpatient department of Mahatma Gandhi Memorial Hospital in Warangal, Telangana, India, among 120 hypertensive patients (75 male; 45 female) with mean age of 53.28 ± 12.98 years, according to NCEP ATP III, showed that 82.5% were falling in this criterion.

Similar studies conducted in a tertiary care hospital in the northern hilly state of Himachal Pradesh, India, by Surendhar Thakur et al[102], showed that the prevalence among 118 hypertensive patients were 68.6%.

Another cross-sectional study including 102 hypertensive outpatients at a teaching hospital in Brazil , including patients >18 years of age, showed a prevalence of 71.6%

according to NCEP ATP III criteria. The prevalence in western countries is because of increasing central obesity among people.

Studies conducted across Europe by Farsang et al reported that highest prevalence of metabolic syndrome from Central Europe and lowest from the Mediterranean. Cohort studies conducted in Spain gave a report of 52% of prevalence of metabolic syndrome.

Thus ranging from 50% to 68% the prevalence shows a wide range. Dietary factors, genetics, lifestyles have been found be the cause for these variations in the prevalence between countries.

INFLUENCE OF GENDER

The prevalence of metabolic syndrome among male were 55.13% and among female were 68.6%. The prevalence among female is higher than that of male. In a study conducted in West Ethiopia showed that the prevalence of metabolic syndrome in female according to NCEP ATP III criteria is 46.5% higher when compared to male which is 31.3%. Female dominance was also seen in study conducted by Arkhangel in Russia, Korea and China. The results are comparable to the study conducted in Pakistan and similar to Frinks cohort study. The female dominance might be due to difference in the cut-off points in metabolic syndrome criteria. An another important reason for increased prevalence in females is due to increasing central obesity in women than men in both developing and developed countries.

INFLUENCE OF AGE

The newly detected hypertensive individual in our study were divided into two groups as people < 50 years and >50 years of age. The prevalence of metabolic syndrome in these age group on analysis showed that there is no significant difference in the prevalence based on age group. Similarly study conducted by Apurva Sawant, Ranjit Mankeshwar et al[103] in Mumbai, India among 560 subjects showed there was no significant difference in the prevalence of metabolic syndrome among different age groups.

The results are similar to the study conducted in Nigeria by Adeseye A. Akintunde et al, in cardiology unit of LAUTECH teaching hospital, which showed no statistical significance in age wise distribution of metabolic syndrome.

INFLUENCE OF LIFESTYLE

The patients routine daily lifestyle was asked about and they were classified as sedentary and non-sedentary lifestyle. In our study there are 60 people with non-sedentary lifestyle and there are 90 people with sedentary lifestyle. The prevalence of metabolic syndrome among sedentary group is 65.55%. The prevalence among non-sedentary group is 50%. The difference between the two is statistically significant.

The study conducted by Antonio Cabrera de Leon, Maria del C. Rodriguez-Perez et al showed that sedentary lifestyle is associated with increased prevalence of metabolic

syndrome both by NCEP ATP III and IDF criteria. Lakka TA, Laaksonen DE et al conducted study among 1069 males showed that sedentary lifestyle has association with metabolic syndrome. The sedentary lifestyle was found to have direct relation with waist and hip circumferences, body mass index, systolic blood pressure, triglycerides, apolipoprotein B and inversely with high-density lipoprotein. So all these factors leads to direct association with metabolic syndrome.

INFLUENCE OF SOCIOECONOMIC STATUS

The prevalence of metabolic syndrome belonging to various socioeconomic class according to modified kuppuswamy scale(2007) was 43.8% among class 2&3 and 56.2% among class 4&5. This shows high prevalence among people with low socioeconomic status. Another study conducted in India also showed high prevalence among people with low per capita income.

Studies conducted by Zhan Y, Yu J, Chen R et al in china and by Matthews KA, Räikkönen K et al showed that prevalence of metabolic syndrome was high in people with low socioeconomic status. This could probably attributed to the stress, poor health conscious diet among people with low socioeconomic class.

INFLUENCE OF SMOKING

In our study the prevalence of metabolic syndrome among smoker male is 78.57% and the prevalence among non-smoker male is 42%. The difference is statistically

significant with p value of 0.0018. A study conducted by Sandra N Slagter, Jana V van Vliet-Ostaptchouk et al showed that increased metabolic syndrome in smokers independent of sex and body mass index. A similar study conducted by Sang Woo Oh, Yeong Sook Yoon et al among 24,389 men and 35,078 women showed that current smoking is associated with increased prevalence of metabolic syndrome. Relation between smoking and different components of metabolic syndrome was studied. Guang-Rong Wang1 , Li Li et al conducted a study in urban residents of China and found that the metabolic syndrome had increased prevalence among smokers.

Smokers have abnormality in lipoprotein metabolism and endothelial function. Smoking also affected the waist circumference. It is found to increase the levels of triglycerides, lower high-density lipoprotein and increase high-density lipoprotein/ apo A1 levels. It produces unfavourable changes in apo A1, apo B and lipoprotein particle size.Smoking also increases the risk of cardiovascular disease.

INFLUENCE OF STAGE OF HYPERTENSION

The hypertensive people where grouped into two groups as stage I and stage II based on JNC 7. The prevalence of metabolic syndrome in each stage has been studied. In our study there are 43.18% of people with metabolic syndrome were in stage I hypertension and 66.02% of people with metabolic syndrome were in stage II hypertension.

The prevalence of metabolic syndrome is high in people with stage II hypertension and the difference is statistically significant. Studies have shown that people with metabolic syndrome have high blood pressure. This is because of multiple risk factors being present like obesity, dyslipidemia , impaired blood glucose levels and genetic factors.

IMPACT OF VARIOUS FACTORS ON METABOLIC SYNDROME

The influence of factors like smoking, socioeconomic status, lifestyle and stage of hypertension on prevalence of metabolic syndrome is analysed by using ANOVA. The results showed that stage of hypertension has most influence, followed by lifestyle, socioeconomic status and finally smoking. The least impact by smoking could probably be explained by the predominat non-smoking female population in our study.

FREQUENCY OF ALL COMPONENTS OF METABOLIC SYNDROME IN HYPERTENSIVE PATIENTS

In our study the frequency of each component of metabolic syndrome is studied in all 150 newly detected hypertensive individuals. The analysis shows that the elevated triglycerides are the most frequent abnormality of all the other components of metabolic syndrome in both gender that is 80.76% in males and 73.61% in females. In females then it is followed by abnormal waist circumference (72.22%), impaired fasting blood glucose (15.27%), then low high density lipoprotein (12.5%). In males following elevated

triglycerides, abnormal waist circumference (46.15%), it is low high density lipoprotein (43.58%), then impaired fasting blood glucose (26.92%).

In a study conducted in Telengana by Anusha Govindula, Chandrasekhar Valupadas et al showed that elevated triglycerides to the most common abnormality among all 5 components according to NCEP ATP III criteria.It is followed by abnormal waist circumference and low high-density lipoprotein.

FREQUENCY OF VARIOUS COMPONENTS OF METABOLIC SYNDROME

The frequency of various components of metabolic syndrome according to our study is in the following order for men – elevated triglycerides (97.67%), abnormal waist circumference (79.06%),low high-density lipoprotein (76.74%) impaired fasting blood glucose (39.53%). Elevated triglycerides is the most frequent component of metabolic syndrome found among men in our study.

The frequency of various components among female is in the following order, low high-density lipoprotein (97.8%) , elevated triglycerides (93.47%), abnormal waist circumference (89.13%) and impaired fasting blood glucose (17.39%). Low high-density lipoprotein is the most frequent component among women.

Studies conducted by D.S.Prasad, Z.Kabir et al in SB Salagre, SM Itolikar et al[104] in eastern part of India showed that low high-density lipoprotein is the most frequent component among female. Among male central obesity was the most common component. The high fasting blood glucose was found to be the least frequent

component. The results of our study is similar to this study conducted in our country.

SB Salagre, SM Itolikar et al conducted study in Mumbai which showed that elevated trigycerides (35.14%)were the most frequent component found among men. Our study also shows similar report. Fasting blood glucose (35.14%) was found to be the most common component overall in the study.

Study conducted in Warangal, India showed that elevated triglycerides, waist circumference, low high-density lipoprotein, elevated fasting blood glucose were the descending order of frequency of components of metabolic syndrome.

PREVALENCE OF LIPID ANORMALITIES IN HYPERTENSIVE INDIVIDUALS

The prevalence of lipid profile abnormalities in our study is in the order of elevated triglycerides (76.8%), low high-density lipoprotein (44.4%), elevated total cholesterol (43.33%), high low-density lipoprotein (28.66%). The mean values of TC, TGL, HDL, LDL in our study are 197.4mg/dl, 195.156 mg/dl, 40.486 mg/dl, 120.522mg/dl respectively.

INFLUENCE OF AGE

In our study the patients are divided into two groups age groups as less than 50years and more than 50 years. The prevalence of elevated total cholesterol among less than 50 years is 28%. And among more than 50 years group is 50.4%. The difference is statistically significant ($p = 0.009$).

The prevalence of elevated LDL is 30.69%, low HDL is 44.55%, LDL is

30.69% among people more than 50 years. But the difference is not statistically significant.

INFLUENCE OF GENDER

The difference in prevalence of dyslipidemia is studied between male and female hypertensive patients. The prevalence of low HDL is statistically significant between male and female with a p value of 0.003.

Studies conducted in Mumbai, Telangana also showed similar results of low HDL among female.

INFLUENCE OF LIFESTYLE

The people in our study are divided into sedentary and non-sedentary. The prevalence of lipid profile abnormalities among sedentary and non-sedentary people is statistically significant by comparing the mean values. The mean values are TC (205.77mg/dl and 185.02 mg/dl), TGL (206.34mg/dl and 178.42 mg/dl), LDL (125.83mg/dl and 112.57mg/dl) and HDL (41.76 mg/dl and 38.58mg/dl). In Brazil, similar study conducted by Ignez Salas Martius et al showed results with similar lipid profile abnormalities.

INFUENCE BY OBESITY

BASED ON BMI

The comparison of lipid profile of people with BMI <25 and ≥25 shows that

there is significant difference in the values of total cholesterol, low density lipoprotein, triglycerides. The values are high in people with BMI ≥25.The results are similar to the study conducted by S .A.Desai et al[105] with lipid profile values higher in obese patients.

BASED ON WAIST CIRCUMFERENCE

On comparison of lipid profile of males with waist circumference <90 cm and ≥90 cm shows that the total cholesterol, low density lipoprotein, triglycerides are significantly high in obese people. The study also shows that the female with waist circumference of ≥80 cm have significantly higher values of total cholesterol and triglycerides.

BASED ON WAIST-HIP RATIO

The lipid profile of male with waist-hip ratio of ≥0.90 that is obese group have statistically high values of total cholesterol, low density lipoprotein, triglycerides compared to male with <0.9 waist-hip ratio. The lipid profile of female with waist-hip ratio <0.85 and ≥0.85 did not show any statistical difference in the lipid values.

By all the methods, it is shown that the obesity is related to lipid profile abnormalities.

CONCLUSION

1. The prevalence of metabolic syndrome is 59.33% and it is more in females compared to males.
2. The smoking habit, lifestyle, stage of hypertension have significant effect on prevalence of metabolic syndrome.
3. Among the components of metabolic syndrome in hypertensive males and females, elevated TGL is most common.
4. Among the components of metabolic syndrome in hypertensive males with metabolic syndrome , elevated TGL is most common.
5. Among the components of metabolic syndrome in hypertensive females with metabolic syndrome , low HDL is most common.
6. The elevated TC in hypertensive patients is significantly higher in age group ≥ 50 years .
7. The low HDL shows significant difference between male and female.
8. The dyslipidemia shows significant difference between people with sedentary and non-sedentary lifestyle.
9. There is a significant difference in the lipid profile values of obese people compared to non-obese individuals.

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METABOLIC SYNDROME IN NEWLY DETECTED HYPERTENSIVE INDIVIDUALS

PROFOMA

No: OP No:
Name: Age: Sex:
Address: Phone No:

Socio-Economic status: (by modified Kuppuswamy scale)

Class	I	II	III	IV	V

Life style: (physical activity $\leq$ 1.5 MET hour / day)

Sedentary	Non Sedentary

H/O smoking: ($\geq$ 5 cigarettes / day)

Yes	No

H/O Hypertension:

Yes	No

Anthropometry

Height (in cm)	Weight (in kg)	BMI
Waist (in cm)	Hip (in cm)	WHR

Blood Pressure

Sitting	R UL	L UL
Supine		

Investigations

Blood Sugar

Fasting -

B. Urea -

TG -

S. Creatinine -

Lipid profile

TC -

HDL -

LDL -

VLDL-

Modified (2007) Kuppuswamy socioeconomic status scale

EDUCATION

Profession / Honors	7	
Graduate / PG	6	
Post high school / diploma	5	
High school certificate	4	
Mid school certificate	3	
Primary certificate	2	
Illiterate	1	

OCCUPATION

Profession	10	
Semi – profession	6	
Clerical / shop owner/ farmer	5	
Skilled worker	4	
Semi-skilled worker	3	
Unskilled worker	2	
Unemployed	1	

Family income / month

≥ 19575	12	
9788- 19574	10	
7323 – 9787	6	
4894 – 7322	4	
2936 – 4893	3	
980 – 2935	2	
< 980	1	

Total Points	Class
26 - 29	Upper – I
16 - 25	Upper middle - II
11 - 15	Lower middle - III
5 - 10	Upper lower – IV
< 5	Lower V
Patients	points [ ] class [ ]

Physical activity assessment

Activity	MET	Duration	MET calculation
Washing / Housekeeping/ driving / cooking	1-2		
Carpentry / walking (4mph) / Dancing	3-5		
Digging in garden / Tennis games / swimming / cycling 10mph	5-7		
Tracking / jogging	7-9		
Carrying loads / walking uphill / running	>9		
Total MET			

Life Style

Sedentary	Non – sedentary

MASTER CHART

SL.NO	NAME	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG	M.S
1	ravi	50	M	5	N	N	170	60	20.761	81	88	0.9204	148	96	1	97	132	N	163	41	101	32.2	156	N
2	madasamy	56	M	5	N	Y	158	65	26.037	92	90	1.0222	166	80	2	99	158	Y	213	32	137	44.1	222	Y
3	sabukumar	51	M	5	N	N	168	56	19.841	78	84	0.9286	150	80	1	97	129	N	179	42	102	33	195	N
4	veerasamy	49	M	4	N	N	172	62	20.957	88	109	0.8991	160	100	2	81	96	N	163	44	103	35.4	177	N
5	seenu	57	M	5	N	N	160	66	25.781	87	102	0.8529	150	88	1	94	118	N	177	42	103	33	197	N
6	chandru	60	M	4	S	N	170	86	29.758	106	108	0.9815	170	88	2	94	136	N	199	38	112	49	245	Y
7	vijayan	57	M	5	N	N	170	61	21.107	88	108	0.8148	156	96	1	81	103	N	161	43	99	33.2	166	N
8	sukumar	49	M	4	N	Y	160	79	30.859	104	102	1.0196	170	86	2	107	127	Y	200	33	135	42	210	Y
9	kumaresan	51	M	5	S	N	168	80	28.345	110	102	1.0784	180	88	2	113	141	Y	201	41	126	34	170	Y
10	kirumurugu	60	M	4	N	N	171	63	21.545	82	88	0.9388	170	90	2	99	114	Y	179	42	102	49	250	N
11	murugesa	68	M	3	S	N	167	76	27.251	103	101	1.0198	164	90	2	107	154	Y	191	41	127	23	115	Y
12	kumar	68	M	4	N	N	170	63	21.799	82	88	0.9388	156	94	1	101	151	Y	185	41	124	20.2	101	N
13	ramu	67	M	3	S	Y	158	68	27.239	116	109	1.0642	162	86	2	107	136	Y	216	38	138	40	200	Y
14	sriramulu	71	M	4	N	N	164	56	20.821	88	96	0.9167	168	88	2	91	97	N	164	28	116	26.2	131	N
15	dinesan	62	M	3	N	N	161	74	28.548	109	112	0.9732	166	90	2	99	157	Y	204	38	130	36	180	Y
16	ganesh	62	M	4	N	N	174	62	20.478	92	99	0.9293	156	80	1	103	131	N	183	31	120	32	160	Y
17	suresh	63	M	3	N	N	162	63	24.005	89	97	0.9184	162	90	2	91	107	N	181	30	121	30	149	N
18	subbu	64	M	4	N	N	170	65	22.491	86	96	0.8958	150	80	1	96	128	N	160	29	100	31.2	156	Y
19	raman	62	M	3	N	N	154	64	26.986	82	88	0.9388	160	80	2	86	114	N	191	42	104	49	245	N
20	ravikumar	67	M	5	S	Y	158	65	26.037	106	104	1.0192	164	86	2	102	144	Y	204	32	131	43.2	216	Y
21	muthu	75	M	3	N	N	164	58	21.565	103	105	0.981	170	88	2	93	97	N	171	33	105	33.4	167	Y
22	sabalagan	63	M	5	S	N	160	72	28.125	112	108	1.037	180	90	2	82	107	N	173	28	99	47	235	Y
23	siva	53	M	3	N	Y	163	60	21.008	86	99	0.8687	156	80	1	86	98	N	163	40	82	57	286.4	N
24	sdhilkesan	52	M	4	S	Y	162	70	26.673	98	104	0.9423	150	86	1	103	136	Y	264	44	183	37	186	Y
25	sriram	46	M	5	S	Y	170	88	30.45	116	118	0.9831	170	90	2	96	117	N	217	40	105	72	302	Y
26	balu	47	M	3	S	N	170	61	21.107	88	96	0.9167	164	80	2	114	142	Y	207	48	103	56	148	N
27	krishnan	62	M	3	S	Y	164	78	29.001	110	118	0.9322	174	96	2	96	132	N	209	40	128	40.6	203	Y
28	kasi	72	M	4	N	Y	159	66	26.107	88	92	0.9592	158	98	1	93	103	N	216	42	142	42	210	N
29	moni	61	M	4	N	Y	158	75	30.043	98	106	0.9245	168	106	2	112	136	Y	220	40	133	47	235	Y
30	noufal	71	M	3	N	Y	164	65	24.167	89	105	0.8519	156	86	1	81	93	N	216	41	140	46.2	231	N

SL.NO	NAME	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG	M.S
31	satheesh	75	M	4	S	Y	160	85	33.203	106	111	0.955	168	110	2	91	99	N	283	41	187	57	285	Y
32	thirumal	65	M	3	S	Y	158	65	26.037	88	94	0.9423	164	106	2	94	149	Y	268	40	169	59	295	N
33	abdul	73	M	4	N	Y	163	72	27.039	113	120	0.9417	156	90	1	78	113	N	216	30	138	48	240	Y
34	avaneetha	63	M	3	S	Y	156	65	26.709	104	106	0.9811	144	100	2	88	98	N	244	36	171	37	185	Y
35	ibrahim	63	M	2	S	N	160	70	27.344	89	91	0.978	150	70	1	104	145	Y	202	47	143	12	58	N
36	arunan	41	M	2	S	N	168	70	24.802	89	89	1	150	90	1	80	106	N	206	40	131	35	175	N
37	sasikuman	42	M	2	S	N	164	80	29.744	96	102	0.9412	130	100	2	93	124	N	182	31	121	30	150	Y
38	sreenivas	31	M	2	S	Y	172	75	25.352	99	102	0.9706	150	90	1	96	112	N	176	28	99	49	246	Y
39	sreeram	53	M	2	S	N	164	72	26.77	88	99	0.9293	144	90	1	96	114	N	218	41	139	42	210	N
40	david	44	M	2	S	N	163	57	21.454	81	89	0.9101	150	100	2	111	143	Y	237	40	154	43	215	Y
41	hariharan	52	M	2	S	N	166	79	28.669	103	103	1	150	90	1	96	130	N	206	38	133	35	175	Y
42	manohara	31	M	2	N	N	170	64	22.145	86	96	0.8958	160	110	2	96	110	N	188	41	124	23	115	N
43	manikanda	36	M	3	N	Y	168	75	26.573	85	96	0.8854	160	100	2	86	117	N	188	34	119	45	225	Y
44	pgeshwar	40	M	3	N	N	167	75	26.892	102	103	0.9903	180	120	2	88	119	N	179	36	112	31	155	Y
45	nandhu	58	M	3	N	Y	154	61	25.721	89	92	0.9674	160	100	2	108	162	Y	206	48	117	40.6	203	Y
46	loganatha	65	M	3	S	N	154	64	26.986	100	112	0.8929	154	96	1	91	136	N	202	42	124	36	180	N
47	santhosh	32	M	3	S	N	171	84	28.727	96	102	0.9412	150	90	1	86	141	Y	205	33	130	42	209	Y
48	boopathy	42	M	3	S	Y	163	67	25.217	84	93	0.9032	150	110	2	120	176	Y	190	31	69	90	451	Y
49	netaji	54	M	3	S	Y	159	65	25.711	104	108	0.963	164	96	2	101	156	Y	192	43	77	72.2	361	Y
50	narendran	63	M	3	S	Y	157	65	26.37	104	109	0.9541	160	90	2	110	158	Y	258	44	159	55	275	y
51	devan	38	M	4	S	Y	166	80	29.032	106	101	1.0495	160	100	2	108	153	Y	198	45	111	42	212	y
52	gowtham	56	M	4	S	N	154	62	26.143	88	88	1	130	140	2	103	122	Y	208	48	123.4	32.6	148	N
53	parthiban	69	M	4	N	N	163	60	22.583	88	90	0.9778	176	106	2	84	128	N	173	45	103	35.4	177	N
54	princde	39	M	4	N	N	173	80	26.73	87	94	0.9314	150	80	1	96	117	N	189	38	102	49	125	N
55	dhamodara	34	M	4	N	Y	163	70	26.346	96	110	0.8727	184	96	2	99	126	N	185	38	117	39.2	196	Y
56	lokesh	46	M	5	N	N	162	72	27.435	89	94	0.9435	140	110	2	88	190	Y	152	28	98	26	130	N
57	rajesh	37	M	5	S	N	154	50	21.083	78	89	0.8764	144	96	1	94	126	N	162	29	100	33.2	166	Y
58	mohan	73	M	5	S	N	165	72	26.446	106	118	0.8983	180	86	2	101	146	Y	215	46	123	46.2	231	Y
59	karthik	72	M	4	N	N	146	60	28.148	98	100	0.98	200	100	2	123	188	Y	184	38	108	37.2	188	Y
60	thammem	58	M	4	N	N	153	58	24.777	80	92	0.8696	156	88	1	96	107	N	175	46	104	29	145	N

SL.NO	NAME	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG	M.S
61	thilagan	57	M	4	N	N	153	71	30.33	94	113	0.8319	170	100	2	72	108	N	185	38	109	38	190	Y
62	sankar	40	M	4	N	N	160	97	37.891	103	117	0.8803	150	100	2	76	110	N	189	35	120	34	170	Y
63	sreedhar	67	M	4	N	N	146	50	23.457	90	106	0.8491	190	90	2	91	105	N	180	34	108	38	190	Y
64	dharani	50	M	4	N	N	163	57	21.454	84	110	0.7636	160	100	2	96	119	N	185	41	109	38	190	N
65	lakshman	65	M	4	N	N	152	57	24.671	80	91	0.8791	158	86	1	99	117	N	180	45	104	29	145	N
66	mahesh	65	M	4	S	Y	153	58	24.777	80	90	0.8889	150	86	1	98	126	N	180	43	104	33	165	N
67	sulothunga	67	M	4	S	N	146	58	27.21	98	100	0.98	160	100	2	86	126	N	190	38	115	37.2	185	Y
68	rajendran	68	M	4	S	Y	145	83	39.477	107	120	0.8917	170	80	2	102	132	Y	207	45	126	36.4	182	Y
69	ramanadas	66	M	4	S	Y	156	60	24.655	86	100	0.86	168	106	2	98	120	N	179	29	107	43	215	Y
70	anbarasan	37	M	4	S	N	157	55	22.313	83	95	0.8737	176	90	2	88	120	N	170	33	105	32.2	161	Y
71	govindan	41	M	4	S	Y	160	56	21.875	85	105	0.8095	148	86	1	92	115	N	175	40	104	31	155	N
72	suruli	40	M	4	S	N	154	61	25.721	87	106	0.8208	148	80	1	80	117	N	175	40	104	31.4	157	N
73	pitchai	38	M	4	S	N	145	53	25.208	82	96	0.8542	158	98	1	90	120	N	183	47	101	34.1	171	N
74	amilsaraas	45	M	4	S	N	152	76	32.895	101	122	0.8279	160	98	2	98	146	Y	230	44	158	28	140	N
75	jeevan	68	M	5	S	Y	156	50	20.546	86	100	0.86	160	100	2	98	120	N	171	29	104	37.6	188	Y
76	varadhan	59	M	5	N	N	167	65	23.307	83	101	0.8218	170	98	2	96	130	N	159	34	99	25.8	129	N
77	ravindran	74	M	5	N	N	149	50	22.522	82	103	0.7961	170	100	2	96	130	N	158	24	100	34	120	N
78	dheeran	32	M	5	S	N	147	55	25.452	82	96	0.8542	160	110	2	92	117	N	187	48	105	34	170	N
79	lakshmi	48	F	5	S	N	139	64	33.125	105	120	0.875	158	98	1	106	156	Y	208	44	64	99	495	Y
80	meena	65	F	3	S	N	148	50	22.827	79	86	0.8646	180	90	2	98	120	N	174	33	109	32	149	N
81	vaneetha	62	F	3	S	N	153	65	27.767	88	95	0.9263	172	98	2	88	102	N	196	45	118	33	166	Y
82	venda	68	F	3	S	N	145	85	40.428	108	127	0.8504	150	90	1	97	105	N	205	44	127	34.4	172	Y
83	dhanam	63	F	3	N	N	150	64	28.444	88	82	1.0241	170	106	2	98	140	Y	194	44	117	33	166	N
84	meenaksh	54	F	3	S	N	138	70	36.757	106	118	0.8983	150	98	1	90	120	N	212	44	68	98.4	493	Y
85	satrala	39	F	3	S	N	152	80	34.626	100	108	0.9259	160	96	2	106	126	Y	235	45	159	31	148	N
86	usha	74	F	3	S	N	153	60	25.631	89	101	0.8812	190	80	2	90	128	N	216	42	123	51	255	Y
87	nandhini	63	F	3	S	N	151	66	28.946	98	100	0.98	160	90	2	98	107	N	206	40	130	36	180	Y
88	priya	52	F	3	S	N	150	65	28.889	96	99	0.9697	160	90	2	96	103	N	207	41	130	36	180	Y
89	gnanamal	30	F	3	S	N	153	65	27.767	75	89	0.8586	170	110	2	96	112	N	195	45	117	33	166	N
90	yogamma	55	F	3	N	N	145	85	40.428	108	127	0.8504	150	90	1	96	106	N	206	45	126	34.4	172	Y

SL.NO	NAME	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG	M.S
31	amudha	60	F	2	S	N	158	58	23.233	77	91	0.8614	170	90	2	96	130	N	150	30	94	25.8	129	N
32	palaniamm	70	F	2	S	N	150	90	40	104	120	0.8667	164	90	2	90	120	N	273	43	190	39.2	196	Y
33	umadevi	67	F	2	S	N	150	90	40	100	123	0.8293	160	80	2	106	148	Y	253	33	190	29.2	146	N
34	deivanai	71	F	2	S	N	153	66	28.194	100	101	0.9901	132	80	2	90	132	N	220	44	125	51	255	Y
35	devipriya	61	F	2	N	N	150	66	29.333	98	98	1	164	86	2	88	96	N	208	39	133	36.8	184	Y
36	loganayag	69	F	2	N	N	160	52	20.313	72	93	0.7961	170	106	2	98	140	Y	166	56	102	37.6	188	N
37	sralam	70	F	2	N	N	153	60	25.631	78	95	0.8211	190	80	2	96	114	N	218	44	123	51	255	Y
38	suba	52	F	2	S	N	150	90	40	102	123	0.8293	164	90	2	88	120	N	263	43	190	29.2	146	Y
39	velammal	55	F	2	S	N	138	70	36.757	106	124	0.8548	160	100	2	96	127	N	210	46	64	99	493	Y
100	venda	39	F	3	N	N	159	72	28.48	93	101	0.9802	170	100	2	93	132	N	156	54	96	25.8	128	N
101	seetha	54	F	3	S	N	152	80	34.626	100	120	0.8333	160	110	2	80	121	N	233	47	159	27	135	Y
102	dhanam	39	F	3	N	N	147	85	39.335	72	86	0.8542	160	110	2	92	128	N	185	50	101	34	170	N
103	nandhanam	48	F	4	N	N	150	62	27.556	89	106	0.8396	142	80	1	85	127	N	172	41	100	31	155	Y
104	rekha	45	F	4	N	N	147	55	25.452	79	91	0.8681	180	100	2	99	126	N	172	35	109	32	160	Y
105	suhasini	60	F	5	N	N	146	65	30.434	95	103	0.9223	170	110	2	112	136	N	169	29	102	38	188	Y
106	sradhamm	70	F	2	S	N	148	60	27.392	77	99	0.7982	180	100	2	92	104	N	235	53	143	39	195	N
107	annamal	60	F	5	N	N	146	65	30.434	95	103	0.9223	170	110	2	112	146	Y	169	29	102	37.6	188	Y
108	chitra	50	F	4	S	N	145	83	39.477	107	120	0.8917	164	82	2	94	142	Y	178	45	104	32	158	Y
109	devi	42	F	4	S	N	158	62	24.76	76	90	0.86	142	82	1	98	128	N	182	50	121	31	162	N
110	nandhini	40	F	4	S	N	155	54	22.57	85	97	0.8763	150	94	1	110	146	Y	204	34	142	46.2	196	Y
111	logeshwar	44	F	4	S	N	163	57	21.475	77	98	0.8056	154	100	1	93	136	Y	205	55	168	36.4	178	N
112	fathima	57	F	4	S	N	158	63	25.236	89	102	0.8725	172	92	2	98	134	N	152	32	96	27.8	131	Y
113	eshwari	55	F	4	S	N	147	54	24.99	77	89	0.8652	162	86	2	94	128	N	275	40	194	37.2	194	Y
114	malar	45	F	4	S	N	155	78	32.466	104	125	0.832	196	98	2	102	145	Y	227	42	128	55	267	Y
115	ragavi	58	F	5	S	N	158	52	20.83	88	102	0.8627	168	96	2	82	98	N	202	42	138	37.8	188	Y
116	lashmi	70	F	5	N	N	166	67	24.314	86	104	0.8269	180	126	2	94	134	N	169	29	108	33.6	192	Y
117	uma	69	F	5	N	N	147	52	24.064	85	105	0.8095	130	90	2	136	154	Y	228	34	132	56	235	Y
118	dharmama	64	F	5	S	N	145	53	25.208	75	87	0.8763	174	96	2	98	128	N	243	48	195	32.2	149	N
119	sofiya	61	F	5	S	N	141	67	33.701	107	123	0.875	154	106	2	84	127	N	224	48	74	112	393	Y
120	sudha	46	F	3	S	N	150	48	21.333	77	89	0.8788	174	120	2	89	122	N	146	46	116	28.8	148	N

SL.NO	NAME	AGE	SEX	SES	LS	SMOKING	HEIGHT	WEIGHT	BMI	WAIST	HIP	WHR	SBP	DBP	STAGE	FBS	PPBS	IFG/IGT	TC	HDL	LDL	VLDL	TG	M.S
121	menaka	57	F	4	N	N	155	73	30.385	96	115	0.9143	156	92	1	93	116	N	216	52	122	35.4	142	N
122	nalini	52	F	4	N	N	153	88	37.592	106	119	0.8908	174	110	2	82	102	N	192	32	106	36.8	194	Y
123	jayanthi	44	F	4	N	N	149	52	23.422	75	94	0.7979	154	106	2	76	108	N	182	38	126	38	182	Y
124	jeyammal	64	F	4	N	N	165	57	20.937	86	112	0.7679	186	100	2	96	109	N	188	36	104	37.5	194	Y
125	jeevamal	56	F	4	N	N	154	59	24.878	74	80	0.9333	164	108	2	86	119	N	182	34	103	36.2	148	N
126	geetha	68	F	4	S	N	158	61	24.435	72	80	0.9111	152	96	1	79	127	N	174	50	124	31.1	155	N
127	kavitha	62	F	4	S	N	148	58	26.479	92	98	0.9388	154	100	1	78	126	N	179	46	108	36	168	Y
128	pavithram	72	F	4	S	N	150	87	38.667	109	117	0.9316	160	100	2	76	126	N	196	32	125	34.2	130	Y
129	maragatham	62	F	4	S	N	160	58	22.656	74	92	0.8235	170	80	2	106	138	Y	209	55	136	38.4	145	N
130	ragavi	71	F	4	S	N	162	53	20.195	75	88	0.8673	172	110	2	92	120	N	184	52	126	39.2	204	N
131	vimala	34	F	4	S	N	162	58	22.1	87	108	0.8056	178	96	2	78	104	N	174	37	103	34.2	172	Y
132	kamalam	44	F	4	S	N	157	63	25.559	81	99	0.8349	154	92	1	82	112	N	182	43	116	33	149	N
133	jaya	42	F	4	S	N	148	56	25.566	84	99	0.8485	152	86	1	92	114	N	187	44	114	33.4	152	Y
134	sasikal	33	F	4	S	N	150	78	34.667	104	126	0.8254	148	94	1	79	108	N	193	53	122	36.1	176	Y
135	selvi	45	F	5	S	N	159	50	20.964	77	91	0.8614	166	94	2	90	152	Y	248	48	150	26	147	N
136	seetha	72	F	5	N	N	169	69	24.159	79	98	0.8	168	110	2	88	108	N	180	32	114	34.6	176	Y
137	savithri	56	F	5	N	N	151	53	23.245	79	99	0.8081	178	108	2	78	128	N	168	48	92	28.8	129	N
138	july	74	F	5	S	N	149	58	26.125	74	87	0.866	170	100	2	86	134	N	159	29	105	34	126	N
139	syeesha	34	F	5	S	N	142	69	33.228	107	123	0.8699	166	110	2	92	107	N	177	48	125	37	188	Y
140	malar	47	F	3	S	N	154	54	22.769	86	100	0.86	152	100	1	126	166	Y	216	48	88	128	386	Y
141	vani	68	F	3	S	N	156	68	27.942	77	88	0.8878	176	98	2	92	114	N	184	38	127	34.6	149	N
142	kamala	59	F	3	S	N	149	89	40.088	112	130	0.8615	178	120	2	98	116	N	192	49	129	38	184	Y
143	latha	72	F	3	N	N	152	63	27.268	76	74	1.0238	154	100	1	77	105	N	227	36	142	35.4	146	N
144	viji	61	F	3	S	N	141	68	34.204	107	121	0.8843	170	110	2	98	158	Y	218	44	128	36	182	Y
145	shanthi	58	F	3	S	N	147	76	35.171	98	106	0.9245	160	90	1	82	132	N	234	44	54	88	476	Y
146	jayanthi	39	F	3	S	N	155	65	27.055	92	102	0.902	164	100	2	118	136	Y	257	45	170	33	162	Y
147	vijaya	73	F	3	S	N	149	63	28.346	100	104	0.9615	180	90	2	90	128	N	229	42	146	38	272	Y
148	janaki	61	F	3	S	N	165	72	26.446	93	103	0.9029	160	100	2	83	120	N	223	47	156	34	194	Y
149	dhanam	57	F	3	S	N	157	68	27.587	77	92	0.8725	168	90	2	76	114	N	195	51	148	32	192	N
150	saradha	32	F	3	N	N	151	87	38.156	110	125	0.88	170	100	2	93	116	N	182	45	104	31	156	Y

KEY TO MASTER CHART

SL.NO – Serial number

SES – Socio Economic Status

LS- Life Style

BMI – Body Mass Index

WHR – Waist Hip Ratio

SBP – Systolic Blood Pressure

DBP – Diastolic Blood Pressure

FBS – Fasting Blood Sugar

TC – Total Cholesterol

HDL – High Density Lipoprotein

LDL – Low Density Lipoprotein

VLDL – Very Low Density Lipoprotein

TG - Triglyceride

MS – Metabolic Syndrome

TeúLtTYoLÞdÏ Bn®u ®YWm

Bn®u úSôdLm

1) ×§RôL LiP±VlThP EVo CWjR AÝjR R² SToL°p Cp Y[o£ûR Uôt\ úSôndÏ± úSônjRôdLm T¥dL,
2) EVo CWjR AÝjR R²SToLs ùLôÝl©p ÑV®YW ùRôPoTô] úLô[ôßLÞm úSônjRôdLØm Øû\T¥dL,
3) EVo CWjR AÝjR R²SToLs ùLôÝl©p ÑV®YWj§p. úSôn UdLh§Ws Utßm NêL LôW¦Ls T¥dL,

Bn®p TeúLtTRtLô] RÏ§Ls :

31 – 75 YVÕ ùLôiP ×§RôL LiÓ©¥dLlThP EVo CWjR AÝjR R²SToLs, ùNnØû\ ®[dLm

CkR Bn®p TeúLtTYoLÞdÏ ØÝ EPp T¬úNôRû] ùNnÕ úSô«u A±Ï±Ls LiP±VlTÓm,

EPp T¬UôQeLs Utßm CWjR AÝjR A[ÅÓLs GÓdLlTÓ¡u\],

CWjR NodLûW Utßm ùLôÝl×Lô] CWjR T¬úNôRû] ùNnVlTÓm,

EPt T¬úNôRû]«]ôp GkR TdL ®û[ÜLÞm úSWôÕ, CWjRl T¬úNôRû]«]ôp £ß Y- Utßm CWjRdL£Ü úS¬PXôm, CR]ôp Gq®R Tô§l×m HtTPôÕ,

Hu TeúLtL úYiÓm

Y[o£ûR Uôt\ úSôndÏ± Es[Rô GuTûR LiP±YRtLôL,

BWônf£ ¨ûXVm

ùTôÕ UÚjÕY Õû\. AWÑ úYío UÚjÕY Lpí¬ Utßm UÚjÕYUû]. úYío,

சுய ஒப்புதல் படிவம்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொண்டேன். நான் ஆய்வில் இருந்து விலகி கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்தி கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் ..... இடம் .....

நாள் .....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம் ..... இடம் .....

நாள் ..... ஆய்வாளரின் பெயர் .....