"WAIST CIRCUMFERENCE TO HEIGHT RATIO AS A SCREENING TOOL IN THE ASSESSMENT OF METABOLIC SYNDROME AND ITS COMPARISON WITH BODY MASS INDEX" - A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL, BELAGAVI"

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ENDORSEMENT

This is to certify that the dissertation entitled **"WAIST CIRCUMFERENCE TO HEIGHT RATIO AS A SCREENING TOOL IN THE ASSESSMENT OF METABOLIC SYNDROME AND ITS COMPARISON WITH BODY MASS INDEX" - A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL, BELAGAVI"** is a bonafide research work done by **REG NO. BG0115013.**

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ABBREVIATIONS USED

%	–	Percentage
WHR	–	Waist Circumference to Height Ratio
WC	–	Waist Circumference
BMI	–	body Mass Index
Mg	–	Milligrams
µg	–	Micrograms
kg	–	Kilogram
cm	–	centimeter
m	–	meter
WHO	–	World Health Organisation
IFG	–	Impaired Fasting Glucose
IGT	–	Impaired Glucose tolerance
g	–	Grams
mmol	–	Millimol
HDL	–	High Density Lipoprotein
LDL	–	Low Density Lipoprotein
TG	–	Triglycerides
IDF	–	International Diabetic Federation
CVD	–	Cardiovascular Disease
T2DM	–	Type 2 Diabetes Mellitus
CHD	–	Coronary Heart Disease
NHANES	–	National Health And Nutrition Examination Survey
FFA	–	Free fatty Acids
TNF α	–	Tumor Necrosis Factor α

IL-6	–	Interleukin 6
CRP	–	C reactive protein
PAI	–	Plasminogen activator Inhibitor
MetS	–	Metabolic Syndrome
tPA	–	Tissue Plasminogen activator
RAS	–	Renin Angiotensin System
CETP	–	Cholesterol Ester Transport Protein
VLDL	–	Very Low Density Lipoprotein

ABSTRACT

Background and Objectives

Metabolic syndrome has become increasingly prevalent globally over the last two decades. Body Mass Index (BMI) has been used as a proxy for obesity and as a screening tool to assess Metabolic syndrome for many years, but, in recent years, indices of abdominal obesity (first waist-hip ratio WHpR) and then waist circumference (WC) and the novel waist circumference to height ratio (WHR) have increasingly been associated with higher cardio metabolic risk in both cross-sectional and prospective studies. The disadvantage of BMI is that it can't account for the weight that comes from muscle instead of fat. Athletes and naturally muscular people will not get accurate BMI scores, nor will apparently slender individuals who carry excess body fat around their waists. BMI will often be misleading because of differences in body type and muscle mass. WHR provides a simple, inexpensive, non-invasive, and gender independent method for screening of cardiovascular disease risk factors.

Methodology

This one year study was done from January 2016 to December 2016 in the Department of Medicine of a tertiary care centre in North Karnataka. Prior to the commencement, ethical clearance was obtained. A total of 150 consecutive patients presenting with Metabolic Syndrome were studied. Anthropometric indices like Height, Weight and Waist Circumference were measured. Patients were subjected to investigations like Blood sugar, HbA1c and lipid profile.

Results

Majority of the patients were males (67%) and the commonest age group was 51 to 70 years (51%). The average weight was found to be 67kgs. The average BMI was 24.9kg/m^2. About 48% patients of Metabolic Syndrome had a BMI ≥ 23, which is the cut off value for Indian population. We observed that majority of patients' waist Circumference to Height Ratio was above the cut off value of ≥ 0.5. The detection rate of Waist Height Ratio for Metabolic syndrome was found to be 95%. Thus WHR proved to be superior anthropometric parameter as compared to BMI(p value – 0.0001). Our further observations revealed that the individual components of Metabolic Syndrome like Diabetes, Hypertension, Dyslipidemia did not reveal any significant association with BMI or Waist Height Ratio.

Conclusion and interpretation

WHR is a better anthropometric tool in the assessment of metabolic syndrome as compared to BMI

Keywords

Metabolic syndrome, Body mass Index, Waist to Height Ratio

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INTRODUCTION

Metabolic syndrome is a major and escalating public health problem. It consists of a variety of risk factors with insulin resistance and adiposity as its key features. It is a preemptive diagnosis and indicative of a person who is likely to suffer from lifestyle diseases in the future.

Metabolic syndrome is defined by a constellation of interconnected physiological, biochemical, clinical, and metabolic factors that directly increases the risk of cardiovascular disease, type 2 diabetes mellitus, and all cause mortality. Insulin resistance, visceral adiposity, atherogenic dyslipidemia, endothelial dysfunction, genetic susceptibility, elevated blood pressure, hypercoagulable state, and chronic stress are the several factors which constitute the syndrome[140]

The syndrome identifies a subgroup of patients with shared pathophysiology who are at high risk of developing cardiovascular disease and type 2 diabetes. It confers a 5-fold increase in the risk of type 2 diabetes mellitus (T2DM) and 2-fold risk of developing cardiovascular disease (CVD) over the next 5 to 10 years. Further, patients with the Metabolic syndrome are at 2- to 4-fold increased risk of stroke, a 3- to 4-fold increased risk of myocardial infarction (MI), and 2-fold the risk of dying from such an event compared with those without the syndrome regardless of a previous history of cardiovascular events [12]

Metabolic syndrome is becoming increasingly prevalent among Asian population. In most countries nearly 1/5th of the adult population or more were affected by Metabolic syndrome with a secular increase in prevalence[129].The Asia-Pacific region typically includes much o f Southeast Asia, and Oceania[130]. Countries

in the region have a wide diversity in socio-cultural background and are at different levels of economic and technological development. Increasing economic development in many of the lower to middle-income countries of the region has been a major contributor to the increasing prevalence of obesity, type-2 diabetes and cardiovascular disease[131-133]. Hence, it is likely that the prevalence of Metabolic syndrome has also increased in the region in recent years.

In view of the increasing prevalence globally, early identification of the high risk individuals is critical in reducing the morbidity and mortality of Metabolic syndrome. Since obesity is one of the key components, various anthropometric parameters have been evaluated to screen high risk individuals. Also, the identification of metabolic syndrome will attract attention to various other related conditions such as fatty liver, polycystic ovary syndrome, and obstructive sleep apnea.

Since the first official definition of the metabolic syndrome put forward by a working group of the World Health Organization (WHO)[134] in 1998, a number of different definitions have been proposed. The latest definition given by NCEP ATP III takes into account the evidence that abdominal obesity is an important component of the metabolic syndrome and proposes gender- and race-specific cut-offs for waist circumference (WC). Although the need for different WC is attributed to ethnic variation, it was observed that even within the same population, people with identical WC but different heights have different risks for metabolic syndrome. Several studies from Asia indicate that waist-to-height ratio (WHtR) is more strongly associated with CVD risk factors than other anthropometric measures such as WC, body mass index (BMI), and waist hip ratio (WHR)[135-138].

The epidemic proportion of Metabolic Syndrome in the world today and its subsequent downstream impact on the cardiovascular system, renal system, cerebrovascular system, immune system, and on cancer diagnoses collectively herald a catastrophic impact on the world population with anticipated tens of millions of avoidable deaths[139]. Considering the health and economic factors projected to occur due to the effects of this syndrome, the forecast is dismal and bodes poor for individual nations and for humanity as a whole. Unless concerted efforts with clear and concentrated action plans are carried out by the world community to address this silent and little noted epidemic, the cost in lives lost may be in excess of those caused by natural disasters, man-made disasters, accidental deaths and even major conflicts and wars. The ever-growing dimension of this health problem justifies a global "call-to-arms" that should be addressed by international bodies

OBJECTIVES

The objectives of the present study are

1. To assess the overall power and precision of the anthropometric index i.e, WHR (Waist to height ratio) in assessment metabolic syndrome
2. To compare WHR with the conventionally used anthropometric parameter BMI(Body Mass Index)

REVIEW OF LITERATURE

Definition and terminology

Metabolic syndrome is a cluster of biological factors characterized by abdominal obesity, dyslipidemia, hypertension, and type 2 Diabetes Mellitus[30]. The link between metabolic syndrome and increased risk of multiple chronic diseases (eg, cardiovascular disease, arthritis, chronic kidney disease, schizophrenia, several types of cancer) and of early death have been reported for many decades[31-42].

Several expert groups have therefore attempted to produce diagnostic criteria. However the current approved criteria employed the National Cholesterol Education Programme (NCEP) Adult Treatment Panel (ATP) III criteria tabulated as below:

	Men	Women
Central obesity	Waist > 40 inches	Waist > 35 inches
Fasting triglycerides	> 150 mg/dL	> 150 mg/dL
Low HDL-C	< 40 mg/dL	< 50 mg/dL
Blood pressure	> 130/85	> 130/85
Fasting glucose	> 110 mg/dL	> 110 mg/dL

The numerous terminologies given to Metabolic syndrome are: the plurimetabolic syndrome, the X syndrome, the X plus syndrome, the X Metabolic Syndrome, the cardiovascular metabolic syndrome, the insulin-resistance - dislipidemia syndrome, the atherogenic metabolic syndrome, the syndrome of atherogenic factors' agglomeration, the deadly quartet).

Historical perspectives

As early as 250 years ago, long before the Metabolic Syndrome description, the Italian physician and anatomist, Morgagni identified the association between visceral obesity, HTA (arterial hypertension), atherosclerosis, the high levels of uric acid in the blood and the frequent respiratory disorders during sleep (the obstructive apnea)[3,2]

In 1920, Nicolae Paulescu, speaking about obesity and diabetes, said "most frequently, the obese people become glycosuric, as if the two affections (obesity and fat diabetes) represent two consequent phases of the same pathological process"[4,].

At the middle of the 20th century (1947), Vague, a French physician, was the first to identify android obesity (adiposity of the superior part of the body) as being the condition the most frequently associated with Diabetes Mellitus and Cardiovascular Diseases[2].

In the 70's, Moga, Orha, Haragus[5,6] supported the idea of the existence of a close connection among the components that constitute the metabolic syndrome at present, correlating them to the cardiovascular diseases.

Towards the end of the 80's, the assembly of glucose, insulin metabolism disorders, obesity, dyslipidemia and arterial hypertension received the mysterious name of "X syndrome"

In 1988, Reaven G., an endocrinologist and physician from Stanford University, was the one who took a big stride forwards, interpreting the association of diabetes, obesity, dyslipidemia and arterial hypertension by their pathogenic relationship with the peripheral insulin-resistance. He named this association "X

syndrome", the name underlining the doubtfulness that accompanied the emitting of the apparently new concept[7]. The insulin resistance and the compensatory hyperinsulinism were associated with each component of the metabolic syndrome, offering thus a physio-pathological connection between them. Continuing this logical chain, one can naturally reach the conclusion that the metabolic syndrome represents a complex disturbance of the energetic metabolism, in close connection with the insulin secretion altering, influenced in its turn by the sensitivity / resistance to insulin [8].

Ferranini and collab. resumed this idea, confirming that this assemblage of disturbances is provoked by the insulin–resistance and, after several years, they named it the "insulin –resistance syndrome"[3].

Zimmet and Serjentson[8] talk about the "plus X syndrome" signalling the association with hyperuricaemia, sedentariness and old age. The X syndrome generates high degrees of free radicals, which are harmful to the cell, causing premature aging.

In 1998, the first definition of the metabolic syndrome was formulated by a group of researchers from the WHO (World Health Organization), the group being concerned with studying diabetes. It made precise the fact that the syndrome is defined by the presence of type 2 diabetes mellitus or the altered tolerance to glucose combined with at least 2 other factors (hypertension, increased level of blood lipids, obesity and microalbuminuria).

The definition of the Metabolic Syndrome according to WHO[9]

- "Diabetes mellitus/ IFG* / IGT** / insulin resistance (evaluated by the euglycemic clamp method***) and at least 2 of the following parameters:
- BMI>30 kg/m2 or the waist/hip ratio > 0.90 for men, >0.85 for women
- Plasmatic triglycerides (TG) ≥ 150 mg/dl(>1.7 mmol/l) or HDL-cholesterol < 35 mg/dl (<0.9 mmol/l) in men, < 39 mg/dl (< 1.0 mmol/l) in women
- The rate of excretion of the urine albumin >20 µg/min or albumin/creatinine ratio ≥30 mg/g
- Blood pressure ≥ 140/90 mmHg."

In 1999 EGIR (European Group for the Study of Insulin resistance) proposed a change in the WHO definition, establishing that insulin resistance is the principal cause of this syndrome[10]. EGIR attached bigger importance to the abdominal obesity than WHO, but excluded the patients with diabetes mellitus type 2.

EGIR definition[11]

"Insulin-resistance or hyperinsulinemia >25% and, at least, 2 of the following parameters:

- Plasmatic glucose≥ 6.1 mmol(excluding diabetes)
- Blood pressure≥ 140/90 mmHg or treatment for Hypertension
- Plasmatic triglycerides ≥ 2 mmol/l or HDL cholesterol < 1 mmol/l or treatment for dyslipidemia
- Waist circumference ≥ 94 cm for men and ≥80 for women.

In 2001 NCEP-ATP III (the USA Cholesterol Education Panel, Adult Treatment Panel III) introduced alternative clinical criteria for defining Metabolic

Syndrome. The ATP III criteria don't require the demonstration of the insulin-resistance presence[12]"

The definition of the Metabolic Syndrome according to NCEPATP III (the USA Cholesterol Education Panel, Adult Treatment Panel III)[13]

"At least 3 of the following parameters:

- Waist circumference > 102 cm for men, >88 cm for women
- Plasmatic triglycerides ≥ 150 mg/dl (>1.7mmol/l)
- HDL cholesterol < 40 mg/dl (1.0 mmol/l) in men, < 50 mg/dl (1.3 mmol/l) in women
- Blood pressure ≥ 130/85 mmHg
- Serum glucose ≥ 110 mg/dl (> 6.1 mmol/l)"

Other definitions of the metabolic syndrome were suggested, complicating the possibility of an accepted international definition.

AACE definition (American College of Endocrinology)[14]

"The presence of at least 1 factor out of the following:

- Diagnosis of cardiovascular diseases, hypertension, polycystic ovary syndrome, nonalcoholic fat liver or acanthosis nigricans disease
- Family history of type 2 diabetes mellitus, hypertension or cardiovascular diseases
- Gestational diabetes history or intolerance to glucose
- Non-Caucasian ethnic
- Sedentariness

- BMI > 25 kg/m2 and/or waist circumference> 102 cm for men and > 88 cm for women
- Age > 40 years.

And at least 2 out of the following parameters:

- Plasmatic triglycerides (TG) ≥ 150 mg/dl
- HDL cholesterol < 40 mg/dl in men, < 50 mg/dl in women
- Blood pressure ≥ 130/85 mmHg
- Fasting glucose 110 – 125 mg/dl or at 2h postprandial 140 – 200 mg/dl (diabetes is excluded from the AACE definition)."

The IDF definition[15]

"The central obesity (defined by the waist circumference ≥ 94 cm for men and ≥ 80 cm for women, of European origin, with characteristic values for various ethnic groups) and ≥ 2 of the following parameters:

1. Low level of the TG ≥ 1.7 mmol/l(150 mg/dl) or drug treatment for hyperlipidemia
2. Low level of the HDL – cholesterol< 1.03 mmol/l (40 mg/dl) in men and < 1.29 mmol/l (50 mg/dl) in women or drug treatment for dyslipidemia
3. Arterial hypertension, systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥85 mmHg or cure for hypertension that was previously diagnosed.
4. The increased levels of the venous glycemia ≥ 5.6 mmol/l (200 mg/dl) or previously diagnosed type 2 DM (with values > 5.6 mmol/l or 200 mg/dl, there is recommended an oral test of tolerance to glucose, but it isn't needed for defining the Metabolic Syndrome presence)."

The National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) definition for metabolic syndrome was updated by the American Heart Association and the National Heart Lung and Blood Institute in 2005. According to the revised definition, metabolic syndrome is present if three or more of the following five criteria are met:

- "Waist circumference above 40 inches (men) or 35 inches (women)
- Blood pressure over 130/85 mm of hg
- Fasting triglyceride (TG) level over 150 mg/dl
- Fasting high-density lipoprotein (HDL) cholesterol level less than 40 mg/dl (men) or 50 mg/dl (women)
- Fasting blood sugar over 100 mg/dl."

The important definitions are summarised in the table below

Clinical Measure	WHO (1998)	EGIR	ATP III (2001)	AACE (2003)	IDF (2005)
Insulin resistance	IGT, IFG, DM-2, or lowered insulin sensitivity* plus any 2 of the following	Plasma insulin _75th percentile plus any 2 of the following	None, but any 3 of the following 5 features	IGT or IFG plus any of the following based on clinical judgment	None
Body weight	Men: waist-to-hip ratio ≥0.90; women: waist-to-hip ratio≥0.85 and/or BMI ≥30 kg/m2	WC≥94 cm in men or ≥80 cm in women	Waist circumference ≥102 cm in men or ≥88 cm in women	BMI ≥25 kg/m2	Increased WC (population specific) plus any 2 of the following
Lipid	TG >150 mg/dL and/ or HDL-C<35 mg/dL in men or <39 mg/Dl in women	TG ≥150 mg/dL and/ or HDL-C<35 mg/dL in men or <39mg/dl in women	TG≥150 mg/dL HDL-C _40 mg/dL in men or <50 in women	TG ≥150 mg/dL and HDL-C <40 mg/dL in men or<50 mg/	Triglycerides ≥150 mg/dL or on TG Rx HDL-C<40 mg/dL in men or <50 mg/dL in women or on HDL-C Rx
Blood pressure	≥140/90 mm Hg	≥140/90 mm Hg or on hypertension Rx	≥130/85 mm Hg	≥130/85 mm Hg	≥130 mm Hg systolic or 85 mm Hg diastolic or on hypertension Rx
Glucose	IGT, IFG, or T2DM	IGT or IFG (but not diabetes)	≥110 mg/dL (includes diabetes)	IGT or IFG (but not diabetes)	≥100 mg/dL (includes diabetes)
Other	Microalbuminura			Other features of insulin resistance	

BMI, body mass index

There are several limitations for the present definition. The criteria are ambiguous or incomplete. The insulin – resistance as a unique aetiology is unsure. Further studies are needed in modifying the actual Metabolic Syndrome definition, with adding the risk parameters for the cardiovascular disease to optimize its predictive value. Identifying a cluster of cardiovascular disease risk factors that confer a higher risk when analysed together proves an unrealistic purpose at present[16,17].

From an anthropological point of view, the metabolic syndrome can only be defined by anthropometry, as the populational/racial anthropological studies are, for the time being, at an incipient stage. Paraphrasing the eminent scientist Jean Rostand, one may say that, the more various the aggressions that the human body has to endure are, the more various the measures taken for protecting it should be[2].

Epidemiology

The prevalence of obesity among US adults increased steadily since the 1990s and is now at epidemic proportions, with over two-thirds of US adults either overweight or obese[18]. Concurrently, the prevalence of type 2 diabetes and hypertension has also steadily increased, cumulating in substantial increases in the proportion of adults who likely meet the criteria for metabolic syndrome and are thus at increased risk for more serious chronic conditions and premature death. It is therefore urgent to understand the trends in metabolic syndrome prevalence with the goal of identifying etiologic factors that are subject to public health intervention strategies.

The NHANES study is one of the largest studies conducted to determine the prevalence of Metabolic syndrome. The study estimated the prevalence of metabolic

syndrome and individual components over time (1988–1994, 1999–2006, and 2007–2012), stratified by race and sex using weighted means and proportions. The overall prevalence of metabolic syndrome in 1988–1994 was 25.3%, declining to 25.0% in 1999–2006 and then increasing substantially to 34.2% in 2007–2012[19]

Similar trend is seen in India and other South Asian countries. The prevalence of obesity and metabolic syndrome is rapidly increasing in India and other South Asian countries, leading to increased mortality and morbidity due to CVD and T2DM[20,21]. Approximately about one third of urban South Asians have evidence of the metabolic syndrome[22].

The main drivers are related to rapid nutritional changes, lifestyle and socioeconomic transitions, consequent to increasing affluence, urbanization, mechanization, and rural-to-urban migration[22,24]. Data also indicate that atherogenic dyslipidemia, glucose intolerance, thrombotic tendency, subclinical inflammation, and endothelial dysfunction are proportionately higher in Asian Indians than Caucasians[20,23]. Many of such manifestations are more severe and are seen at an early age in Asian Indians than Caucasians.[20,24] Metabolic syndrome and cardiovascular risk in Asian Indians/South Asians are also heightened by their relative increase in body fat mass, truncal subcutaneous fat mass, intra-abdominal fat mass, and also in ectopic fat deposition. Cardiovascular risk cluster also manifests at a lower level of adiposity and abdominal obesity[20,21]

Asian Indians have an increased prevalence of coronary heart disease (CHD) and T2DM amongst all ethnic groups[20,25]. This Asian Indian or South Asian Paradox refers to the fact that high prevalence of diabetes is seen in people originating from South Asian nations of Bangladesh, India, Nepal, Pakistan, and Sri Lanka, despite

lower rates of obesity (as defined by conventional body-mass-index criteria)[27,28.] South Asians also seem to have a peculiar body phenotype known as South Asian Phenotype, characterized by increased waist circumference, increased waist hip ratio, excessive body fat mass, increased plasma insulin levels and insulin resistance, as well as an atherogenic dyslipidemia, with low levels of HDL cholesterol and increased triglyceride levels[27,28]. All such factors predispose South Asians not only to T2DM but also to premature CHD. In addition, unique genetic markers could potentially make South Asians more susceptible to cardiometabolic risk[20,24,26]

The global and Indian trends of prevalence of metabolic syndrome are tabulated as below:

Age-specific and age-standardized prevalence of metabolic syndrome subjects in South Asians

Age (in years)	Total study subjects No			Metabolic syndrome subjects No (%)		
	Males	Females	Total	Males	Females	Total
20–29	68	81	149	2 (2.9)	8 (9.9)	10 (6.7)
30–39	108	145	253	24 (22.2)	50 (34.5)	74 (29.3)
40–49	145	153	298	44 (30.3)	87 (56.9)	131 (43.9)
50–59	137	118	255	64 (46.7)	87 (73.7)	151 (59.2)
60–69	87	67	154	46 (52.9)	55 (82.1)	101 (65.6)
70–79	38	21	59	20 (52.6)	18 (85.7)	38 (64.4)
80+	7	3	10	2 (28.6)	2 (66.7)	4 (40.0)
Total	590	588	1178	202 (24.9)	307 (42.3)	509 (33.5)

Prevalence and Odds Ratios for Metabolic Syndrome in US Adults Stratified by Race and Sex, National Health and Nutrition Examination Survey (NHANES), 1988–2012

Characteristic	NHANES Period		
	1988–1994	**1999–2006**	**2007-2012**
Metabolic syndrome, % (SE)	25.29 (0.85)	24.99 (0.55)	34.17 (0.74)
Elevated waist circumference	31.12 (0.60)	47.98 (0.79)	51.92 (0.91)
Elevated triglycerides	26.52 (0.82)	24.99 (0.55)	28.77 (0.73)
Reduced HDL cholesterol	32.53 (1.09)	25.13 (0.65)	44.03 (0.94)
Elevated blood pressure	33.92 (0.83)	40.62 (0.65)	42.72 (0.89)
Elevated fasting glucose	28.49 (1.05)	19.65 (0.63)	26.07 (0.64)
Race–male sex, adjusted OR (95% CI)			
Non-Hispanic white	1 [Reference]		
Non-Hispanic black	0.55 (0.46–0.67)	0.64 (0.53–0.76)	0.77 (0.66–0.89)
Mexican American	1.10 (0.87–1.40)	0.82 (0.68–0.99)	1.04 (0.89–1.23)
Race–female sex, adjusted OR (95% CI)			
Non-Hispanic white	1 [Reference]		
Non-Hispanic black	1.12 (0.96–1.31)	1.18 (0.99–1.39)	1.20 (1.02–1.40)
Mexican American	1.65 (1.36–2.00)	1.30 (1.05–1.60)	1.20 (0.98–1.46)

Metabolic syndrome can be used as a simple tool for monitoring the future societal risk for diabetes and CVD based on risk factors that can easily be monitored.

This type of monitoring at regional or country level may guide health authorities in prioritising and targeting their preventive efforts.

At the individual level, presence or absence of the metabolic syndrome appears to create a tool for guiding the clinician and the patient with respect to the risk of developing diabetes, hypertension, CVD.

Pathophysiology

Metabolic syndrome is not a specific disease. It is a constellation of metabolic derangements such as insulin resistance, hyperinsulinemia, abdominal obesity, impaired glucose tolerance, dyslipidaemia, hypertension, and a proinflammatory and prothrombotic state[29]. Metabolic syndrome is due to a complex interplay between genetic and environmental factors. The pathophysiology of individual components is discussed below:

1. Insulin Resistance and Glucose Intolerance:

Insulin is an antiatherogenic hormone and this metabolic effect involves the activation of phosphatidylinosital (PI) 3-kinase. In case of Insulin resistance, PI 3- kinase pathway is impaired and Insulin is no longer antiatherogenic[43]. Obesity in particular abdominal adiposity is one of the main reasons for Insulin resistance. Non-esterified fatty acids (NEFA) are released from excess adipose tissues, which increase insulin resistance. In case of Insulin resistance there is increased lipolysis from the adipose tissue which increases the free fatty acids, further inhibiting the anti-lipolytic effect of Insulin[44]. Visceral or omental fat appears to be the most detrimental and contributes most to the development of lipotoxicity in peripheral tissues by the secretion of adipocytokines[45]. Metabolic Syndrome is associated with a high amount of intra-abdominal fat, low adiponectin levels, and elevated levels of cytokines (interleukin 1RA and interleukin 1beta)[46]. Hyperinsulinemia may increase the

production of very low-density lipoprotein, triglycerides and thus raise triglycerides. Insulin resistance can raise blood pressure[47].

Additional contributors to insulin resistance include abnormalities in insulin secretion and insulin receptor signaling, impaired glucose disposal, and proinflammatory cytokines. The relation of impaired glucose tolerance and Insulin resistance is well documented. To compensate for defects in insulin activity, insulin secretion or clearance needs to be modified to sustain normal glucose levels. Hyperglycemia is the end result if these mechanisMetabolic Syndrome fail[44.] Since insulin resistance increases a person's risk for developing cardiovascular disease and Type 2 diabetes, several researchers have proposed measures of insulin resistance in obese individuals with and without Metabolic Syndrome. Some believe that insulin assays or alternative biomarkers of insulin resistance may facilitate cardiovascular risk prediction in individuals with Metabolic Syndrome[48].

2. Obesity:

The “obesity epidemic” is principally driven by an increased consumption of cheap, calorie-dense food and reduced physical activity. Adipose tissue is a heterogeneous mix of adipocytes, stromal preadipocytes, immune cells, and endothelium, and it can respond rapidly and dynamically to alterations in nutrient excess through adipocytes hypertrophy and hyperplasia[49]. With obesity and progressive adipocytes enlargement, the blood supply to adipocytes may be reduced with consequent hypoxia[50]. Hypoxia has been proposed to be an inciting etiology of necrosis and macrophage infiltration into adipose tissue that leads to an overproduction of biologically active

metabolites known as adipocytokines which includes glycerol, free fatty acids (FFA), proinflammatory mediators (tumor necrosis factor alpha (TNFα) and interleukin-6 (IL-6)), plasminogen activator inhibitor-1 (PAI-1), and C-reactive protein (CRP)[51]. This results in a localized inflammation in adipose tissue that propagates an overall systemic inflammation associated with the development of obesity related comorbidities[52]. Adipocytokines integrate the endocrine, autocrine, and paracrine signals to mediate the multiple processes including insulin sensitivity[53], oxidant stress[54], energy metabolism, blood coagulation, and inflammatory responses[55] which are thought to accelerate atherosclerosis, plaque rupture, and atherothrombosis. This shows that the adipose tissue is not only specialized in the storage and mobilization of lipids but it is also a remarkable endocrine organ releasing the numerous cytokines.

2.1. FFA

Upper body subcutaneous adipocytes generate a majority of circulating FFA while an intra-abdominal fat content has been positively correlated with the splanchnic FFA levels which may contribute to the liver fat accumulation commonly found in abdominal obesity[56]. Further, an acute exposure of skeletal muscle to the elevated levels of FFA induces insulin resistance by inhibiting the insulin-mediated glucose uptake, while, a chronic exposure of the pancreas to the elevated FFA impairs a pancreatic β-cell function[57]. FFAs increase fibrinogen and PAI-1 production[58].

2.2 TNF α

It is a paracrine mediator in adipocytes and appears to act locally to reduce the insulin sensitivity of adipocytes[51]. Evidence suggests that TNF-*α* induces adipocytes apoptosis[59] and promotes insulin resistance by the inhibition of the insulin receptor substrate 1 signalling pathway[58]. The paracrine action would further tend to exacerbate the FFA release, inducing an atherogenic dyslipidemia[61]. Plasma TNF*α* is positively associated with the body weight, WC, and triglycerides (TGs), while, a negative association exists between the plasma TNF*α* and High density lipoprotein–cholesterol (HDL-C)[59].

2.3. CRP

Elevated levels of CRP are associated with an increased WC[62], insulin resistance[63], BMI[64], and hyperglycemia[62] and are increased with the number of the Metabolic Syndrome components. It is more likely to be elevated in obese insulin-resistant, but, not in obese insulin-sensitive subjects[65]. In addition, it has been demonstrated that regardless of the presence or degree of the Metabolic Syndrome in an individual, CRP levels independently predicted the occurrence of future CVD events[66]. Because the Metabolic Syndrome has been linked with a greater chance of future CVD events[67], CRP levels may be an important independent predictor of unfavorable outcomes in the Metabolic Syndrome.

2.4. IL-6

It is released by both adipose tissue and skeletal muscle in humans[68]. It has both an inflammatory and an anti-inflammatory action. IL-6 receptor is also

expressed in the several regions of the brain, such as the hypothalamus, in which it controls an appetite and energy intake[69]. It is a systemic adipokine, which not only impairs insulin sensitivity but is also has a major determinant of the hepatic production of CRP[70]. IL-6 is capable of suppressing lipoprotein lipase activity. It has been shown to be positively associated with BMI, fasting insulin, and the development of T2DM[71] and negatively associated with HDL-C[72].

2.6. PAI-1

A serine protease inhibitor is secreted from intra-abdominal adipocytes, platelets, and the vascular endothelium[51]. It exerts its effects by inhibiting the tissue plasminogen activator (tPA)[73] and thus is considered as a marker of an impaired fibrinolysis and atherothrombosis. Plasma PAI-1 levels are increased in abdominally obese subjects[74] and inflammatory states[75], thus, increasing the risk of an intravascular thrombus and adverse cardiovascular outcomes[76].

2.7. Adiponectin

It regulates the lipid and glucose metabolism, increases insulin sensitivity, regulates food intake and body weight, and protects against a chronic inflammation[77]. It inhibits hepatic gluconeogenic enzymes and the rate of an endogenous glucose production in the liver. It increases glucose transport in muscles and enhances fatty acid oxidation. It has a multifactorial antiatherogenic action which includes an inhibition of endothelial activation, a reduced conversion of macrophages to foam cells, and inhibition of the smooth muscle proliferation and arterial remodeling that characterizes the

development of the mature atherosclerotic plaque[78]. Adiponectin is inversely associated with CVD risk factors such as blood pressure, low density lipoprotein cholesterol (LDL-C), and TGs[79]. Moreover, studies have shown Adiponectin to be a strong inverse independent risk factor for CVD[80]. Further, Fumeron et al. concluded that hypoadiponectinemia is associated with insulin resistance, hyperinsulinemia, and the possibility of developing T2DM, independent of fat mass[61]. The anti-inflammatory molecule, adiponectin, is negatively associated with the body weight, WC, TGs, fasting insulin, insulin resistance (HOMA-Homeostasis Model Assessment)[59], BMI, and blood pressure, whereas a positive association exists between adiponectin and HDL-C[59,82]. Its expressions and secretions are reduced by TNFα[83], possibly through a stimulated production of IL-6, which also inhibits adiponectin secretion[84]. Adiponectin is seen to be "protective," not only in its inverse relationship with the features of Metabolic Syndrome [85] but also through its antagonism of TNFα action[86].

2.8. Leptin

It is an adipokine involved in the regulation of satiety and energy intake[51]. Levels of leptin in the plasma increase during the development of obesity and decline during the weight loss. Leptin receptors are located mostly in the hypothalamus and the brain stem and signals through these receptors controls satiety, energy expenditure, and neuroendocrine function. Most overweight and obese individuals have an elevated level of leptin that do not suppress appetite, or in other words, leptin resistance. Leptin resistance is thought to be a fundamental pathology in obesity[87]. Besides its effect on appetite and

metabolism, leptin acts in the hypothalamus to increase the blood pressure through activation of the sympathetic nervous system (SNS)[88]. High circulating levels of leptin are reported to explain much of the increase in the renal sympathetic tone observed in obese human subjects[89]. Leptin-induced increase in renal sympathetic activity and blood pressure is mediated by the ventromedial and dorsomedial hypothalamus[90]. Leptin is a nitric oxide (NO) dependent vasodilator but also increases the peripheral vascular resistance and the sympathetic nerve activity[91]. The concentration of plasma leptin is correlated with adiposity, and hyperleptinemia is indeed considered an independent cardiovascular disease risk factor[92].

3. Hypertension

One of the key symptoms of Metabolic Syndrome is hypertension. It is a salient symptom which may remain undetected for long. It is an important risk factor for development of cardiovascular disease. All the hemodynamic and metabolic disorders of essential hypertension and insulin resistance are closely related. Essential hypertension is frequently associated with several metabolic abnormalities, of which obesity, glucose intolerance and dyslipidemia are the most common[93]. Obesity may be the strongest risk factor for uncontrolled hypertension. Studies have shown that obesity provides a connection between hypertension, insulin resistance and dyslipidemia[94]. In another study three factors were found in the clustering of metabolic variables. These three factors were insulin resistance, hypertension and dyslipidemia. Both general and central obesity was associated with insulin resistance and hypertension and only weakly linked to dyslipidemia[95]. The results of Farmingham Heart Study

estimate the risk of excess weight was the cause of hypertension in 78% of men and 65% of women [96]. Studies also suggest that both hyperglycemia and insulin activate the RAS (Renin-Angiotensin System) by increasing the expression of angiotensinogen AII, and the AT1 receptor, which, in concert, may contribute to the development of hypertension in patients with insulin resistance[97]. There is cross talk between the RAS and insulin signalling at multiple levels, and the RAS appears to be important in atherogenesis. Activation of RAS may inhibit the action of Insulin via the PI3 pathway[98]. There is also evidence which supports a strong relation between hypertension and obesity, which may involve insulin and leptin as well as sympathetic nervous system. Leptin and insulin are considered to be compensatory mechanisms. They are required to restore energy balance with sympathetic nervous system as one of the effective mediator[99].

4. ***Proinflammatory state***

Low-grade inflammation is associated with insulin resistance and endothelial dysfunction and adipose tissue generates inflammatory cytokines that may link insulin resistance with vascular disease[100]. The origin of the inflammatory state and of endothelial dysfunction is adipocyte-generated inflammatory cytokines, which correlate strongly with insulin resistance. Circulating signal molecules from fat could include FFAs, adiponectin, IL-6 (particularly at the liver, where IL-6 increases CRP production), resistin, leptin, and TNF-α. This study has sought associations of levels of C-reactive protein and interleukin-6 with measures of obesity and of chronic infection as their putative determinants. The study also related levels of C-reactive protein and

interleukin-6 to markers of the insulin resistance syndrome and of endothelial dysfunction. Metabolic syndrome and obesity are a kind of stress that leads to activation of inflammatory pathways. The causation of inflammation is multifactorial. The inflammation in metabolic syndrome is not accompanied by infection, autoimmunity or massive tissue injury. In fact the inflammation is low grade chronic inflammation. Researchers have attempted to name this inflammatory state as ―metaflammation‖, meaning metabolically triggered inflammation. A few studies have confirmed the positive association between obesity indices and inflammatory markers, mainly CRP (C - reactive protein) in women[101], but also other inflammatory markers, both in women and men[102].

Increased concentrations of inflammatory mediators, such as, C-reactive protein, tumor necrosis factor-alpha, interleukin-6 and others have been found in the obese. Adipose tissue has been found to express most of these inflammatory markers. Obesity was the most important feature associated with C-reactive protein[103].

5. Prothrombotic state:

It is characterized by increased plasma plasminogen activator inhibitor (PAI)-1 and fibrinogen and also associates with the metabolic syndrome. Fibrinogen, an acute-phase reactant like CRP, rises in response to a high-cytokine state. Thus, prothrombotic and proinflammatory states may be metabolically interconnected[17]

PAI-1 is an important risk factor for metabolic syndrome. Three other biomarkers, CRP, IL6, and fibrinogen also associate importantly with the Metabolic Syndrome cluster.

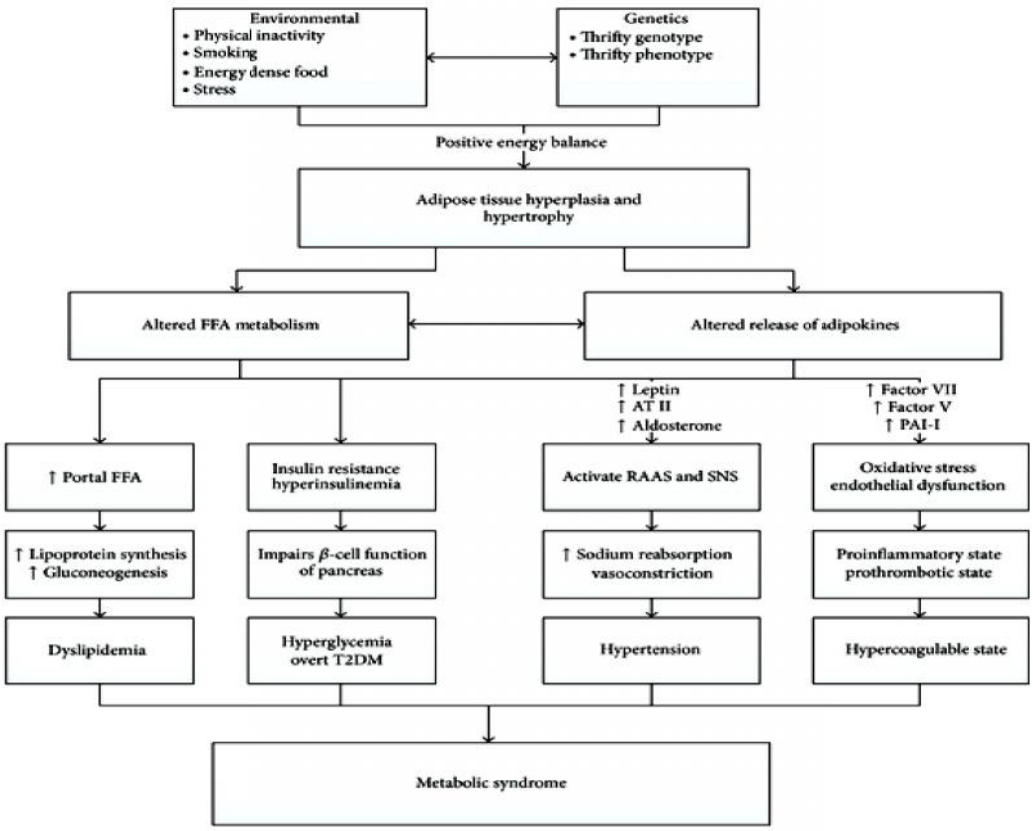
6. Atherogenic dyslipidemia

The key features of atherogenic dyslipidemia are high plasma TG levels, low HDL cholesterol levels and an increase in small dense LDL. Insulin resistance and visceral obesity are associated with atherogenic dyslipidemia[104].

Insulin resistance leads to atherogenic dyslipidemia in several ways. First, insulin normally suppresses lipolysis in adipocytes, so impaired insulin signaling increases lipolysis, resulting in increased FFA levels. In the liver, FFAs serve as a substrate for synthesis of TGs. FFAs also stabilize the production of apoB, the major lipoprotein of very-low-density lipoprotein (VLDL) particles, resulting in more VLDL production. Second, insulin normally degrades apoB through PI3K-dependent pathways, so insulin resistance directly increases VLDL production. Third, insulin regulates the activity of lipoprotein lipase, the rate-limiting and major mediator of VLDL clearance.

Thus, hypertriglyceridemia in insulin resistance is the result of both an increase in VLDL production and a decrease in VLDL clearance. VLDL is metabolized to remnant lipoproteins and small dense LDL, both of which can promote atheroma formation. The TGs in VLDL are transferred to HDL by the cholesterol ester transport protein (CETP) in exchange for cholesteryl esters, resulting in TG-enriched HDL and cholesteryl ester-enriched VLDL particles.

The TG-enriched HDL is a better substrate for hepatic lipase, so it is cleared rapidly from the circulation, leaving fewer HDL particles to participate in reverse cholesterol transport from the vasculature.



Schematic presentation of Metabolic Syndrome.

(FFA: free fatty acid, ATII: angiotensin II, PAI-1: plasminogen activator inhibitor-1, RAAS: renin angiotensin aldosterone system, SNS: sympathetic nervous system.)

Anthropometric indices:

Obesity is one of the cardinal features of metabolic syndrome. General and central obesity are associated with CVD risk[105-112].Various Anthropometric indices are suggested for obesity. These include BMI, Waist Circumference, Waist to Hip Ratio, Waist circumference to height ratio.

BMI(Body Mass Index):

Body mass index (BMI) is a measure of weight adjusted for height, calculated as weight in kilograms. Metabolic Syndrome divided by the square of height in meters (kg/m2). Although BMI is often considered an indicator of body fatness, it is a surrogate measure of body fat because it measures excess weight rather than excess fat.

BMI is a simple, inexpensive, and non-invasive surrogate measure of body fat. Studies have shown that BMI is correlated to more direct measures of body fat, such as underwater weighing and dual energy x-ray absorptiometry.

Despite its usefulness BMI has certain limitations. Factors such as age, sex, ethnicity, and muscle mass can influence the relationship between BMI and body fat. Also, BMI does not distinguish between excess fat, muscle, or bone mass, nor does it provide any indication of the distribution of fat among individuals.

The following are some examples of how certain variables can influence the interpretation of BMI:

- On average, older adults tend to have more body fat than younger adults for an equivalent BMI.
- On average, women have greater amounts of total body fat than men with an equivalent BMI
- Muscular individuals, or highly-trained athletes, may have a high BMI because of increased muscle mass

There is a vast ethnic difference seen when the distribution of body fat is considered. South Asians and Indians have a greater visceral adiposity in the form of truncal subcutaneous adipose tissue, which is metabolically more active and is associated with a higher cardiovascular risk. Thus BMI is less sensitive for determining adiposity in South Asians and Indian population as it does not distinguish fat from lean muscle mass.

The cut off values of BMI has been shown in the table below:

Nutritional Status	WHO criteria BMI cut-off	"Asian criteria" BMI cut-off
Underweight	<18.5	<18.5
Normal	18.5 – 24.9	18.5 – 22.9
Overweight	25 – 29.9	23 – 24.9
Pre-Obese	-	25 – 29.9
Obese	≥30	≥30
Obese Type 1 (obese)	30 – 40	30 – 40
Obese Type 2 (morbid obese)	40.1 – 50	40.1 – 50
Obese Type 3 (super obese)	>50	>50

Waist Circumference:

To overcome the limitations of BMI, waist circumference was used to measure abdominal obesity. Waist circumference is taken midway between lowest point on the costal margin and the iliac crest. It takes into account the visceral fat, the dangerous fat the surrounds the internal organs. But the drawback was that it did not take built of the person into account. Tall and short people cannot have a same cut off value. The cut off values of Waist circumference in different ethnic groups is tabulated as below:

Country of origin	Males(cm)	Females(cm)
USA(ATP III)	102	88
Europeans	94	80
Middle Eastern, African	94	80
Asians	90	80
Ethnic central Americans	90	80

Waist Circumference to height ratio:

It is a novel anthropometric parameter that also takes height (built) of the patient into account. It is a simple, easily measurable and gender independent marker that indicates early health risk for cardiovascular adverse effects.

Treatment of metabolic syndrome

Metabolic syndrome is a state of chronic low grade inflammation with the profound systemic effects. Clinical identification and management of patients with the Metabolic Syndrome are important to begin efforts to adequately implement the treatments to reduce their risk of subsequent diseases[113]. Effective preventive approaches include lifestyle changes, primarily weight loss, diet, and exercise, and the treatment comprises the appropriate use of pharmacological agents to reduce the specific risk factors. Pharmacological treatment should be considered for those whose

risk factors are not adequately reduced with the preventive measures and lifestyle changes[114].

1. Weight reduction:

Four therapies can be used for weight reduction: calorie restriction (e.g., 500 kcal/d deficit), increased physical activity, behavioral modification, and, in appropriate patients, FDA-approved weight-reducing drugs[128]. Several authors[17] recommend a weight loss goal of 10% reduction in body weight in the first six months to a year and continued weight loss thereafter until BMI is less than 25.

a. Diet: The effective and healthful methods for the long-term weight loss are reduced-energy diets, consisting of a modest 500 to 1000 calories/day reduction. In the PREMIER study[115], the Dietary Approaches to Stop Hypertension (DASH) diet plus lifestyle interventions improved the metabolic parameters, particularly blood pressure. ATP III[116] recommended that the diet should contain 25% to 35% of calories as total fat for the individuals entering cholesterol management. Low sodium intake, high potassium intake, intake of foods rich in MUFA and PUFA and intake of food with low glycemic index is recommended

b. Physical activity: Current physical activity guidelines[117] recommend practical, regular and moderate regimens for exercise. The standard exercise recommendation is a daily minimum of 30 minutes of moderate-intensity physical activity. The impact of exercise on insulin sensitivity is evident for 24 to 48 hours and disappears within three to five days. Thus, an individual would need to follow the AHA and American College of Sports Medicine recommendation to exercise at least 30 min/d most days of the week[118].

Physical training has been shown to reduce the skeletal muscle lipid levels and insulin resistance, regardless of BMI

c. Pharmacological therapy: The National Institutes of Health guidelines for the treatment of obesity recommend a consideration of pharmaceutical therapy for weight loss for the individuals with a BMI of at least 30 kg/m^2 or for those with a BMI of at least 27 kg/m^2 and co morbidities associated with their excess weight. Pharmacological approaches to weight loss include two main classes: appetite suppressants and inhibitors of nutrient absorption. A single agent is generally recommended and an average weight loss ranges greatly from 5% to 10% of initial weight[119]. Appetite suppressants include phentermine derivatives and sibutramine. Treatment with the drug decreases visceral fat, improves lipid levels, and decreases glycosylated haemoglobin and uric acid concentrations. Orlistat (an inhibitor of gastrointestinal lipase) is the only nutrient absorption inhibitor currently available. It prevents absorption of up to 30% of the fat consumed. Undesirable side effects such as flatulence and oil leakage in the stool often occur early in the course of treatment with this medication.

d. Bariatric surgery: Surgery is recommended for the individuals who do not respond to weight loss diet or medications, are extremely obese (BMI > 40 kg/m^2), or if they have a BMI > 35 to 40 kg/m^2 and one or more co morbid conditions[119].Bariatric surgery techniques using laparoscopic adjustable banding of stomach along with Roux-en-Y and other for Metabolic Syndrome of gastric bypass are now favored for the severe and morbid obesity[44]. It results in a weight loss of 25–30% and rapid normalization of glucose

handling and blood pressure in patients with diabetes and hypertension[120] with 95% of patients being free of the syndrome one year after a surgery[44].

2. **Dyslipidemia:** The guidelines recommend that the LDL-C goals should be set at less than 130 mg/dL with the option of targeting less than 100 mg/dL in the moderately high-risk individuals. Statins are considered to be the most effective class of drugs for reducing the LDL-C concentrations due to their minimal drug-drug interactions and side effects[116]. Depending on the dose and the specific type of statin used, LDL-C reductions of 15 to 60 mg/dL are observed[121]. Statins increase HDL-C by 5–10%, reduce TGs concentrations by 7–30%[122] and decrease very low density lipoprotein (VLDL) levels by 39%[123]. Non-lipid-lowering or pleiotropic effects of statins have also been implicated in their beneficial effects on inflammation, endothelial function, and CVD events. Niacin is considered the most effective agent for raising HDL-C (15 to 35%). The two fibrates currently used clinically are gemfibrozil and fenofibrate, both of which can lower TGs by 25% to 30%. Fibrates further increases HDL-C by 5–15% and reduces LDL-C by 0–30%[124].Bile acid sequestrants (BAS) and cholesterol absorption inhibitors (CAI) lower the LDL-C by decreasing the absorption of intestinal bile acids and cholesterol, respectively. BAS results in 15 to 30% reductions in LDL-C[125].
3. **Hypertension:** In the general population, pharmacologic treatment should be initiated when blood pressure is 150/90 mm Hg or higher in adults 60 years and older, or 140/90 mm Hg or higher in adults younger than 60 years. In patients with hypertension and diabetes, pharmacologic treatment should be initiated when blood pressure is 140/90 mm Hg or higher, regardless of age. Initial antihypertensive treatment should include a thiazide diuretic, calcium channel

blocker, ACE inhibitor, or ARB in the general nonblack population or a thiazide diuretic or calcium channel blocker in the general black population.

4. **Insulin Resistance:** In Metabolic Syndrome, patients with IFG (or IGT if assessed), weight reduction, increased physical activity, or both will delay (or prevent) the onset of T2DM[126]. In addition, metformin[126], thiazolidinediones[127], and acarbose[128] will lower the risk of T2DM in people with IFG or IGT.

A: assessment	Calculate Framingham risk score: high risk (10-year risk ≥20%), moderately high risk (10-year risk 10% to 20%), or lower to moderate risk (10-year risk ≤10%). Make diagnosis of Metabolic Syndrome using the diagnostic criteria.
A: aspirin	High risk: aspirin definitely beneficial. High-intermediate risk (10–20%): aspirin likely to be beneficial. Low-intermediate risk (6–10%): "individual clinical judgment", depending on sex and risk of bleeding. Low risk (<6%): Risk of haemorrhage outweighs the benefits.
B: BP control	Initiate treatment: categorical hypertension (BP ≥ 140/≥90 mm Hg). Patients with established diabetes (≥130/≥80 mm Hg). ACEIs/ARBs first line agent may reduce incident diabetes mellitus. Beta-blockers and thiazides may have an adverse effect on impaired glucose tolerance but outweighed by the benefits of reaching BP goal and lowering the risk of CVD events.
C: cholesterol First target: LDL Second target: non-HDL Third target: HDL Fourth target: CRP	Statin to achieve LDL-C <100 mg/dL in high-risk, <130 mg/dL in intermediate-risk, and <160 mg/dL in low risk patients. Statin intensification, consider niacin and/or fibrates once statin maximized. Consider fibrates, especially for those with combined hypertriglyceridemia/low HDL-C. Consider further reduction in LDL-C with statin therapy to mitigate a risk of low HDL-C, consider niacin. Statin therapy for those with high sensitivity CRP (hsCRP) ≥3 mg/dL.

D: diabetes prevention/diet	Intensive lifestyle modification is the most important therapy. Weight reduction of 5–10% of preintervention weight over a period of four to six months. Sodium intake of <65–100 mmol/day with a goal of 90–120 mmol of potassium per day. Mediterranean diet: high consumption of fruits, vegetables, legumes, and grains, moderate alcohol intake, a moderate-to-low consumption of dairy products and meats/meat products, and a high monounsaturated- to-saturated fat ratio. DASH diet: rich in fruits, vegetables, and low-fat dairy products, and low in saturated and total fat intake. Consider low glycemic index food, complex unrefined carbohydrates, viscous soluble fibres, protein intake of 10–35% of total calorie intake and 25% to 35% of calories as total fat. Metformin is second line in delaying the onset of T2DM. Thiazolidinediones (Pioglitazones) and alpha-glucosidase inhibitors (Acarbose) have shown benefit in smaller studies and are therefore third line.
E: exercise	Daily moderate intensity activity of minimum 30 minutes for most days of the week. Recommend use of pedometer with goal >10,000 steps/ day.

The above facts prompt extensive research to evaluate various anthropometric parameters to screen high risk patients. The future development of metabolic syndrome can be prevented by early lifestyle modification and behavioral therapy. Combined efforts of clinicians and health organizations are needed to mitigate this pandemic.

METHODOLOGY

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Study design and duration

The study design was a hospital based cross-sectional study.

Study period

This study was done for the period of one year from January 2016 to December 2016.

Place

The present study was carried out in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belagavi.

Source of Data

Patients above 18 years of age, who were diagnosed to have Metabolic Syndrome as per NCEP ATP III guidelines admitted in the Department of General Medicine, KLES Dr Prabhakar Kore Hospital and MRC, Belagavi

Sample size

A total of 150 patients with spontaneous Metabolic Syndrome were studied.

Sampling procedure

The sample size was determined considering the prevalence of Metabolic syndrome. According to the studies conducted in South Indian population the prevalence was found to be 30%. Thus by using the Chi Square test, the sample size was found to be 150.

Selection criteria

Inclusion Criteria

- All patients who fit in to the criteria of Metabolic syndrome admitted in KLE's Prabhakar Kore hospital and Medical Research Centre

Exclusion Criteria

- Spinal deformities
- Ascites, Abdominal mass, Pregnancy or any other causes of abdominal distension other than abdominal obesity

Ethical clearance

Prior to the commencement, the study was approved by the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi.

Informed consent

The patients who fulfilled the selection criteria were informed about the nature of study and a written informed consent was obtained (Annexure–I).

Data collection

The selected patients were interviewed for the demographic data, history of presenting illness and other comorbid conditions. Further these patients underwent clinical examination followed by systemic examination. Patients were evaluated for the following parameters on admission:

- Pulse rate was measured by palpatory method.
- Blood pressure was measured by a Sphygmomanometer on right upper arm.

The following anthropometric measurements were taken after an overnight fast

1. Weight : Weight was measured on a standard electronic weighing scale
2. Height: Height was measured on a Stadiometer
3. Waist Circumference: Waist circumference was measured at the narrowest part of the waist, between the lowest rib cage and the iliac crest

The following indices were calculated

BMI = Weight (kg) / Height(m^2)

Waist Height Ratio = Waist Circumference (cm) / Height(cm)

These findings were noted on a predesigned and pretested proforma (Annexure-II).

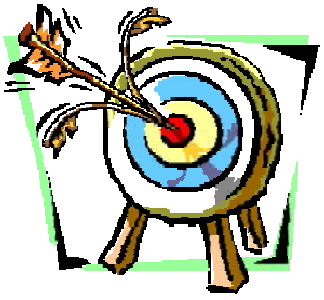
Investigations

Blood samples were collected from the subjects with all aseptic precautions. 10 ml of venous blood was collected from the median cubital vein by disposable plastic syringe. The needle was detached from the nozzle and blood transferred

immediately in to a dry, clean, ionized, graduated, screw capped plastic test tube with a gentle push avoid hemolysis. The following investigation were done:

1. FBS, PPBS / RBS
2. Lipid profile.
3. HbA1c levels
4. Other necessary investigations as and when required.

Introduction



Objectives

Review of Literature

Methodology

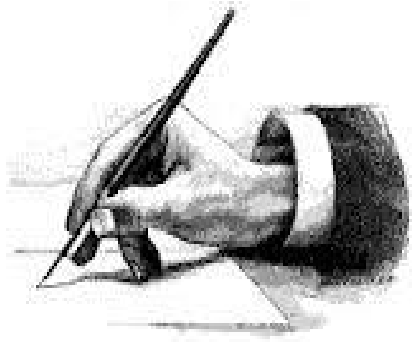
Results

Discussion

Conclusion

Summary

Bibliography



Annexure-I

Annexure-II

Annexure-III

Annexure-IV

Annexure-V

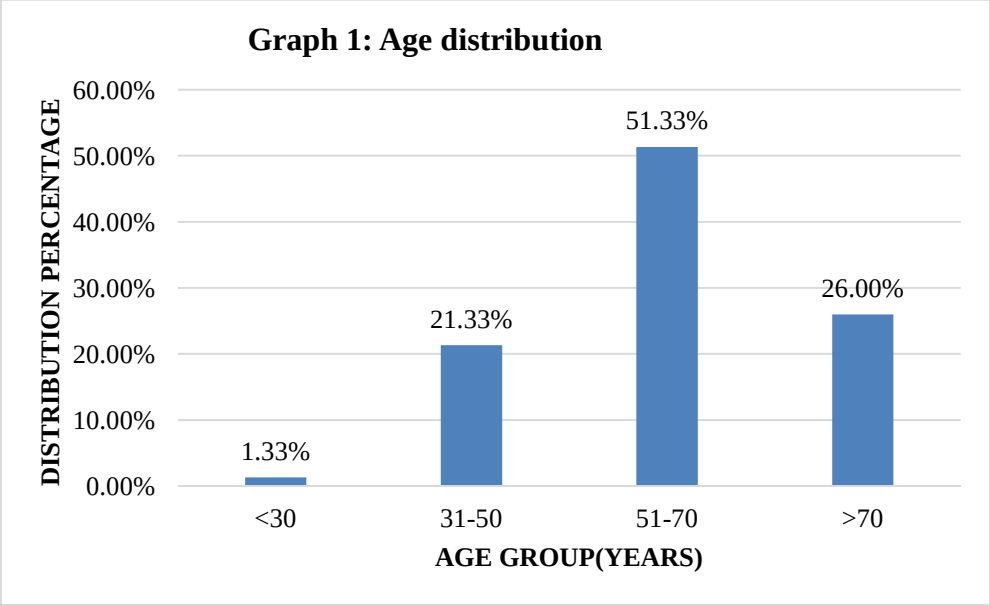
RESULTS

The present study was conducted on 150 patients presenting with Metabolic syndrome, in KLES Dr Prabhakar Kore hospital and MRC Belagavi during the period Jan 2016 to Dec 2016.

The data obtained was tabulated as below.

Table 1. Age distribution

Age group (Years)	Distribution (n=150)	
	Number	Percentage
<30	2	1.33
31 to 50	32	21.33
51 to 70	77	51.33
>70	39	26.00
Total	**150**	**100.00**

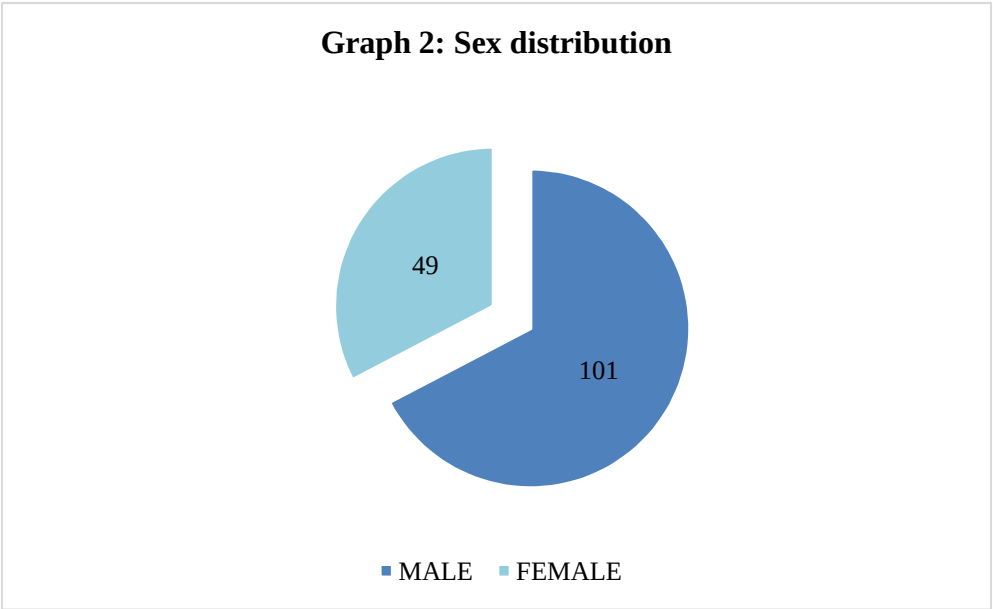


In the present study, patients' age ranged from 19-88 years, maximum number of patients were in the age group 51 to 70 years that is 77(51.33%), followed by 39(26%) patients in the age group of >70 years. The youngest patient was 19 years old and the oldest patient was 88 years old.

Table 2. Sex distribution

Sex	Distribution (n=150)	
	Number	**Percentage**
Male	101	67.33
Female	49	32.66
Total	**150**	**100.00**

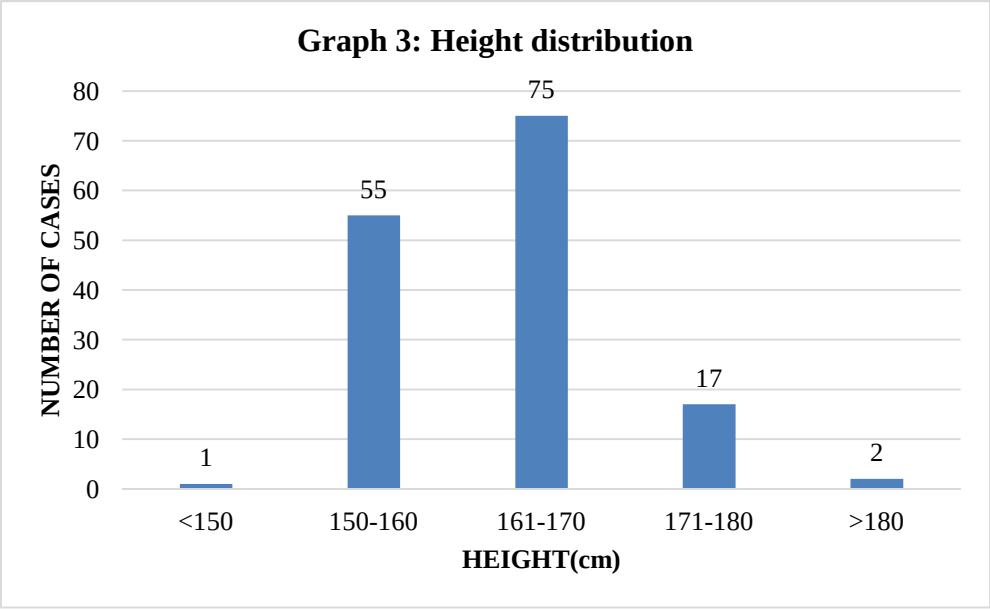
Graph 2: Sex distribution



In the present study out of 150 cases 101 (67.33%) were males and 49 (32.66%) were females. Male preponderance was seen with Male to Female ratio of 2.06 : 1.00

Table 3. Height distribution

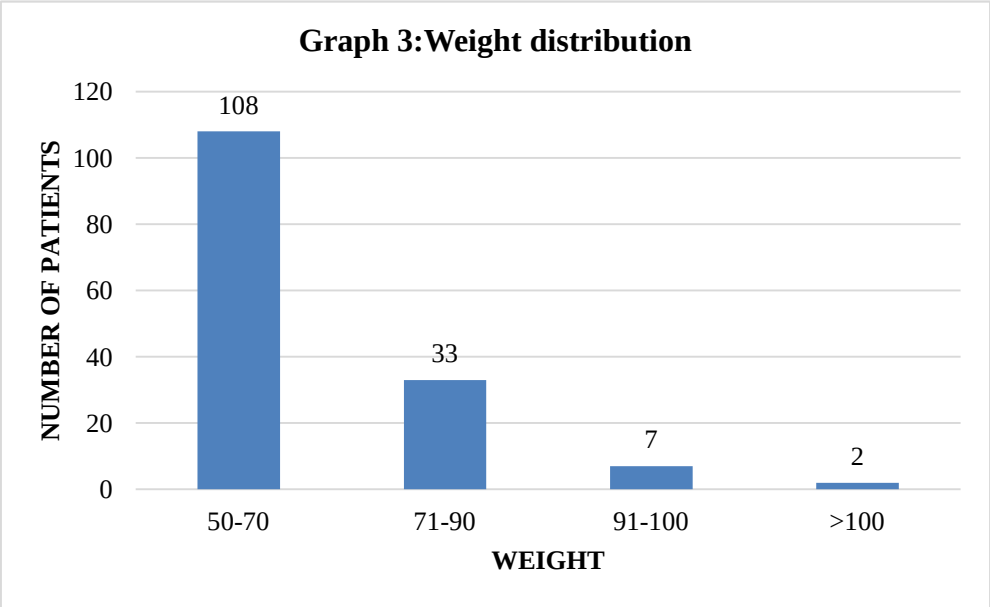
Height(cm)	Distribution (n=150)	
	Number	Percentage
<150	1	0.66
151-160	55	36.66
161-170	75	0.5
171-180	17	11.3
>180	12	8
Total	**150**	**100.00**



In the present study, maximum number of patients, 75 (50%) height ranged from 161-170cm, followed by 55 patients (36.66%) height ranged from 151-160cm. The lowest height seen was 148cm and maximum height was 184cm

Table 4. Weight distribution

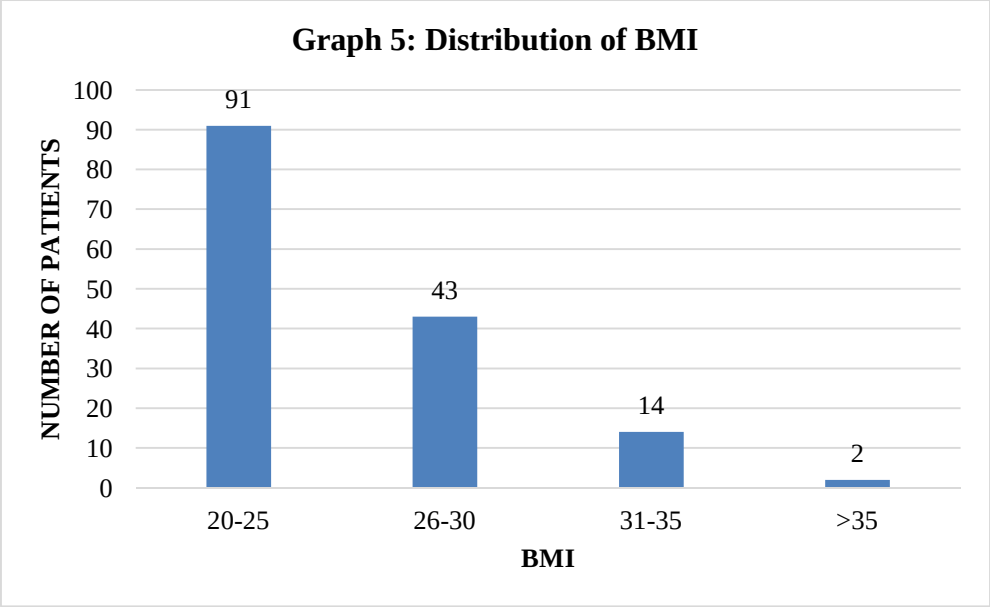
Weight(kg)	Distribution (n=150)	
	Number	**Percentage**
50-70	108	72
71-90	33	22
91-100	7	4.6
>100	2	1.3
Total	**150**	**100.00**



Out of the 150 patients studied, maximum number of patients weighed between 50-70kgs that is 108 patients(72%), followed by 33 patients (22%) weighed between 71-90kgs. The lowest weight recorded was 50kg, and the maximum weight recorded was 102kgs

Table 5. BMI distribution

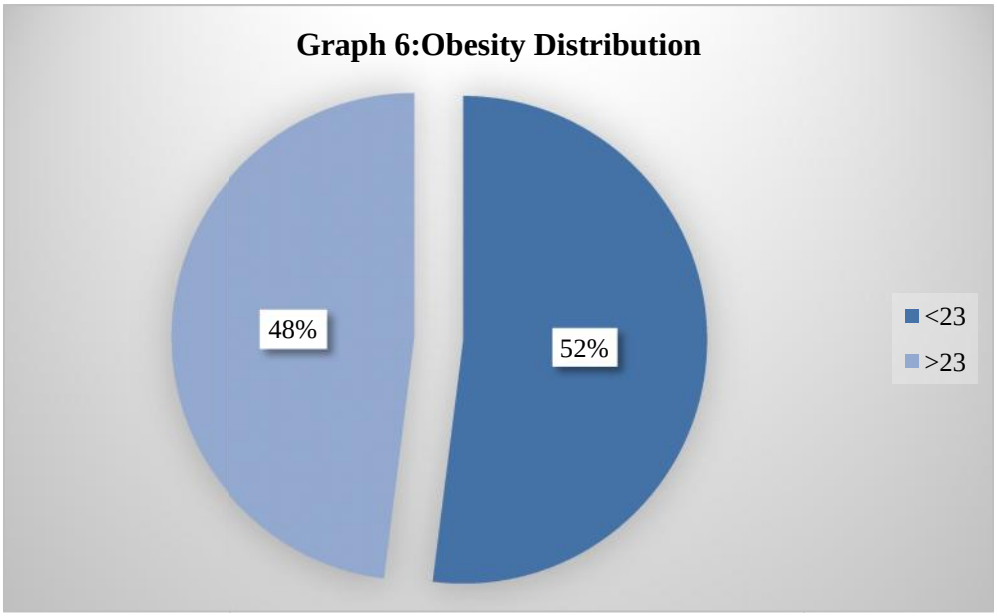
BMI(kg/m^2)	Distribution (n=150)	
	Number	**Percentage**
20-25	91	60.66
26-30	43	28.66
31-35	14	9.33
>35	12	8
Total	**150**	**100.00**



In the present study, the BMI of patients ranged from 20.5-35.8kg/m^2, maximum numbers of patients were in the BMI of 20-25 that is 91(60.66%), followed by 43 patients (28.66%) in the range of 26-30 BMI.

Table 6. Distribution of patients with BMI values (<23 / ≥23)

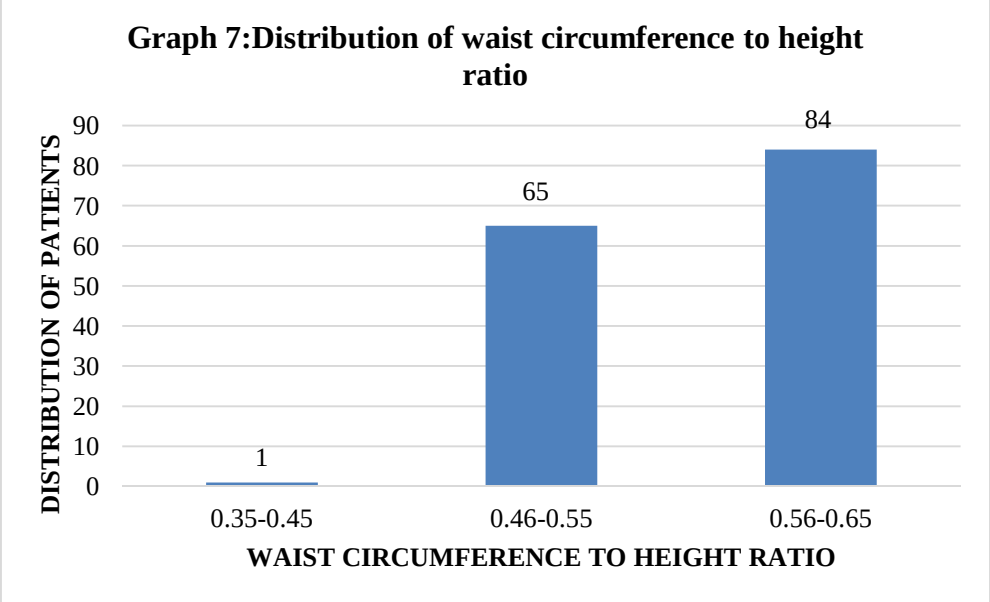
BMI(kg/m^2)	Distribution (n=150)	
	Number	**Percentage**
<23	78	52
≥23	72	48
Total	**150**	**100.00**



In the present study, out of 150 patients, 72 patients (48%) had BMI ≥ 23 and 78 patients (52%) had BMI <23. The lowest BMI was 20.5 and the highest BMI was 35.8.

Table 7: Waist height ratio(WHR) distribution

WHR	Distribution (n=150)	
	Number	Percentage
0.35-0.45	1	0.66%
0.46-0.55	65	43.33
0.56-0.65	84	56
Total	**150**	**100.00**

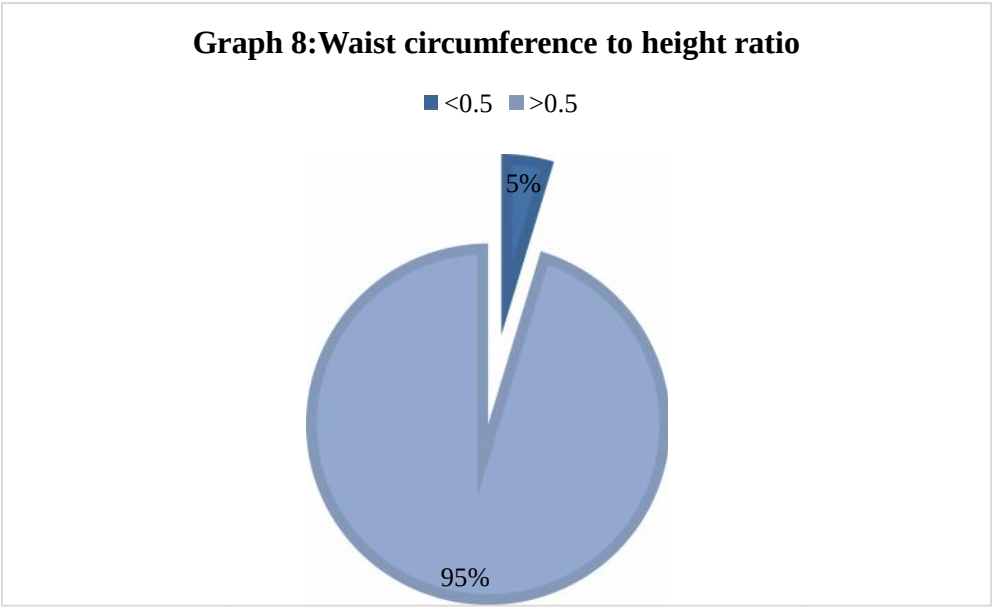


In the present study, maximum number of patients Waist height ratio ranged from 0.56-0.65 that is 84 patients (56%), followed by 65 patients (43.33%) WHR ranged from 0.46-0.55. The highest WHR recorded was 0.65 and the lowest WHR recorded was 0.38

Table 8: Comparison of Metabolic syndrome patients with WHR <0.5 / ≥ 0.5

WHR	Distribution (n=150)	
	Number	Percentage
<0.5	7	4.66
≥0.5	143	95.33
Total	**150**	**100.00**

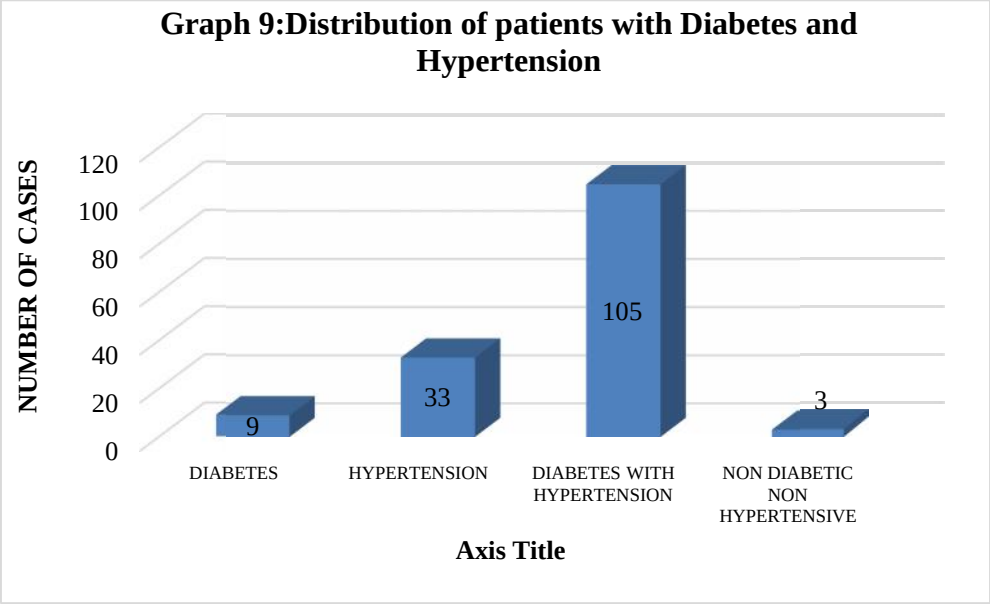
Graph 8:Waist circumference to height ratio



In the present study, out of 150 patients, 143 patients (95.33%) had WHR ≥ 0.5 whereas only 7 patients (4.66%) had WHR below 0.5

Table 9: Distribution of patients with Diabetes and Hypertension

Co morbidity	Distribution (n=150)	
	Number	Percentage
Diabetes	9	6
Hypertension	33	22
Diabetes and Hypertension	105	70
Others(Obesity/Dyslipidemia)	3	2
Total	150	100

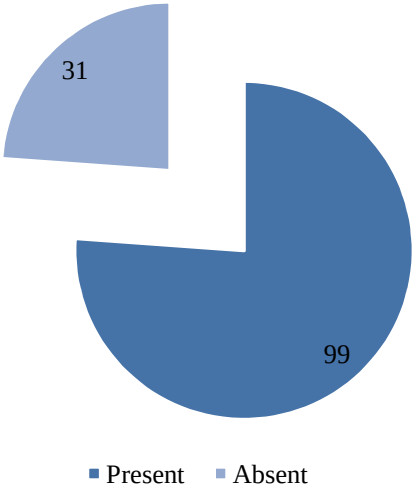


In the present study, out of 150 patients, 9 patients (6%) had Diabetes, 33 patients (22%) had hypertension, 105 patients (70%) had both Diabetes and Hypertension and 3 patients (2%) had neither Diabetes nor Hypertension but had other components of Metabolic syndrome

Table 10: Distribution of Dyslipidemia

Patients with Dyslipidemia	Distribution (n=150)	
	Number	Percentage
Present	99	66
Absent	51	34
Total	150	100

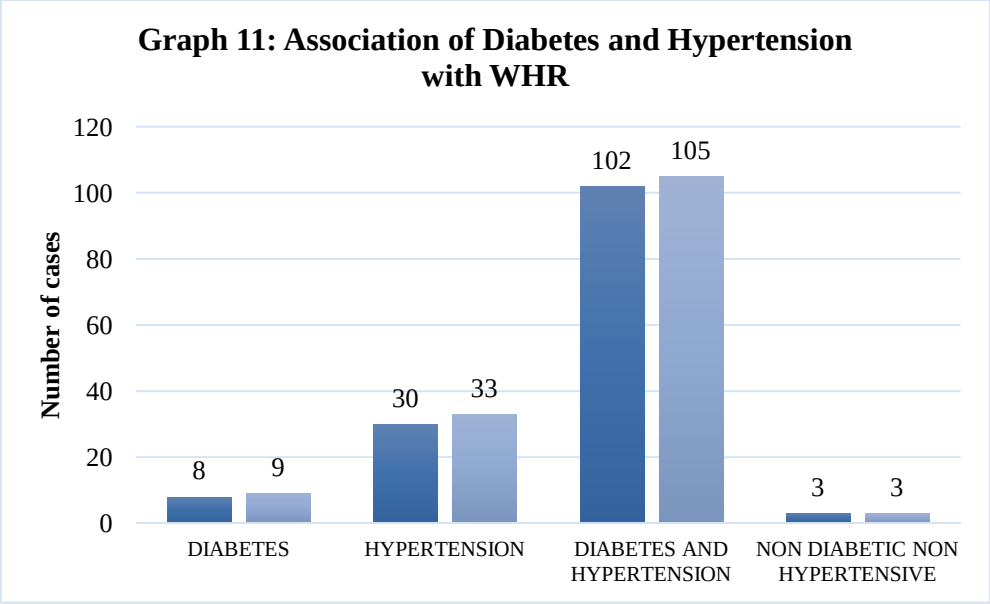
Graph 10: Distribution of Dyslipidemia



In the present study, out of 150 patients, 99 patients (66%) had dyslipidemia, whereas 51 patients (34%) did not have dyslipidemia.

Table 11: Association of WHR with Metabolic components:

Co morbidity	WHR			
	≥ 0.5	< 0.5	Total	Percentage(WHR≥0.5)
Diabetes	8	1	9	88.88
Hypertension	30	3	33	90.90
Diabetes and Hypertension	102	3	105	97.14
Other	3	0	3	100



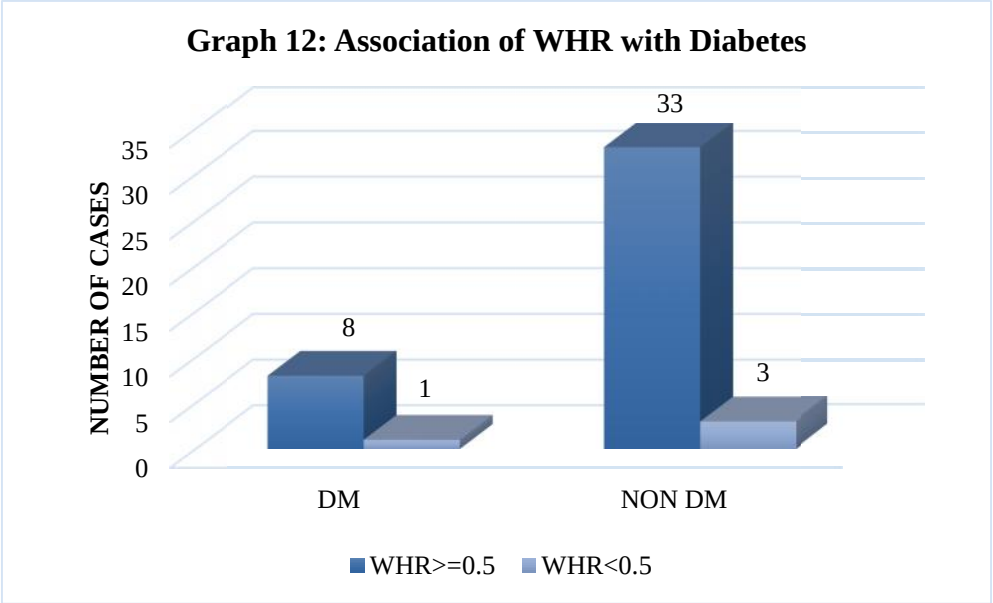
In our present study, the association of various components of Metabolic syndrome with WHR is shown in the above table

Table 12: Association of Diabetes with WHR

Co morbidity	WHR ≥ 0.5	WHR < 0.5
DIABETIC	**8**	**1**
NON DIABETIC	**33**	**3**

P value: 0.999

Graph 12: Association of WHR with Diabetes

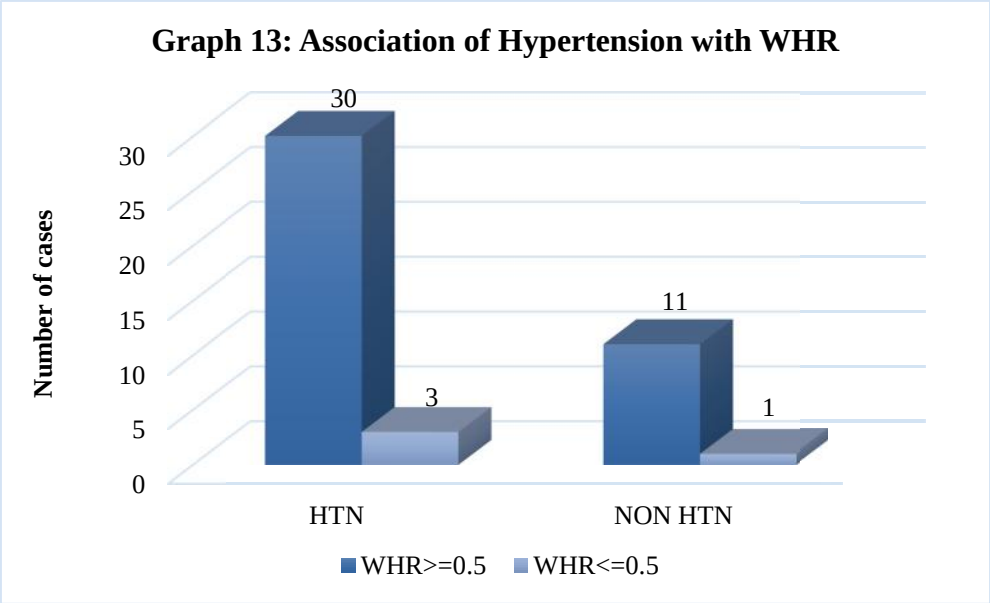


In the present study, out of 9 patients with Diabetes, 8 patients had WHR ≥ 0.5, and out of 36 non diabetic patients, 33 patients had WHR < 0.5. The p value was found to be 0.999 which was statistically not significant

Table 13: Association of Hypertension with WHR:

Co morbidity	WHR ≥ 0.5	WHR < 0.5
HYPERTENSION	**30**	**3**
NON HYPERTENSION	**11**	**1**

P value: 0.999

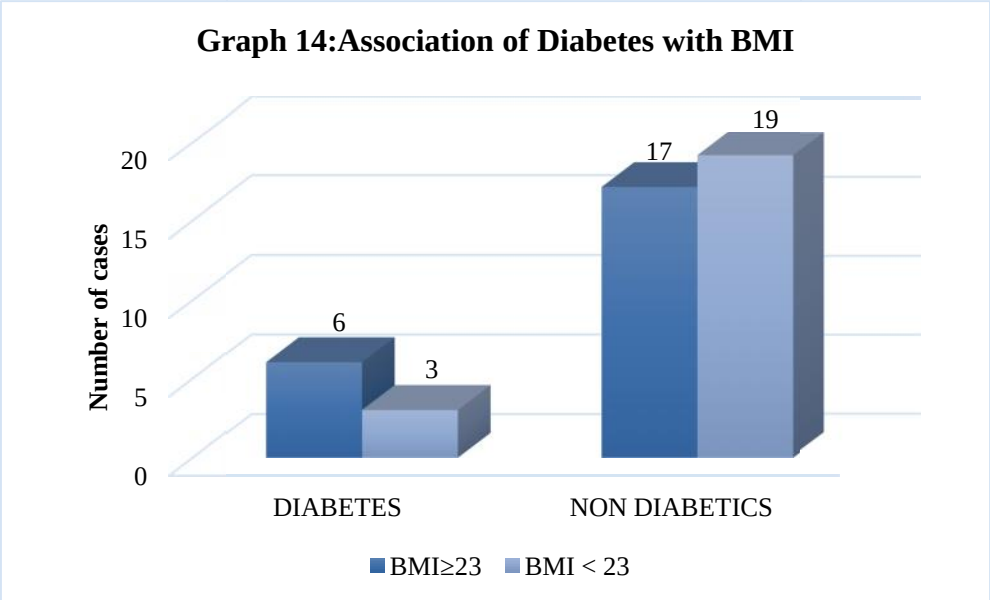


In the present study, out of 33 patients with Hypertension, 30 patients had WHR ≥ 0.5 and 3 had WHR < 0.5. And out of 12 patients who were non hypertensive, 11 patients had WHR ≥ 0.5 and 1 patient had WHR < 0.5. The p value was 0.999 which is statistically not significant

Table 14: Association of Diabetes with BMI

Co morbidity	Distribution		Total
	BMI ≥ 23	BMI < 23	
DIABETES	6	3	9
NON DIABETES	17	19	36

P value : 0.4591

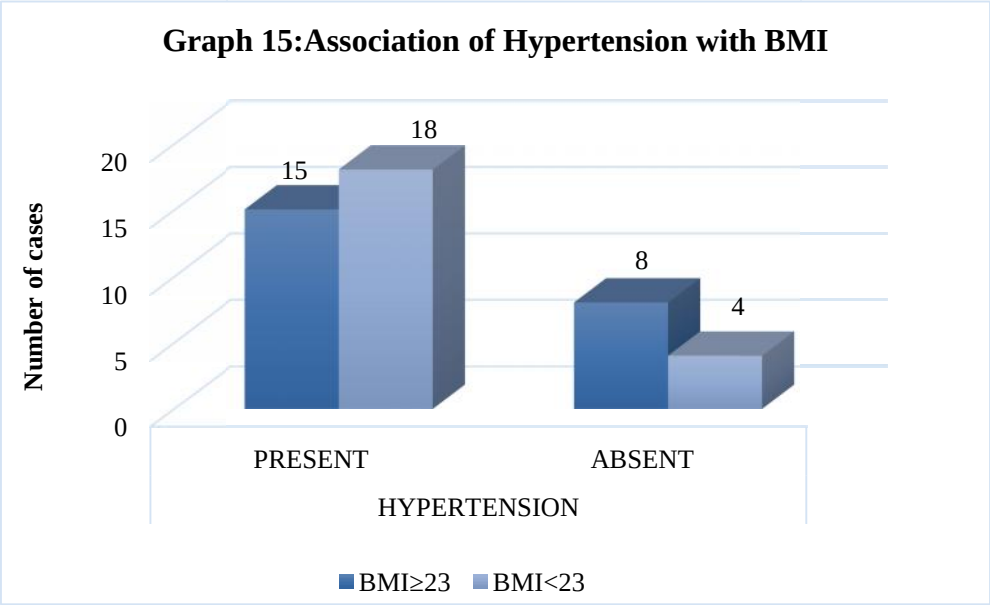


In the present study out of the 9 patients with Diabetes, 6 patients (66.66%) patients had BMI>23, whereas 3 patients (33.33%) patients had BMI below 23. The P value was 0.4591 which was statistically insignificant.

Table 15: Association of Hypertension with BMI

Co morbidity	Distribution		
	BMI ≥ 23	**BMI < 23**	**Total**
HYPERTENSION	15	18	33
NON HYPERTENSION	8	4	12

P value: 0.3141

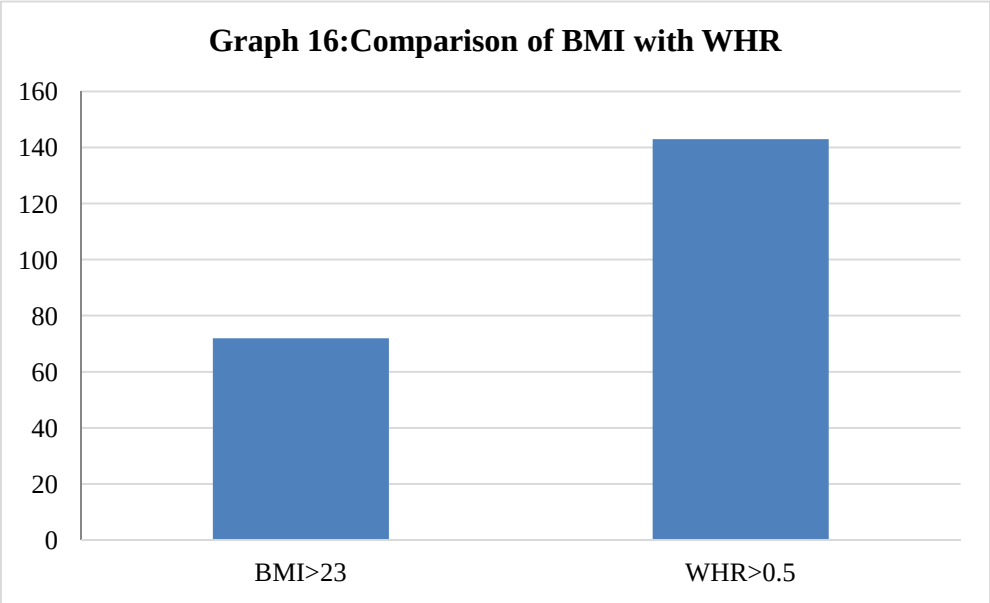


In the present study, out of 33 patients with Hypertension, 15 patients were obese, with BMI ≥ 23**.** And out of 12 non hypertensive patients, 8 patients had BMI ≥ 23. The P value was 0.3141 which was statistically insignificant.

Table 16: Comparison of BMI with WHR

Patients with Metabolic syndrome	Distribution (n=150)	
	Number	Percentage
BMI ≥ 23	72	48
WHR ≥ 0.5	143	95.33

P value: <0.0001



In the present study, out of 150 cases 143 had WHR > 0.5; hence the detection rate of WHR for metabolic syndrome is 95.31%. Whereas only 72 out of 150 patients had BMI > 23. The P value was found to be <0.0001 which was statistically significant. Thus WHR was a far better predictor for metabolic syndrome than the routinely used counterpart, BMI. This is in co relation with the study done by Rajesh Rajput, Meena Rajput et al.

DISCUSSION

In the present study, 150 patients with Metabolic syndrome were studied and were compared to various anthropometric parameters and the same was compared to components of Metabolic syndrome.

In our study, the patients' age ranged from 19-88years. The maximum number of patients were in the age group of 51-70years that is 77 patients (51.33%) followed by 39 patients (26%) in the age group > 70years. This is almost similar to the study done by Anthonia O Ogbera et al.

Taking gender into consideration, we observed in our study that Male patients, 101(67.33%) were more as compared to female patients, 49(32.66%) with Male to Female ratio of 2.06:1.00. Similar conclusion was drawn by most of the authors, study done by SD Hsieh, H Yoshinaga et al.

On comparing the weight of the patients, most of the patients weighed between 50-70kgs that is 108 patients(72%) and only 42 patients weighed > 70kgs. The mean weight was 67kgs. Similar observations were made by Farzad Shidfar, Fatemah Alborzi et al.

Taking BMI into account, 91 patients (60.66%) were in the BMI group of 20-25; 57 patients (38%) were in the group of >25 to <35. Similar observations were done by Fatemah et al and Yoshinaga et al.

Similarly height of the patients was studied, and most of the patients' height was between 150-180cm. only 12 patients had a height of > 180cm. This is similar to the study done by H Yoshinaga et al.

Taking into consideration the cut off values of BMI, we observed 78 patients (52%) were having BMI < 23 and remaining 72 patients had BMI > 23. In our study we found the average BMI of the patients to be 24.9kg/m^2, whereas a study by H Yoshinaga et al found average BMI of 22kg/m^2. In our study mean BMI of males was found to be 25.2kg/m^2 and that of females was found to be 24.7kg/m^2

On comparing Waist Height ratio, we observed 65 patients(43.33%) were in the groups of 0.46-0.55, 84 patients(56%) were in the group of 0.56-0.65. Most of the authors have not compared Waist Height ratio in relation to various groups in their study population.

We tried to compare various Metabolic syndrome patients with Waist Height Ratio and found, 143 patients(95.33%) were having ratio ≥ 0.5 and only 7 patients(4.60%) had ratio < 0.5. Studies by different authors have compared individual metabolic components with Waist Height ratio whereas in our study we have tried comparing Waist Height ratio with Metabolic syndrome as a whole.

When an attempt was made to look for individual components of Metabolic syndrome, we found Hypertension 33 patients(22%), Diabetes Mellitus 9 patients(6%), Diabetes and Hypertension together 105 patients(70%) and other 3 patients had obesity and dyslipidemia.

Patients with Diabetes mellitus or Hypertension alone or together did have overlapping of either obesity or dyslipidemia. Some studies have done compared distribution of Diabetes Mellitus alone, Hypertension alone and Dyslipidemia alone like Ashwell et al.

On comparing other components of Metabolic syndrome that is dyslipidemia 99 patients (66%) had dyslipidemia whereas 51 patients(34%) did not have dyslipidemia. Studies done by D. S. Prasad, Z. Kabir et al have found dyslipidemia in one of their components 46%.

We tried comparing individual components of Metabolic syndrome with Waist Height Ratio and found to have various results depicted in Table no 11. Study done by Rajesh Rajput et al found similar results as our study.

An attempt to find association of Diabetes with Waist height Ratio was done and found to have 8 patients of Diabetes had ratio of ≥ 0.5, and 1 had ratio <0.5. Of the remaining 36 patients 33 had ratio ≥ 0.5 and 3 had < 0.5. Thus we found that Waist Height Ratio above the cutoff of ≥ 0.5 occurred in similar frequency in both Diabetics and non diabetics in our study population. The p value was 0.9999 which was statistically insignificant. Similar study done by Margaret Ashwell, Sigrid Gibson et al did not find significant correlation between Diabetes and Waist Hip ratio.

Similarly we tried to find association of Hypertension with Waist Height Ratio and found to have 30 patients of hypertension had ratio ≥ 0.5, and 3 had ratio < 0.5. Of the remaining 12 patients 11 had ratio ≥ 0.5 and 1 had < 0.5. This is in sharp contrast to study done by PE Mishra, L Shastri et al where they found significant correlation between Hypertension and Waist Height ratio

Similarly, Diabetes was compared with Body Mass Index. Out of 9 diabetic patients, 6 patients (66.66%) had BMI ≥ 23 and 3 patients(33.33%) had BMI < 23. The P value was 0.45 which was statistically insignificant. A study done by Farzad

Shidfar, Fatemah Alborzi et al also found no significance on comparison of Diabetes with Body Mass Index.

An attempt was made to find association of Hypertension with Body Mass Index. Out of the 33 patients with Hypertension 15 patients(45.45%) had BMI ≥ 23 and 18 patients(54.54%) had BMI < 23. The P value was 0.3141 which was statistically insignificant. In our study we did not find significant correlation between Hypertension and Body Mass Index, whereas a study by FarzadShidfar, Fatemah Alborzi et al and Margaret Ashwell, Sigrid Gibson et al found significant correlation

Comparison of Body Mass Index and Waist Height Ratio revealed 72 patients had BMI ≥ 23 and 143 patients had WHR ≥ 0.5. The detection rate of Waist Height Ratio for Metabolic syndrome was found to be 95.31%. FarzadShidfar, Fatemah Alborzi et al and Margaret Ashwell, Sigrid Gibson et al also had similar observation in their study.

Finally we have drawn conclusion by analyzing our 150 patients of Metabolic syndrome comparing various components of Metabolic syndrome with factors Age, Sex, Weight , Height, individual components of Metabolic syndrome or together and found to have significant correlation with Age, Mean BMI, Height of the patients.

However our correlation between factors like Diabetes and Hypertension with BMI, Diabetes and Hypertension with WHR did not have significance.

We feel necessary to overcome these biases and to find whether there is true association with these factors, and that needs more number of patients with Metabolic syndrome. May be because of small sample size we were not able to correlate these factors.

CONCLUSION

In the present study of 150 patients with Metabolic syndrome, we observed a significant correlation with various factors.

Based on the findings of the present study, the prominent features are:

1. Patients above 50years of age, had significant association with Metabolic syndrome
2. Comparison of Metabolic syndrome with Waist Height Ratio had significant correlation.
3. Similarly on comparing Waist Height Ratio with Body Mass Index in patients with Metabolic syndrome, Waist Height Ratio had significant correlation.
4. Patients with Diabetes alone had no significant correlation with BMI/WHR
5. Similarly patients with Hypertension alone did not have significant correlation with BMI/WHR
6. We did not correlate dyslipidemia patients with WHR or BMI because of overlapping of cases.

The above limitation is owing to a small sample size of 150, a large sample size is required to overcome this bias

SUMMARY

In the present study of 150 patients titled, Waist Circumference to Height Ratio as a screening tool in the assessment of Metabolic syndrome and its comparison with Body Mass Index, conducted during the study period, Jan 2016 to Dec 2016, in the department of General Medicine, KLE'S Dr Prabhakar Kore Hospital and MRC, Belagavi, the findings are summarized as follows

1. There were more patients of Metabolic syndrome above the age of 50years
2. In our study there were 67.33% Males and 32.66% Females
3. Patients with Metabolic syndrome presented with Diabetes/Hypertension, Diabetes and Hypertension together, Dyslipidemia and Obesity
4. Majority of patients with Metabolic syndrome had Waist Height Ratio ≥ 0.5
5. Out of 150 patients, 105 patients were with Diabetes and Hypertension
6. Patients of Dyslipidemia in our study were 99 patients(66%), but were not compared to WHR or BMI because of overlapping
7. Diabetes and Hypertension did not have correlation with BMI or WHR when compared individually.
8. There were more number of patients with Metabolic syndrome with WHR ≥ 0.5, 95.33%, whereas only 48% patients with Metabolic syndrome had BMI ≥ 23

On comparing with other studies, correlation was found with Age, Height, Mean BMI and WHR.

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ANNEXURE I

CONSENT FOR PARTICIPATION IN RESEARCH

Mr. /Mrs._____ we are requesting you to enroll yourself in study titled **"WAIST CIRCUMFERENCE TO HEIGHT RATIO AS A SCREENING TOOL IN THE ASSESSMENT OF METABOLIC SYNDROME AND ITS COMPARISON WITH BODY MASS INDEX" -**A Study conducted by J.N.Medical College, Belagavi.

You have been requested to participate in research because your profile matches with the study group. All the patients admitted with Metabolic Syndrome can become participants of study. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge.

Your participation in the research is absolutely voluntary. Your decision to participate in the study or otherwise will not affect your relationship with J.N.M.C. If you decide not participate you are free to withdraw at any time.

The purpose of research is to study the Waist Height Ratio in the assessment of Metabolic Syndrome and compare it with the routinely used anthropometric parameter BMI.

Procedure involved

Detailed history taking, clinical examination, measurement of anthropometric parameters and blood investigations were done

Risks and benefits

There are no risks involved and benefits are many. The study helps to screen high risk individuals from the general population who are more likely to get Metabolic Syndrome using a more sensitive parameter, Waist Height Ratio instead of the conventionally used anthropometric parameter Body Mass Index. The results deduced

at the end of study will help all similar patients admitted in the hospital to assess their prognosis.

Alternatives

Even if you decline to participate, there will not be any change in the line of your management or the relationship with your doctor. You will be told about all the new information that may affect your decision to participate in the study.

Withdrawing/removal from study

You can withdraw any time from the study as you wish. You will not be penalized for such a decision.

Privacy and confidentiality

The only people to know that you are a research subject are the members of research team. No information about you or provided by you during the research will be disclosed to others without your written permission except in case of emergency to protect your rights and welfare, if required by law.

Financial incentives for participation

You will not be paid any monetary benefits or free gifts for participation in the research. You will not be reimbursed for expenses.

Authorization to publish results

When the results of the research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

CONSENT STATEMENT

I, the undersigned, have been explained in my own vernacular language about the study and my participation in the study is voluntary. If I want I can withdraw at any time. Also I have been given enough time to clear my doubts about the study and my rights as a study participant.

In case you have any questions about your rights as a study participant you can contact Dr. Ganga Pilli (0831-2471350).

Signature or the left thumb impression of the participant or legally authorized representative.

Participant's name: _____Signature: _____

Witness name: _____ Signature: _____

Experimenter's name: _____ Signature: _____

Guardian's name: _____ Signature: _____

Place: _____

Date: _____

ANNEXURE II – PROFORMA

WAIST CIRCUMFERENCE TO HEIGHT RATIO AS A SCREENING TOOL IN THE ASSESSMENT OF METABOLIC SYNDROME AND ITS COMPARISON WITH BODY MASS INDEX- a cross sectional study at KLE'S Dr. Prabhakar Kore Hospital and MRC, Belagavi

Name of the patient :

In Patient Number :

Age :

Sex :

Date of Admission :

Occupation :

Address :

Chief Complaints: :

Risk factors:

Sedentary lifestyle, Family history, Medication intake

Diagnosis:

General physical examination

Build & nourishment	:	Vital signs	
Height	:	PR	:
Weight	:		
Waist Circumference:		BP	:

Systemic Examination:

Cardiovascular System :

Respiratory System :

Per Abdomen :

Central Nervous System :

Investigations :

RBS:

FBS / PPBS:

HbA1c:

Lipid Profile:

Seria	In pa	Age(Y	Sex	Comp	Hy	Di	Pulse (
1	757803	63	M	Imbalance on walking	P	P	80
4	757983	49	M	Fever, Sore throat	P	P	68
5	757450	74	M	Syncope	P	A	70
6	757402	36	M	Headache, Vomiting	P	A	130
7	757418	83	M	Polyuria	P	P	80
8	757284	79	M	Chest pain	P	P	90
9	757229	72	M	Nocturia	P	P	80
10	756688	56	M	Slurring of speech	P	A	88
11	756729	59	M	Jaundice	P	P	80
12	756369	74	M	Altered sensorium	P	P	90
13	755662	46	M	Weakness of right upper and lower limb	A	P	82
16	754764	58	M	Headache	P	P	80
17	754113	62	M	Ulcer on the tongue	P	P	80
18	753250	60	M	Slurring of speech	P	A	104
19	753053	62	M	Facial puffiness	P	P	80
24	751163	71	M	Giddiness	A	P	80
25	751083	72	M	Drowsiness	P	P	100
27	750334	55	M	Burning micturition	P	P	80
28	750429	49	M	Dyspnea on exertion	A	P	80
29	750001	74	M	Tremors, Reduced activity	P	P	84
32	749567	32	M	Swellimg of left lower limb	P	A	80
36	748443	62	M	Imbalance on walking	P	P	96
37	747769	65	M	Burning micturition	P	P	94
38	747726	72	M	Vertigo	P	P	62
40	746438	62	M	Weakness of right upper and lower limb	P	A	84
41	746202	37	M	Headache with Seizures	P	P	100
44	745325	66	M	Weakness of all 4 limbs	P	P	80
46	745298	50	M	Giddiness	P	P	100
47	745189	70	M	Chest pain	P	A	100
48	745068	65	M	Burning micturition	P	P	66
49	477674	61	M	Weakness of right upper and lower limb	P	P	82
50	744759	58	M	Dyspnea on exertion	P	P	56
52	743997	64	M	Giddiness	A	P	80
54	743486	63	M	Weakness of left upper and lower limb	P	P	80
56	743256	54	M	Dyspnea	A	P	80
57	743199	64	M	Sore throat	P	P	80

58	743150	71	M	Seizures	P	P	80
60	742927	71	M	Fever	P	P	92
61	742983	50	M	Polyuria	P	P	72
62	742831	58	M	Headache	P	P	82
64	742070	58	M	Acute loss of vision	P	P	80
65	741573	74	M	Frequency, Hesitency of micutrition	P	P	82
66	741497	69	M	Left sided tingling, numbness	P	A	80
69	740807	68	M	Headache with vomiting	P	P	100
70	740658	59	M	Seizures	P	P	80
71	740452	45	M	Weakness of right upper and lower limb	P	P	100
72	740413	33	M	Left sided limb weakness	P	P	80
73	740134	62	M	Cough	P	P	80
74	739601	33	M	Pain in right lower limb	P	P	80
75	739449	52	M	Weakness of left upper and lower limb	A	P	90
78	738314	32	M	Sore throat	A	A	74
80	736766	75	M	Fever with Seizures	P	P	96
82	736714	48	M	Weakness of right upper and lower limb	P	A	72
83	736065	72	M	Chest pain	P	P	80
84	758506	34	M	Weakness of both lower limbs, pain and tingling of right hand	P	P	90
88	758785	39	M	Fever with chills	P	A	90
89	758816	65	M	Loose stools	P	P	78
90	758985	64	M	Giddiness	P	P	68
91	759037	81	M	Weakness of right upper and lower limb	P	P	80
92	759006	66	M	Fever with chills	P	P	80
93	759208	78	M	Seizures	P	A	86
94	759676	65	M	Weakness of left upper and lower limb	P	P	80
95	759609	35	M	Headache	P	P	86
98	759909	19	M	Weakness of left upper and lower limb	A	A	102
100	760208	73	M	Cough with expectoration	P	A	98
103	760254	78	M	Slurring of speech	A	P	88
104	760382	66	M	Cough	P	P	80
106	760932	76	M	Loss of memory	P	P	80
108	760992	62	M	Headache	P	P	72
109	761138	65	M	Seizures	P	P	88
110	761139	52	M	Weakness of right upper and lower limb	P	P	80
111	761336	66	M	Pain and swelling over right side of face	P	P	80
115	761631	66	M	Cough with expectoration	P	A	56
116	761674	72	M	Weakness of right upper and lower limb	P	A	80
117	761866	64	M	Vomiting, Altered sensorium	P	P	80
118	761923	54	M	Slurring of speech	P	A	98
119	762136	44	M	Syncope	P	P	80
120	761954	44	M	Headache	P	A	80
121	761956	65	M	Chest pain	P	P	88
122	762032	52	M	Headache	P	P	80
123	762050	65	M	Headache, Weakness of right upper and lower limb	P	P	98
124	763791	46	M	Vertigo	P	A	110
126	763931	46	M	Altered sensorium	P	P	120
127	763994	82	M	Weakness of left upper and lower limb	P	P	68
128	764180	54	M	Slurring of speech	P	A	98
130	764259	55	M	Weakness of right upper and lower limb	P	P	78
131	764414	62	M	Dyspnea on exertion	P	P	80
132	764413	53	M	Weakness of left upper and lower limb	P	A	80

134	764751	70	M	Pain and redness over left eye	P	P	72
135	764054	65	M	Weakness of right upper and lower limb	P	P	88
136	764973	81	M	Palpitations	P	P	90
138	764947	60	M	Seizures	P	P	80
139	764960	63	M	Giddiness	P	P	80
140	765185	48	M	Dyspnea on exertion	P	A	99
141	765413	36	M	Blurring of vision	P	P	88
142	765571	64	M	Burning micturition	P	P	80
143	765569	49	M	Headache	P	P	88
144	765564	56	M	Weakness of left lower limb	P	P	80
145	765889	51	M	Headache, diplopia	P	A	74
147	766208	55	M	Seizures	P	P	88
150	766925	62	M	Weakness of right upper and lower limb	P	P	80

							System ic									
(m mH		Wt	He	Wa	Bo	wa	Re	Ca	Pe	Ce		Hb	ing lipids			
Sy	Di										RBS		Total c	Low d	High d	Trigly
160	80	70	162	88	26.6	0.54	Clear	S1 S2 heard	Soft	Ataxia	171	8.3	142	89	26	141
130	80	64	168	90	22.7	0.53	Clear	S1 S2 heard	Soft	WNL	224		161	82	20	488
160	80	67	174	94	22.1	0.54	Clear	S1 S2 heard	Soft	WNL	86	7.3	208	151	38	95
160	100	102	184	104	30.1	0.56	Clear	S1 S2 heard	Soft	eck stiffne	394					
110	70	72	179	94	22.5	0.52	Clear	S1 S2 heard	Soft	WNL	476	13	199	104	33	261
170	90	61	170	86	21.1	0.5	Clear	S1 S2 heard	Soft	WNL	190	11	98	42	41	175
170	100	70	160	92	27.3	0.57	Clear	S1 S2 heard	Soft	WNL	154		172	104	27	204
140	90	62	168	90	21.9	0.53	Clear	S1 S2 heard	Soft	Dysarthria	205	5.7	200	139	31	95
110	70	70	163	95	26.3	0.58	Clear	S1 S2 heard	Soft	WNL	200		126	58	49	59
120	70	64	154	90	26.9	0.58	Clear	S1 S2 heard	Soft	levant tall	76					
150	90	68	160	95	26.5	0.59	Clear	S1 S2 heard	Soft	nt hemipl	140		181	93	40	237
170	100	62	168	86	21.9	0.51	Clear	S1 S2 heard	Soft	WNL	164	7.9	213	147	38	138
100	70	61	166	86	22.1	0.51	Clear	S1 S2 heard	Soft	WNL	276	11	186	98	34	180
170	100	70	176	102	22.5	0.57	Clear	S1 S2 heard	Soft	nt hemipa	129		193	136	37	98
140	90	63	170	90	21.7	0.52	Clear	S1 S2 heard	Soft	WNL	214	10	144	75	117	117
110	70	85	169	102	29.7	0.6	Clear	S1 S2 heard	Soft	WNL	52	6.1				
160	90	78	168	101	27.6	0.6	Clear	S1 S2 heard	Soft	Drowsy	282					
160	100	60	162	84	22.8	0.51	Clear	S1 S2 heard	Soft	WNL	167	7.4	222	165	40	165
170	100	62	158	80	24.8	0.5	Clear	ESM +	Soft	WNL	94		226	135	32	189
160	90	70	175	102	22.8	0.58	Clear	S1 S2 heard	Soft	Rigidity	233	8				
120	70	90	170	84	31.1	0.49	Clear	S1 S2 heard	Soft	WNL	289	7.6	211	140	38	166
166	100	62	166	84	22.4	0.5	Clear	S1 S2 heard	Soft	incoordin	324	12	184	113	42	143
100	70	67	160	82	26.1	0.51	Clear	S1 S2 heard	Soft	WNL	121	6.8	199	141	38	98
140	90	64	168	86	22.6	0.51	Clear	S1 S2 heard	Soft	incoordi	157	11	146	100	29	146
220	100	93	174	110	30.7	0.63	Clear	S1 S2 heard	Soft	nt hemipa	106		209	145	42	209
140	90	65	170	102	22.4	0.6	Clear	S1 S2 heard	Soft	WNL	341	9.4	176	83	64	142
160	100	64	168	90	22.6	0.53	Clear	S1 S2 heard	Soft	uadripare	130	6.2	238	158	58	108
146	90	63	168	91	22.3	0.54	Clear	S1 S2 heard	Soft	t incordia	200	12	206	123	24	295
80	60	69	175	90	22.5	0.51	Clear	S1 S2 heard	Soft	WNL	208		125	49	36	200
160	100	65	163	94	24.4	0.57	Clear	S1 S2 heard	Soft	WNL	229	8	263	49	49	174
130	80	58	160	88	22.6	0.55	Clear	S1 S2 heard	Soft	ense hem	125		165	100	36	146
150	90	86	176	104	27.7	0.59	Clear	S1 S2 heard	Soft	WNL	90	8.7	158	88	43	133
120	70	59	164	94	21.9	0.57	Clear	S1 S2 heard	Soft	incoordin	148		149	78	31	197
190	70	74	170	92	25.6	0.54	Clear	S1 S2 heard	Soft	t hemipar	131	6.9	121	26	43	261
160	90	60	158	90	24	0.56	eath soun	S1 S2 heard	Soft	WNL	154	8	174	83	35	282
130	80	63	168	92	22.3	0.54	Clear	S1 S2 heard	Soft	WNL	218	9.9	107	42	38	133

100	70	69	168	104	24.4	0.61	Clear	S1 S2 heard	Soft	Drowsy	389	12	100	34	49	86
120	70	64	168	87	22.6	0.51	Clear	S1 S2 heard	Soft	WNL	180		192	104	50	192
130	80	63	156	90	25.8	0.57	Clear	S1 S2 heard	Soft	WNL	315	13	181	117	39	126
160	90	66	172	103	22.3	0.59	Clear	S1 S2 heard	Soft	WNL	229	7.8	228	53	157	87
210	120	86	164	103	31.9	0.62	Clear	S1 S2 heard	Soft	PL Absen	222		235	128	44	299
160	80	60	164	92	22.3	0.56	Clear	S1 S2 heard	Soft	WNL	304	9.4	198	130	41	133
190	100	63	164	102	23.4	0.62	Clear	S1 S2 heard	Soft	emihypoa	137		193	122	36	175
180	100	64	171	98	22.1	0.57	Clear	S1 S2 heard	Soft	Drowsy	281	8.5	131	76	28	133
140	92	68	167	86	24.3	0.51	Clear	S1 S2 heard	Soft	WNL	312	6.8	124	79	35	50
180	100	78	169	103	27.3	0.6	Clear	S1 S2 heard	Soft	dense hem	291		153	84	53	82
100	70	62	166	102	22.4	0.61	Clear	S1 S2 heard	Soft	axic hemi	219	8.4	156	101	49	31
130	80	60	166	105	21.7	0.63	t basal c	S1 S2 heard	Soft	WNL	102	8.3	127	78	42	36
170	100	59	163	88	22.2	0.53	Clear	S1 S2 heard	Soft	WNL	480	10	104	63	30	56
200	100	87	168	106	30.8	0.6	Clear	S1 S2 heard	Soft	axic hemi	183	7.4	196	119	34	217
130	90	88	175	102	28.7	0.58	Clear	S1 S2 heard	Soft	WNL	144		264	184	32	243
140	90	63	168	102	22.3	0.6	Clear	S1 S2 heard	Soft	eck stiffne	341	8.6	109	50	45	71
190	90	64	167	102	22.9	0.61	Clear	S1 S2 heard	Soft	dense hem	99		275	130	38	231
110	70	60	163	92	22.5	0.56	Clear	S1 S2 heard	Soft	WNL	361	11	142	84	38	103
190	100	94	162	103	35.8	0.63	Clear	S1 S2 heard	Soft	uadripare	249	8.2	159	106	33	102
150	90	83	164	104	30.8	0.63	Clear	S1 S2 heard	Soft	WNL	125		214	149	36	145
140	80	59	162	96	22.4	0.59	Clear	S1 S2 heard	Soft	WNL	343	8	156	109	31	78
150	80	62	164	89	23	0.54	Clear	S1 S2 heard	Soft	WNL	313	9.4	152	102	34	79
130	80	64	168	106	22.6	0.63	Clear	S1 S2 heard	Soft	t hemipa	214	8.4	184	117	55	61
160	90	94	169	102	32.9	0.6	Clear	S1 S2 heard	Soft	WNL	127	12	288	217	48	115
160	80	72	163	101	27	0.61	Clear	S1 S2 heard	Soft	WNL	71		187	142	28	84
100	70	62	158	90	24.8	0.56	Clear	S1 S2 heard	Soft	axic hemi	74		78	131	30	84
190	90	64	168	86	22.6	0.51	Clear	S1 S2 heard	Soft	WNL	394	12	170	119	23	141
110	70	92	168	105	32.5	0.62	Clear	S1 S2 heard	Soft	axic hemi	92		164	48	14	509
160	80	84	169	103	29.4	0.6	teral Ro	S1 S2 heard	Soft	WNL	85		117	69	39	42
110	80	66	165	96	24.2	0.58	Clear	S1 S2 heard	Soft	Dysarthri	65		152	90	32	151
120	70	64	168	103	22.6	0.61	Clear	S1 S2 heard	Soft	WNL	118	5.5	165	94	41	149
130	70	59	161	89	22.7	0.55	Clear	S1 S2 heard	Soft	ive impa	104	9	193	21	113	297
210	100	66	160	82	25.7	0.51	Clear	S1 S2 heard	Soft	WNL	213	8.4	96	68	17	56
160	100	73	165	101	26.8	0.61	Clear	S1 S2 heard	Soft	WNL	269	7.8	183	122	42	97
170	100	82	168	101	29	0.6	Clear	S1 S2 heard	Soft	t hemipa	252		190	113	49	138
120	90	56	158	80	22.4	0.5	Clear	S1 S2 heard	Soft	WNL	421	16	161	105	30	132
180	70	71	165	88	26.6	0.53	basal c	S1 S2 heard	Soft	WNL	82		130	78	35	85
200	100	76	167	104	27.2	0.62	Clear	S1 S2 heard	Soft	WNL	96		121	71	28	108
150	90	94	174	101	31	0.58	Clear	S1 S2 heard	Soft	Drowsy	388	12	143	75	44	119
160	100	54	156	86	22	0.55	Clear	S1 S2 heard	Soft	arapares	128	6.5	199	135	28	179
180	100	86	166	102	31.2	0.61	Clear	S1 S2 heard	Soft	WNL	485	11	261	185	55	104
110	70	60	166	81	21.7	0.48	Clear	S1 S2 heard	Soft	WNL	92		235	158	35	211
180	100	57	159	82	22.5	0.51	Clear	S1 S2 heard	Soft	WNL	166	5.7	207	133	34	81
160	70	61	166	88	22.1	0.52	Clear	S1 S2 heard	Soft	WNL	376	13	111	53	32	132
170	90	57	159	79	22.5	0.49	Clear	S1 S2 heard	Soft	t hemipl	128	12	154	94	34	129
100	60	78	172	101	26.3	0.58	Clear	S1 S2 heard	Soft	uadriplec	111		140	97	28	76
130	80	94	163	105	35.3	0.64	Clear	S1 S2 heard	Soft	Irritable	341	9.3	133	71	40	111
130	70	57	159	88	22.5	0.55	Clear	S1 S2 heard	Soft	ncoordin	163	8.4	182	118	35	147
160	100	53	154	79	22.3	0.51	Clear	S1 S2 heard	Soft	arapares	128	6.5	199	135	28	179
110	70	74	168	101	26.2	0.6	Clear	S1 S2 heard	Soft	t hemipa	147	7.8	235	175	43	85
140	90	74	164	90	27.5	0.54	Clear	S1 S2 heard	Soft	WNL	163	8.7	140	87	28	127
150	90	76	171	101	25.9	0.59	Clear	S1 S2 heard	Soft	hemipar	284	7.5	96	43	39	68

170	100	86	172	102	29.1	0.59	Clear	S1	S2	heard	Soft	WNL	165	12	227	149	59	96
140	90	61	166	91	22.1	0.54	Clear	S1	S2	heard	Soft	ıt hemipl	342	14	172	106	29	184
130	90	80	172	102	27	0.59	Clear	S1	S2	heard	Soft	WNL	82	8.6	150	67	69	71
140	80	66	174	86	21.7	0.49	Clear	S1	S2	heard	Soft	Gait ataxi	158	8	163	113	23	137
140	90	63	168	91	22.3	0.54	Clear	S1	S2	heard	Soft	and gait	243	9.5	298	144	39	579
110	70	95	170	102	32.8	0.6	ıteral cre	S1	S2	heard	Soft	WNL	118		216	169	30	85
160	90	74	166	102	26.8	0.61	Clear	S1	S2	heard	Soft	WNL	281	7.6	206	140	44	109
140	90	80	164	102	29.7	0.62	Clear	S1	S2	heard	Soft	WNL	151	8.1	119	51	49	95
150	90	64	171	89	21.8	0.52	Clear	S1	S2	heard	Soft	WNL	351	11	176	117	32	134
160	70	63	168	94	22.3	0.55	Clear	S1	S2	heard	Soft	limb mo	214	7.8	159	29	29	111
140	80	56	158	79	22.4	0.5	Clear	S1	S2	heard	Soft	ck stiffne	117		157	118	34	162
170	90	86	164	103	31.9	0.62	Clear	S1	S2	heard	Soft	WNL	227	8.6	199	142	41	41
160	90	75	164	102	27.8	0.62	Clear	S1	S2	heard	Soft	t hemipa	114	7.8	184	119	44	105

Serial number	In patient number	Age(years)	Sex	Complaint at presentation											Systemic examination						Fasting lipi		
					Hypertension	Diabetes mellitus	Pulse (/min)	BP (mmHg)		Weight (kgs)	Height (cms)	Waist circumference (cm)	Body mass index (Kg/m2)	waist circumference to Height ratio	Respiratory system	Cardiovascular system	Per abdomen	Central nervous system	RBS	HbA1C	Total cholesterol (mg/dL)	Low density lipoprotein (mg/dL)	High density lipoprotein (mg/dL)
								Systolic	Diastolic														
1	757803	63	M	Imbalance on walking	P	P	80	160	80	70	162	88	26.6	0.54	Clear	S1 S2 heard	Soft	Ataxia	171	8.3	142	89	26
2	757544	65	F	Pruritis	P	P	90	100	70	50	148	75	28.76	0.5	Clear	S1 S2 heard	Soft	WNL	356	10.4	206	89	101
3	758281	70	F	Tingling right arm	P	A	80	110	70	50	155	80	22.6	0.51	Clear	S1 S2 heard	Soft	Hypoaesthesia	134	6	246	158	44
4	757983	49	M	Fever, Sore throat	P	P	68	130	80	50	168	90	22.7	0.53	Clear	S1 S2 heard	Soft	WNL	224		161	82	20
5	757450	74	M	Syncope	P	A	70	160	80	50	174	94	22.1	0.54	Clear	S1 S2 heard	Soft	WNL	86	7.3	208	151	38
6	757402	36	M	Headache, Vomiting	P	A	130	160	100	50	184	104	30.12	0.56	Clear	S1 S2 heard	Soft	Neck stiffness	394		150	77	65
7	757418	83	M	Polyuria	P	P	80	110	70	50	179	94	22.47	0.52	Clear	S1 S2 heard	Soft	WNL	476	13.4	199	104	33
8	757284	79	M	Chest pain	P	P	90	170	90	51	170	86	21.1	0.5	Clear	S1 S2 heard	Soft	WNL	190	11.3	98	42	41
9	757229	72	M	Nocturia	P	P	80	170	100	51	160	92	27.3	0.57	Clear	S1 S2 heard	Soft	WNL	154		172	104	27
10	756688	56	M	Slurring of speech	P	A	88	140	90	51	168	90	21.9	0.53	Clear	S1 S2 heard	Soft	Dysarthria	205	5.7	200	139	31
11	756729	59	M	Jaundice	P	P	80	110	70	51	163	95	26.3	0.58	Clear	S1 S2 heard	Soft	WNL	200		126	58	49
12	756369	74	M	Altered sensorium	P	P	90	120	70	52	154	90	26.9	0.58	Clear	S1 S2 heard	Soft	Irrelevant talking	76		144	100	55
13	755662	46	M	Weakness of right upper and lower limb	A	P	82	150	90	52	160	95	26.5	0.59	Clear	S1 S2 heard	Soft	Right hemiplegia	140		181	93	40
14	755813	77	F	Dyspnea on exertion	P	P	94	130	70	53	156	90	26.6	0.57	Clear	S1 S2 heard	Soft	WNL	191	7.2	190	120	52
15	755168	56	F	Giddiness	P	P	88	180	70	53	153	79	21.3	0.51	Clear	S1 S2 heard	Soft	WNL	413		188	127	22
16	754764	58	M	Headache	P	P	80	170	100	54	168	86	21.9	0.51	Clear	S1 S2 heard	Soft	WNL	164	7.9	213	147	38
17	754113	62	M	Ulcer on the tongue	P	P	80	100	70	54	166	86	22.1	0.51	Clear	S1 S2 heard	Soft	WNL	276	11.1	186	98	34
18	753250	60	M	Slurring of speech	P	A	104	170	100	54	176	102	22.5	0.57	Clear	S1 S2 heard	Soft	Right hemiparesis	129		193	136	37
19	753053	62	M	Facial puffiness	P	P	80	140	90	54	170	90	21.7	0.52	Clear	S1 S2 heard	Soft	WNL	214	10.3	144	75	117
20	751992	70	F	Tremors	P	P	100	160	100	54	155	80	20.8	0.51				Tremors	173		213	84	26
21	751725	65	F	Weakness of right upper and lower limb	P	P	120	220	120	54.5	154	72	21	0.46	Clear	S1 S2 heard	Soft	Right hemiparesis	300	9	209	137	31
22	751847	56	F	Cough, Fever	P	P	90	190	90	55	160	88	23.4	0.55	Right basal crepts	S1 S2 heard	Soft	WNL	128	9	130	89	18
23	751295	50	F	Loss of consciousness	P	A	80	110	70	56	165	90	29.3	0.54	Clear	S1 S2 heard	Soft	E2 M1 V1	250	6.3	150	81	33
24	751163	71	M	Giddiness	A	P	80	110	70	56	169	102	29.7	0.6	Clear	S1 S2 heard	Soft	WNL	52	6.1	165	74	55
25	751083	72	M	Drowsiness	P	P	100	160	90	56	168	101	27.6	0.6	Clear	S1 S2 heard	Soft	Drowsy	282		174	88	65
26	750529	52	F	Left sided weakness	P	A	80	100	70	56	169	90	22.7	0.53	Clear	S1 S2 heard	Soft	Left dense hemiplegia	227	9.9	227	161	30
27	750334	55	M	Burning micturition	P	P	80	160	100	56	162	84	22.8	0.51	Clear	S1 S2 heard	Soft	WNL	167	7.4	222	165	40
28	750429	49	M	Dyspnea on exertion	A	P	80	170	100	56	158	80	24.8	0.5	Clear	ESM +	Soft	WNL	94		226	135	32
29	750001	74	M	Tremors, Reduced activity	P	P	84	160	90	56	175	102	22.8	0.58	Clear	S1 S2 heard	Soft	Rigidity	233	8	198	120	52
30	749900	74	F	Blurring of vision	P	P	88	200	120	57	160	90	22.6	0.56	Clear	S1 S2 heard	Soft	WNL	200	6.5	158	83	55
31	749747	47	F	Weakness of right upper and lower limb	P	P	88	170	100	57	160	94	26.6	0.58	Clear	S1 S2 heard	Soft	Right dense hemoplegia	178	7.2	201	102	60
32	749567	32	M	Swellimg of left lower limb	P	A	80	120	70	57	170	84	31.1	0.49	Clear	S1 S2 heard	Soft	WNL	289	7.6	211	140	38
33	749353	70	F	Loss of vision	P	P	80	150	90	58	152	76	22.5	0.5	Clear	S1 S2 heard	Soft	Bilateral cortical blindness	169	8.4	178	126	30
34	748814	70	F	Pain bilateral knee joint	A	P	80	170	80	58	156	60	22.6	0.38	Clear	S1 S2 heard	Soft	WNL	131		130	8	28
35	748522	83	F	Weakness of all 4 limbs	P	P	90	100	70	59	154	78	21.5	0.5	Clear	S1 S2 heard	Soft	Bilateral quadriparesis with areflexia	85		88	34	36
36	748443	62	M	Imbalance on walking	P	P	96	166	100	59	166	84	22.4	0.5	Clear	S1 S2 heard	Soft	Left incoordination	324	11.7	184	113	42
37	747769	65	M	Burning micturition	P	P	94	100	70	59	160	82	26.1	0.51	Clear	S1 S2 heard	Soft	WNL	121	6.8	199	141	38

Serial number	In patient number	Age(years)	Sex	Complaint at presentation											Systemic examination						Fasting lipi		
					Hypertension	Diabetes mellitus	Pulse (/min)	BP (mmHg)		Weight (kgs)	Height (cms)	Waist circumference (cm)	Body mass index (Kg/m2)	waist circumference to Height ratio	Respiratory system	Cardiovascular system	Per abdomen	Central nervous system	RBS	HbA1C	Total cholesterol (mg/dL)	Low density lipoprotein (mg/dL)	High density lipoprotein (mg/dL)
								Systolic	Diastolic														
38	747726	72	M	Vertigo	P	P	62	140	90	59	168	86	22.6	0.51	Clear	S1 S2 heard	Soft	Right incoordination	157	10.6	146	100	29
39	747471	55	F	Palpitations	A	P	90	140	90	59	156	90	20.5	0.57	Clear	S1 S2 heard	Soft	WNL	165		187	127	41
40	746438	62	M	Weakness of right upper and lower limb	P	A	84	220	100	60	174	110	30.7	0.63	Clear	S1 S2 heard	Soft	Right hemiparesis	106		209	145	42
41	746202	37	M	Headache with Seizures	P	P	100	140	90	60	170	102	22.4	0.6	Clear	S1 S2 heard	Soft	WNL	341	9.4	176	83	64
42	745591	74	F	Altered sensorium	P	P	130	90	60	60	152	90	30.2	0.59	Clear	S1 S2 heard	Soft	WNL	249	11.2	232	157	57
43	745516	72	F	Left sided weakness	P	A	80	130	80	60	158	89	22.4	0.56	Clear	S1 S2 heard	Soft	Left hemiparesis	152		126	67	39
44	745325	66	M	Weakness of all 4 limbs	P	P	80	160	100	60	168	90	22.6	0.53	Clear	S1 S2 heard	Soft	Quadriparesis	130	6.2	238	158	58
45	745325	88	F	Aphasia	A	A	80	100	70	60	155	90	22.4	0.5	Clear	S1 S2 heard	Soft	Aphasia	100		145	98	33
46	745298	50	M	Giddiness	P	P	100	146	90	60	168	91	22.3	0.54	Clear	S1 S2 heard	Soft	Left incordiation	200	12.4	206	123	24
47	745189	70	M	Chest pain	P	A	100	80	60	60	175	90	22.5	0.51	Clear	S1 S2 heard	Soft	WNL	208		125	49	36
48	745068	65	M	Burning micturition	P	P	66	160	100	61	163	94	24.4	0.57	Clear	S1 S2 heard	Soft	WNL	229	8	263	49	49
49	477674	61	M	Weakness of right upper and lower limb	P	P	82	130	80	61	160	88	22.6	0.55	Clear	S1 S2 heard	Soft	Right dense hemoplegia	125		165	100	36
50	744759	58	M	Dyspnea on exertion	P	P	56	150	90	61	176	104	27.7	0.59	Clear	S1 S2 heard	Soft	WNL	90	8.7	158	88	43
51	744237	75	F	Weakness of right upper and lower limb	P	P	88	160	90	61	169	103	22.4	0.6	Clear	S1 S2 heard	Soft	Right dense hemiplegia	363	12.4	192	118	63
52	743997	64	M	Giddiness	A	P	80	120	70	62	164	94	21.9	0.57	Clear	S1 S2 heard	Soft	Left incoordination	148		149	78	31
53	743765	69	F	Seizures	P	P	100	120	70	62	150	98	28.4	0.65	Clear	S1 S2 heard	Soft	WNL	195	10.4	200	126	55
54	743486	63	M	Weakness of left upper and lower limb	P	P	80	190	70	62	170	92	25.6	0.54	Clear	S1 S2 heard	Soft	Left hemiparesis	131	6.9	121	26	43
55	743401	50	F	Fever with chills	P	A	70	110	70	62	158	102	22.4	0.64	Clear	S1 S2 heard	Soft	WNL	131		210	106	69
56	743256	54	M	Dyspnea	A	P	80	160	90	62	158	90	24	0.56	Bronchial breath sounds bilaterally	S1 S2 heard	Soft	WNL	154	8	174	83	35
57	743199	64	M	Sore throat	P	P	80	130	80	62	168	92	22.3	0.54	Clear	S1 S2 heard	Soft	WNL	218	9.9	107	42	38
58	743150	71	M	Seizures	P	P	80	100	70	62	168	104	24.4	0.61	Clear	S1 S2 heard	Soft	Drowsy	389	12.4	100	34	49
59	749175	73	F	Seizures	P	P	82	160	90	62	156	90	21.3	0.57	Clear	S1 S2 heard	Soft	Drowsy	253	8.9	104	48	31
60	742927	71	M	Fever	P	P	92	120	70	62	168	87	22.6	0.51	Clear	S1 S2 heard	Soft	WNL	180		192	104	50
61	742983	50	M	Polyuria	P	P	72	130	80	63	156	90	25.8	0.57	Clear	S1 S2 heard	Soft	WNL	315	13.4	181	117	39
62	742831	58	M	Headache	P	P	82	160	90	63	172	103	22.3	0.59	Clear	S1 S2 heard	Soft	WNL	229	7.8	228	53	157
63	742333	82	F	Headache	P	P	112	190	100	63	158	90	21.6	0.56	Clear	S1 S2 heard	Soft	WNL	141	5.6	268	178	76
64	742070	58	M	Acute loss of vision	P	P	80	210	120	63	164	103	31.9	0.62	Clear	S1 S2 heard	Soft	PL Absent	222		235	128	44
65	741573	74	M	Frequency, Hesitency of micutrition	P	P	82	160	80	63	164	92	22.3	0.56	Clear	S1 S2 heard	Soft	WNL	304	9.4	198	130	41
66	741497	69	M	Left sided tingling, numbness	P	A	80	190	100	63	164	102	23.4	0.62	Clear	S1 S2 heard	Soft	Left hemihypoasthesia	137		193	122	36
67	741400	45	F	Seizures	P	P	110	100	60	63	156	94	22.1	0.6	Clear	S1 S2 heard	Soft	WNL	223		210	136	52
68	741111	60	F	Weakness of left upper and lower limb	P	P	84	150	90	63	158	101	22.4	0.63	Clear	S1 S2 heard	Soft	Left hemiparesis	125		173	104	61
69	740807	68	M	Headache with vomiting	P	P	100	180	100	63	171	98	22.1	0.57	Clear	S1 S2 heard	Soft	Drowsy	281	8.5	131	76	28
70	740658	59	M	Seizures	P	P	80	140	92	63	167	86	24.3	0.51	Clear	S1 S2 heard	Soft	WNL	312	6.8	124	79	35
71	740452	45	M	Weakness of right upper and lower limb	P	P	100	180	100	64	169	103	27.3	0.6	Clear	S1 S2 heard	Soft	Right dense hemiplegia	291		153	84	53
72	740413	33	M	Left sided limb weakness	P	P	80	100	70	64	166	102	22.4	0.61	Clear	S1 S2 heard	Soft	Left ataxic hemiparesis	219	8.4	156	101	49
73	740134	62	M	Cough	P	P	80	130	80	64	166	105	21.7	0.63	Right basal crepts	S1 S2 heard	Soft	WNL	102	8.3	127	78	42
74	739601	33	M	Pain in right lower limb	P	P	80	170	100	64	163	88	22.2	0.53	Clear	S1 S2 heard	Soft	WNL	480	10.4	104	63	30

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					Hypertension	Diabetes mellitus	Pulse (/min)	BP (mmHg)		Weight (kgs)	Height (cms)	Waist circumference (cm)	Body mass index (Kg/m2)	waist circumference to Height ratio	Respiratory system	Cardiovascular system	Per abdomen	Central nervous system	RBS	HbA1C	Total cholesterol (mg/dL)	Low density lipoprotein (mg/dL)	High density lipoprotein (mg/dL)
								Systolic	Diastolic														
75	739449	52	M	Weakness of left upper and lower limb	A	P	90	200	100	64	168	106	30.8	0.6	Clear	S1 S2 heard	Soft	Left ataxic hemiparesis	183	7.4	196	119	34
76	739193	60	F	Weakness of right upper and lower limb	P	A	100	140	90	64	158	93	21.2	0.58	Clear	S1 S2 heard	Soft	Right hemiplegia	135		231	146	35
77	738768	65	F	Bilateral lower limb weakness	P	P	82	130	80	64	153	92	21.3	0.6	Clear	S1 S2 heard	Soft	Asymmetrical paraparesis	151	10.6	166	103	43
78	738314	32	M	Sore throat	A	A	74	130	90	64	175	102	28.7	0.58	Clear	S1 S2 heard	Soft	WNL	144		264	184	32
79	737835	67	F	Weakness of right upper and lower limb	P	P	60	150	90	64	155	93	25.8	0.6	Clear	S1 S2 heard	Soft	Right ataxic hemiparesis	384	14.3	163	96	50
80	736766	75	M	Fever with Seizures	P	P	96	140	90	64	168	102	22.3	0.6	Clear	S1 S2 heard	Soft	Neck stiffness	341	8.6	109	50	45
81	736501	69	F	Weakness of right upper and lower limb	P	P	100	180	100	64	152	90	22.07	0.59	Clear	S1 S2 heard	Soft	Right hemiplegia	241	10.2	253	153	87
82	736714	48	M	Weakness of right upper and lower limb	P	A	72	190	90	64	167	102	22.9	0.61	Clear	S1 S2 heard	Soft	Right dense hemiplegia	99		275	130	38
83	736065	72	M	Chest pain	P	P	80	110	70	64	163	92	22.5	0.56	Clear	S1 S2 heard	Soft	WNL	361	11.2	142	84	38
84	758506	34	M	Weakness of both lower limbs, pain and tingling of right hand	P	P	90	190	100	64	162	103	35.8	0.63	Clear	S1 S2 heard	Soft	Quadriparesis	249	8.2	159	106	33
85	758517	70	F	Burning micturition	P	P	120	130	70	65	158	92	26.4	0.58	Clear	S1 S2 heard	Lumbar tenderness	WNL	257	12.2	198	134	41
86	758624	45	F	Weakness of right upper and lower limb	P	P	80	140	90	65	154	90	23.6	0.58	Clear	S1 S2 heard	Soft	Right incoordination	208	7.6	115	65	37
87	758575	51	F	Weakness of right upper and lower limb	P	P	82	170	100	65	153	86	21.3	0.56	Clear	S1 S2 heard	Soft	Right hemiplegia	176	9.7	139	76	30
88	758785	39	M	Fever with chills	P	A	90	150	90	65	164	104	30.8	0.63	Clear	S1 S2 heard	Soft	WNL	125		214	149	36
89	758816	65	M	Loose stools	P	P	78	140	80	66	162	96	22.4	0.59	Clear	S1 S2 heard	Soft	WNL	343	8	156	109	31
90	758985	64	M	Giddiness	P	P	68	150	80	66	164	89	23	0.54	Clear	S1 S2 heard	Soft	WNL	313	9.4	152	102	34
91	759037	81	M	Weakness of right upper and lower limb	P	P	80	130	80	66	168	106	22.6	0.63	Clear	S1 S2 heard	Soft	Right hemiparesis	214	8.4	184	117	55
92	759006	66	M	Fever with chills	P	P	80	160	90	66	169	102	32.9	0.6	Clear	S1 S2 heard	Soft	WNL	127	12	288	217	48
93	759208	78	M	Seizures	P	A	86	160	80	66	163	101	27	0.61	Clear	S1 S2 heard	Soft	WNL	71		187	142	28
94	759676	65	M	Weakness of left upper and lower limb	P	P	80	100	70	66	158	90	24.8	0.56	Clear	S1 S2 heard	Soft	Left ataxic hemiparesis	74		78	131	30
95	759609	35	M	Headache	P	P	86	190	90	67	168	86	22.6	0.51	Clear	S1 S2 heard	Soft	WNL	394	12.4	170	119	23
96	759953	40	F	Chest pain	P	P	80	130	70	67	163	91	25.5	0.55	Clear	S1 S2 heard	Soft	WNL	84	5.5	202	148	52
97	759935	73	F	Weakness of right upper and lower limb	P	A	88	140	70	68	170	92	24.9	0.54	Clear	S1 S2 heard	Soft	Right dense hemiplegia	90	5.6	134	88	12
98	759909	19	M	Weakness of left upper and lower limb	A	A	102	110	70	68	168	105	32.5	0.62	Clear	S1 S2 heard	Soft	Left ataxic hemiparesis	92		164	48	14
99	760084	24	F	Fever	P	A	94	110	80	68	158	91	28.8	0.57	Clear	S1 S2 heard	Soft	WNL	90		193	123	41
100	760208	73	M	Cough with expectoration	P	A	98	160	80	68	169	103	29.4	0.6	Bilateral Ronchi	S1 S2 heard	Soft	WNL	85		117	69	39
101	760192	78	F	Vomiting, Altered sensorium	P	P	82	130	80	68	165	86	22.7	0.52	Clear	S1 S2 heard	Soft	WNL	117	9.5	178	122	40
102	760245	64	F	weakness of right upper and lower limb	P	A	80	170	90	69	166	91	22.8	0.54	Clear	S1 S2 heard	Soft	Right hemiparesis	103		238	160	48
103	760254	78	M	Slurring of speech	A	P	88	110	80	69	165	96	24.24	0.58	Clear	S1 S2 heard	Soft	Dysarthria	65		152	90	32
104	760382	66	M	Cough	P	P	80	120	70	70	168	103	22.6	0.61	Clear	S1 S2 heard	Soft	WNL	118	5.5	165	94	41
105	760963	80	F	Cough with expectoration, Fever	P	P	110	200	120	70	181	92	27.8	0.59	Right basal crepts	S1 S2 heard	Soft	WNL	153	10.4	197	112	57
106	760932	76	M	Loss of memory	P	P	80	130	70	70	161	89	22.7	0.55	Clear	S1 S2 heard	Soft	Cognitive impairment	104	9	193	21	113
107	760723	50	F	Headache	P	P	96	110	80	70	158	91	25	0.57	Clear	S1 S2 heard	Soft	WNL	186	10.4	224	152	54
108	760992	62	M	Headache	P	P	72	210	100	70	160	82	25.7	0.51	Clear	S1 S2 heard	Soft	WNL	213	8.4	96	68	17
109	761138	65	M	Seizures	P	P	88	160	100	71	165	101	26.8	0.61	Clear	S1 S2 heard	Soft	WNL	269	7.8	183	122	42
110	761139	52	M	Weakness of right upper and lower limb	P	P	80	170	100	72	168	101	29	0.6	Clear	S1 S2 heard	Soft	Right hemiparesis	252		190	113	49
111	761336	66	M	Pain and swelling over right side of face	P	P	80	120	90	72	158	80	22.4	0.5	Clear	S1 S2 heard	Soft	WNL	421	15.7	161	105	30

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								Systolic	Diastolic														
112	761434	60	F	Headache	P	P	90	200	90	72	164	90	21.9	0.54	Clear	S1 S2 heard	Soft	WNL	154	5.1	251	176	52
113	761601	72	F	Acute onset vertigo	P	P	80	140	90	72	154	82	25.2	0.53	Clear	S1 S2 heard	Soft	E2M2V2	168	7.4	176	102	49
114	761586	43	F	Seizures	P	P	80	130	80	73	156	90	27.9	0.57	Clear	S1 S2 heard	Soft	WNL	335	8.6	125	288	22
115	761631	66	M	Cough with expectoration	P	A	56	180	70	74	165	88	26.6	0.53	Right basal crepts	S1 S2 heard	Soft	WNL	82		130	78	35
116	761674	72	M	Weakness of right upper and lower limb	P	A	80	200	100	74	167	104	27.2	0.62	Clear	S1 S2 heard	Soft	WNL	96		121	71	28
117	761866	64	M	Vomiting, Altered sensorium	P	P	80	150	90	74	174	101	31	0.58	Clear	S1 S2 heard	Soft	Drowsy	388	11.7	143	75	44
118	761923	54	M	Slurring of speech	P	A	98	160	100	74	156	86	22	0.55	Clear	S1 S2 heard	Soft	Paraparesis	128	6.5	199	135	28
119	762136	44	M	Syncope	P	P	80	180	100	74	166	102	31.2	0.61	Clear	S1 S2 heard	Soft	WNL	485	10.8	261	185	55
120	761954	44	M	Headache	P	A	80	110	70	75	166	81	21.7	0.48	Clear	S1 S2 heard	Soft	WNL	92		235	158	35
121	761956	65	M	Chest pain	P	P	88	180	100	76	159	82	22.5	0.51	Clear	S1 S2 heard	Soft	WNL	166	5.7	207	133	34
122	762032	52	M	Headache	P	P	80	160	70	76	166	88	22.1	0.52	Clear	S1 S2 heard	Soft	WNL	376	12.7	111	53	32
123	762050	65	M	Headache, Weakness of right upper and lower limb	P	P	98	170	90	78	159	79	22.5	0.49	Clear	S1 S2 heard	Soft	Right hemiplegia	128	12	154	94	34
124	763791	46	M	Vertigo	P	A	110	100	60	78	172	101	26.3	0.58	Clear	S1 S2 heard	Soft	Quadriplegia	111		140	97	28
125	763755	85	F	Headache	P	P	64	110	70	78	158	90	22.4	0.56	Clear	S1 S2 heard	Soft	Drowsy	112		217	139	63
126	763931	46	M	Altered sensorium	P	P	120	130	80	78	163	105	35.3	0.64	Clear	S1 S2 heard	Soft	Irritable	341	9.3	133	71	40
127	763994	82	M	Weakness of left upper and lower limb	P	P	68	130	70	80	159	88	22.5	0.55	Clear	S1 S2 heard	Soft	Left incoordination	163	8.4	182	118	35
128	764180	54	M	Slurring of speech	P	A	98	160	100	80	154	79	22.3	0.51	Clear	S1 S2 heard	Soft	Paraparesis	128	6.5	199	135	28
129	764288	40	F	Giddiness	P	A	80	140	90	80	153	76	21.7	0.49	Clear	S1 S2 heard	Soft	Nystagmus	99		251	118	33
130	764259	55	M	Weakness of right upper and lower limb	P	P	78	110	70	80	168	101	26.2	0.6	Clear	S1 S2 heard	Soft	Right hemiparesis	147	7.8	235	175	43
131	764414	62	M	Dyspnea on exertion	P	P	80	140	90	82	164	90	27.5	0.54	Clear	S1 S2 heard	Soft	WNL	163	8.7	140	87	28
132	764413	53	M	Weakness of left upper and lower limb	P	A	80	150	90	83	171	101	25.9	0.59	Clear	S1 S2 heard	Soft	Left hemiparesis	284	7.5	96	43	39
133	764731	70	F	Slurring of speech	P	A	110	120	70	84	154	80	21.5	0.51	Clear	S1 S2 heard	Soft	Right hemiplegia	119		197	126	37
134	764751	70	M	Pain and redness over left eye	P	P	72	170	100	85	172	102	29.06	0.59	Clear	S1 S2 heard	Soft	WNL	165	11.6	227	149	59
135	764054	65	M	Weakness of right upper and lower limb	P	P	88	140	90	86	166	91	22.1	0.54	Clear	S1 S2 heard	Soft	Right hemiplegia	342	13.5	172	106	29
136	764973	81	M	Palpitations	P	P	90	130	90	86	172	102	27	0.59	Clear	S1 S2 heard	Soft	WNL	82	8.6	150	67	69
137	764787	86	F	Acute progressive weakness of all 4 limbs	P	P	80	150	90	86	158	90	29.6	0.56	Clear	S1 S2 heard	Soft	Quadriparesis	246	9.6	219	152	62
138	764947	60	M	Seizures	P	P	80	140	80	86	174	86	21.7	0.49	Clear	S1 S2 heard	Soft	Gait ataxia	158	8	163	113	23
139	764960	63	M	Giddiness	P	P	80	140	90	86	168	91	22.3	0.54	Clear	S1 S2 heard	Soft	Limb and gait ataxia	243	9.5	298	144	39
140	765185	48	M	Dyspnea on exertion	P	A	99	110	70	87	170	102	32.8	0.6	Bilateral crepts	S1 S2 heard	Soft	WNL	118		216	169	30
141	765413	36	M	Blurring of vision	P	P	88	160	90	88	166	102	26.8	0.61	Clear	S1 S2 heard	Soft	WNL	281	7.6	206	140	44
142	765571	64	M	Burning micturition	P	P	80	140	90	90	164	102	29.7	0.62	Clear	S1 S2 heard	Soft	WNL	151	8.1	119	51	49
143	765569	49	M	Headache	P	P	88	150	90	92	171	89	21.8	0.52	Clear	S1 S2 heard	Soft	WNL	351	11.2	176	117	32
144	765564	56	M	Weakness of left lower limb	P	P	80	160	70	93	168	94	22.32	0.55	Clear	S1 S2 heard	Soft	Left lower limb monoparesis	214	7.8	159	29	29
145	765889	51	M	Headache, diplopia	P	A	74	140	80	94	158	79	22.4	0.5	Clear	S1 S2 heard	Soft	Neck stiffness	117		157	118	34
146	766020	75	F	Tingling sensatin left upper and lower limb	P	P	98	180	90	94	154	91	33.7	0.53	Clear	S1 S2 heard	Soft	Left hemihypoasthesia	373	9.6	149	78	58
147	766208	55	M	Seizures	P	P	88	170	90	94	164	103	31.9	0.62	Clear	S1 S2 heard	Soft	WNL	227	8.6	199	142	41
148	766456	83	F	Involuntary movement	P	A	68	130	80	94	154	78	22.7	0.5	Clear	S1 S2 heard	Soft	WNL	88		175	107	37

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								Systolic	Diastolic						Respiratory system	Cardiovascular system	Per abdomen	Central nervous system			Total cholesterol (mg/dL)	Low density lipoprotein (mg/dL)	High density lipoprotein (mg/dL)
149	766254	65	F	Fever	P	P	84	140	90	95	165	104	28	0.63	Clear	S1 S2 heard	Soft	WNL	193	9.3	208	154	42
150	766925	62	M	Weakness of right upper and lower limb	P	P	80	160	90	102	164	102	27.8	0.62	Clear	S1 S2 heard	Soft	Right hemiparesis	114	7.8	184	119	44

ls
Triglycerides (mg/dL)
141
82
106
488
95
68
261
175
204
95
59
140
237
140
237
138
180
98
117
180
207
117
179
135
148
182
165
189
135
149
132
166
112
590
92
143
98

ds
Triglycerides (mg/dL)
146
95
209
142
87
170
108
231
295
200
174
146
133
55
197
132
261
162
282
133
86
126
192
126
87
68
299
133
175
112
39
133
50
82
31
36
56

ls
Triglycerides (mg/dL)
217
249
101
243
83
71
63
231
103
102
114
66
166
145
78
79
61
115
84
84
141
62
170
509
146
42
:130
150
151
149
110
297
92
56
97
138
132

ls
Triglycerides (mg/dL)
117
123
86
85
108
119
179
104
211
81
132
129
76
73
111
147
179
293
85
127
68
170
96
184
71
119
137
579
85
109
95
134
111
162
65
41
153

ls
Triglycerides (mg/dL)
60
105

ANNEXURE – IV – KEY TO MASTER CHART

A	–	Absent
Cm	–	Centimeter
EMV	–	Eye Opening, Motor response, Verbal response
F	–	Female
Kg	–	Kilograms
M	–	Male
Mg/dl	–	Milligrams per deciliter
Mm of Hg	–	Millimeter of mercury
P	–	Present
RBS	–	Random Blood Sugar
S1 S2	–	First and Second heart sound
WNL	–	Within Normal Limits