A STUDY ON VISCERAL ADIPOSITY INDEX IN PATIENTS WITH ACUTE CORONARY SYNDROME

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(GENERAL MEDICINE)

CHENGALPATTU MEDICAL COLLEGE & HOSPITAL

THE TAMIL NADU DR.M.G.R.MEDICAL UNIVERSITY

TAMILNADU, INDIA

APRIL 2017

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY ON VISCERAL ADIPOSITY INDEX IN PATIENTS WITH ACUTE CORONARY SYNDROME",** is a bonafide work of **Dr. K.SARANYA DEVI** in partial fulfilment of the requirements for **M.D.BRANCH-I (GENERAL MEDICINE)** examination of **THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY** to be held in April 2017. The period of study was from January 2016 to June 2016.

Signature of the H.O.D

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Date:

Place :

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DECLARATION BY THE CANDIDATE

I, **Dr. K. SARANYA DEVI**, hereby declare that this dissertation titled **"A STUDY ON VISCERAL ADIPOSITY INDEX IN PATIENTS WITH ACUTE CORONARY SYNDROME"** is a bonafide and genuine research work done by me at **CHENGALPATTU MEDICAL COLLEGE & HOSPITAL** from January 2016 – June 2016 under the direct guidance and supervision of **DR.M.ANUSUYA, M.D.,** Professor, Department of General Medicine, Chengalpattu Medical College and Hospital, Chengalpattu. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the University regulations for the award of MD degree in General Medicine Examination to be held in April 2017.

Date: Signature of the Candidate

Place: Chengelpattu **Dr. K. SARANYA DEVI**

INSTITUTIONAL ETHICAL COMMITTEE

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

Title of Work	:	A Study on Visceral Adiposity Index in patients with Acute coronary Syndrome.
Principal Investigator	:	Dr.Saranya Devi
Designation	:	Post Graduate in MD General Medicine
Co-Investigators	:	Dr.M.Anusuya,MD Associate Professor of General Medicine
Department	:	General Medicine

The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.01.2016 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 11.00 PM.

The Members of the committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

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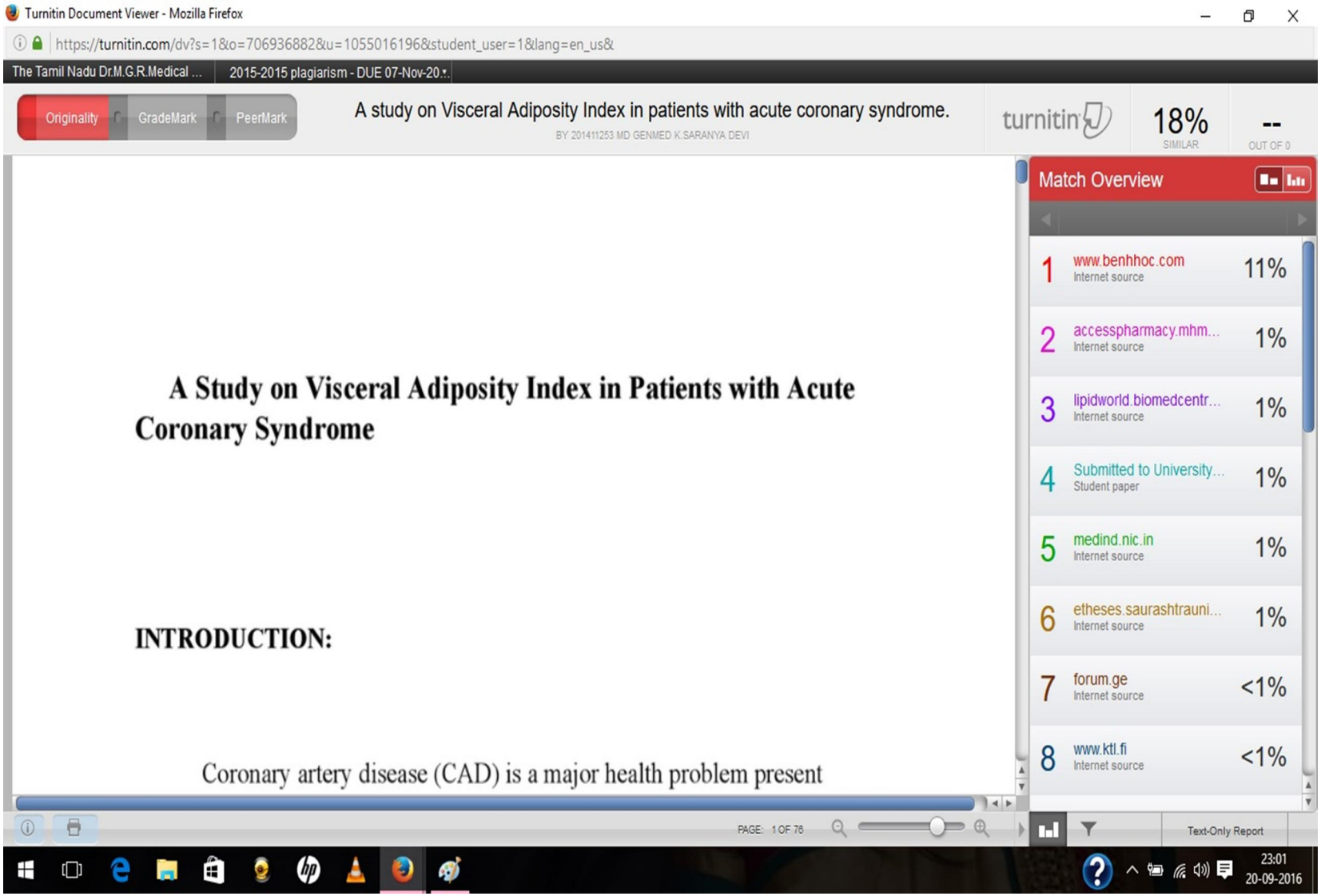
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for my study

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I bow my head in respect before God Almighty.

Date: Signature of the Candidate

Place: **Dr. K. SARANYA DEVI**

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LIST OF ABBREVIATIONS USED

%	Percentage
CAD	Coronary Artery Disease
CHD	Coronary Heart Disease
BMI	Body Mass Index
WC	Waist Circumference
TG	Triglycerides
HDL	High Density Lipoprotein
VAI	Visceral Adiposity Index
WHR	Waist Hip Ratio
NAFLD	Non Alcoholic Fatty Liver Disease
NASH	Non Alcoholic Steato Hepatitis
PCOD	Polycystic Ovarian Disease
WHO	World Health Organisation
DM	Diabetes
SHT	Systemic Hypertension
WT	Weight
HT	Height
MRI	Magnetic Resonance Imaging
CT	Computed Tomography
STEMI	ST segment elevation myocardial infarction
NSTEMI	Non ST segment elevation MI.

INTRODUCTION

Coronary heart disease (CHD) is a health epidemic present worldwide. Results of mortality data from Global Burden of Diseases Studies states that CVD especially CAD are important cause of death in India.

Major causes of death around the world: cardiovascular disease, Cancer, Chronic respiratory diseases, HIV/AIDS, TB, Diabetes.

Worldwide 30 % of all deaths are attributed to CVD, out of which more than half are caused by CHD. It is expected that in future the number will grow due to lifestyle changes in developing countries. Of those dying from CVD, 80% are in developing countries.

Myocardial infarction in India:

The incidence of MI in India is steadily increasing over the past few decades. Rural to urban ratio in rise in MI cases is around 2:9. This could probably be due to migration from rural to urban cities. Another major problem is the occurrence of MI at a younger age in the Indian population.

STEMI is an emergency due to acute total occlusion of an epicardial coronary artery, most often due to atherosclerotic plaque rupture / erosion and subsequent thrombus formation. Compared to UA /

NSTEMI, STEMI is associated with a higher in -hospital and 30 day morbidity and mortality. If untreated, the mortality rate of STEMI can exceed 30 % and the presence of mechanical complications increases the mortality rate to 90%.

Obesity is an important risk factor for cardiovascular disease and is an increasing pandemic across the globe. Although BMI has traditionally been the standard index to classify and define obesity, nowadays importance has been given to other indices of adiposity such as waist circumference, waist hip ratio, waist height ratio, atherogenic index, body adiposity index and visceral adiposity index.

- Measurement of the degree of visceral fat has become an important determinant of the degree of cardiovascular risk.
- Visceral adiposity is closely related to CVD via increased adipokine production, proinflammatory activity and the deterioration of insulin sensitivity.
- A study concluded that association between visceral fat and BMI was only minimal and visceral adiposity was able to stratify cardiovascular risk better than BMI.
- The gold standard assessment method for the estimation of visceral fat content has been Magnetic resonance imaging and Computerized tomography.

- These methods are not used due to the cost and time involved in their utilization.

- Amato et al. have recently individualized a novel sex-specific index based on waist circumference, body mass index (BMI), triglycerides (TGs), high-density lipoprotein cholesterol (HDL-C), and indirectly expressing visceral fat and termed it the visceral adiposity index (VAI).

- VAI had significant correlation with visceral adiposity and its increase was strongly associated with cardiometabolic risk.

- Studies have used the visceral adiposity index using the formula in different populations such as women with PCOS, patients with diabetes and general population without metabolic syndrome. However there are limited studies that have used visceral adiposity index in patients with myocardial infarction.

Unger and Scherer proposed defining hypoleptinemia as a state of visceral adiposity and ‘‘high TG/low HDL cholesterol dyslipidemia,’’ which were not observed in generalized obesity. Hence, a factor like VAI needs to be evaluated in delineating visceral adiposity and thus more precisely cardiometabolic risk.

AIM AND OBJECTIVES

- To calculate the VAI in patients with acute coronary syndrome.
- To study the factors influencing high VAI among patients with acute coronary syndrome

REVIEW OF LITERATURE

INTRODUCTION

IHD causes more deaths and disability and incurs greater Economic costs than any other illness in the world. Genetic factors, a high-fat and energy-rich diet, smoking and a sedentary lifestyle are associated with the emergence of IHD. IHD is growing among low-income groups, but primary prevention has delayed the disease to later in life in all socioeconomic groups. But epidemiologic data show a decline in the rate of deaths due to IHD, about half of which is attributable to treatments and half to prevention by risk factor modification.

Powerful risk factors for IHD like Obesity, insulin resistance, and type 2 diabetes mellitus are increasing. These trends are occurring due to population growth and as a result of the increase in the average age of the world's population. With urbanization in countries with emerging economies and a growing middle class, elements of energy rich Western diet are being adopted.

As a result, the prevalence of risk factors for IHD and the prevalence of IHD itself are both increasing rapidly, so that in Analyses of the global burden of disease, there is a shift from communicable to non-communicable diseases. Population subgroups that appear to be particularly affected are men in South Asian countries, especially India

and the Middle East. IHD is likely to become the most common cause of death worldwide by 2020.

Coronary heart disease in India

India is going through an epidemiologic transition whereby the burden of communicable diseases have declined slowly, but that of non-communicable diseases (NCD) has risen rapidly, thus leading to a dual burden. There has been a 4-fold rise of CHD prevalence in India during the past 40 years.Current estimates from epidemiologic studies from various parts of the country indicate a prevalence of CHD to be between 7% and 13% in urban and 2% and 7% in rural populations . Epidemiologic studies have shown that there are at present over 30 million cases of CHD in India. The Global Burden of Diseases Study reported that the disability-adjusted life years lost by CHD in India during 1990 was 5.6 million in men and 4.5 million in women; the projected figures for 2020 were 14.4 million and 7.7 million in men and women respectively. There are no prospective studies in India that have determined factors of risk for CHD. Risk factors for premature CHD have been quantified in the case control INTERHEART study. In the INTERHEART study, 8 common risk factors explained >90% of incident acute myocardial infarctions in South Asian and Indians. Risk factors cited in interheart study;

- Dyslipidemia (high apolipoprotein B/apolipoprotein A1 ratio),
- Smoking or tobacco use,
- Known hypertension,
- Known diabetes,
- Abdominal obesity,
- Physical inactivity,
- Low fruits and vegetables intake and
- Psychosocial stress.

An important change in risk factor dynamics in India is a more rapid increase in CVD risk factors in rural and slum populations compared with urban populations. Smoking and non-smoked tobacco continues to increase in rural and less literate populations, while it is declining in more educated urban populations.

The epidemic of sedentariness has penetrated rural households with rapidly increasing use of labour-saving technologies. Dietary habits have undergone a change with greater consumption of fats, saturated fats, trans fats, and processed foods. Calorie-dense fast foods (comfort foods) are easily available and both Indian-style and Western-style fast foods are being consumed widely.

Increasing abdominal obesity and other cardiometabolic risk factors among the rural populations in India. This Further escalates the CHD epidemic in India.

Other Risk Factors

A number of case-control studies have reported that abnormalities of lipids other than low-density lipoprotein cholesterol may be important in Indians. These lipoprotein lipids include low high-density lipoprotein cholesterol and high triglycerides, very-low-density lipoprotein cholesterol metabolites, lipoprotein remnants, and lipoprotein.

MODIFIABLE AND NONMODIFIABLE RISK FACTORS FOR CAD

- High LDL cholesterol
- Cigarette smoking
- Hypertension (BP ≥140/90 mmHg or on antihypertensive medication)
- Low HDL cholesterol (<1.0 mmol/L <40 mg/dL)
- Diabetes mellitus
- Family history of premature CHD
- Age (men ≥45 years; women ≥55 years)

Lifestyle risk factors

- Obesity (BMI ≥30 kg/m2)
- Physical inactivity

Emerging risk factors

- Lipoprotein (a)
- Prothrombotic factors
- Proinflammatory factors
- Impaired fasting glucose
- Subclinical atherosclerosis

(HDL cholesterol ≥1.6 mmol/L (≥60 mg/dL) has been viewed as a "negative" risk factor).

CARDINAL FEATURES OF CORONARY ARTERY DISEASE

AMONG INDIANS COMPARED TO OTHER POPULATIONS

- Higher rates 2 to 4 fold higher prevalence, incidence, hospitalization and mortality.
- Greater prematurity

- 5 to 10 year earlier onset of first Myocardial infarction (Ml)
- 5 to 10 fold higher rate of Ml and death in young «40 years of age)

• Greater severity

- Three vessel disease common even among young premenopausal Women.
- Large Ml with greater muscle damage

• Higher prevalence of glucose intolerance- Insulin resistance syndrome, diabetes, central obesity.

• Lower prevalence of conventional risk factors

- Hypertension, cigarette smoking

• Higher prevalence of emerging (thrombogenic) risk factors

- High levels of lipoprotein a, homocysteine, Apo protein B
- High levels of triglycerides, fibrinogen
- Low levels of HDL
- Small dense LDL

- Higher rates of clinical events for a given degree of atherosclerosis
    - Double that of Whites
    - 4 fold higher than Chinese
    - Higher proportion of unstable or vulnerable plaques.

OBESITY AND CORONARY ARTERY DISEASE

Obesity is a state of excess adipose tissue mass. Although often viewed as equivalent to increased body weight, this need not be the case, muscular individuals may be overweight by numerical standards without having increased adiposity. Body weights are distributed continuously in populations, so that choice of a medically meaningful distinction between lean and obese is somewhat arbitrary. Obesity is therefore defined by assessing its linkage to morbidity or mortality.

Although not a direct measure of adiposity, the most widely used method to gauge obesity is the body mass index (BMI), which is equal to weight/height2 (in kg/m2). Other approaches to quantifying obesity include;

- visceral adiposity index
- waist hip ratio
- waist circumference

- anthropometry (skinfold thickness),
- densitometry (underwater weighing),
- computed tomography (CT) or
- magnetic resonance imaging (MRI), and
- Electrical impedance.

The distribution of adipose tissue in different anatomic depots also has substantial implications. Specifically, intraabdominal and abdomen subcutaneous fat have more significance than subcutaneous fat present in the buttocks and lower extremities. This distinction is most easily made clinically by determining the waist-to -hip ratio, with a ratio >0.9 in women and >1.0 in men being abnormal.

The mechanism underlying this association is unknown but may relate to the fact that intraabdominal adipocytes are more lipolytically active than those from other depots. Release of free fatty acids into the portal circulation has adverse metabolic actions, especially on the liver. Adipokines and cytokines that are differentially secreted by adipocyte depots may play a role in the systemic complications of obesity.

PHYSIOLOGIC REGULATION OF ENERGY BALANCE

Body weight is regulated by both endocrine and neural components that ultimately influence the effector arms of energy intake

and expenditure. This complex regulatory system is necessary because even small imbalances between energy intake & expenditure will ultimately have large effects on body weight. This regulation of energy balance cannot be monitored easily by calorie-counting in relation to physical activity. Rather, regulation or dysregulation depends on a complex interplay of hormonal and neural signals.

Alterations in stable weight by forced overfeeding or food deprivation induce physiologic changes that resist these perturbations:

- With weight loss, appetite increases and energy expenditure falls;
- With over feeding, appetite falls and energy expenditure increases.

This latter compensatory mechanism frequently fails, however, permitting obesity to develop when food is abundant and physical activity is limited.

A major regulator of these adaptive responses is the adipocyte-derived hormone leptin, which acts through brain circuits predominantly in the hypothalamus to influence appetite, energy expenditure, and neuroendocrine function. Appetite is influenced by many factors that are integrated by the brain, most importantly within the hypothalamus.

Signals that impinge on the hypothalamic centre include;

1. Neural afferents,
2. Hormones, and
3. Metabolites.

Neural – vagal inputs are particularly important, bringing information from viscera, such as gut distention.

Hormonal signals - leptin, insulin, cortisol, and gut peptides. Among the latter is ghrelin, which is made in the stomach and stimulates feeding, peptide YY (PYY) and cholecystokinin, which is made in the small intestine and signals to the brain through direct action on hypothalamic control centres and or via the vagus.

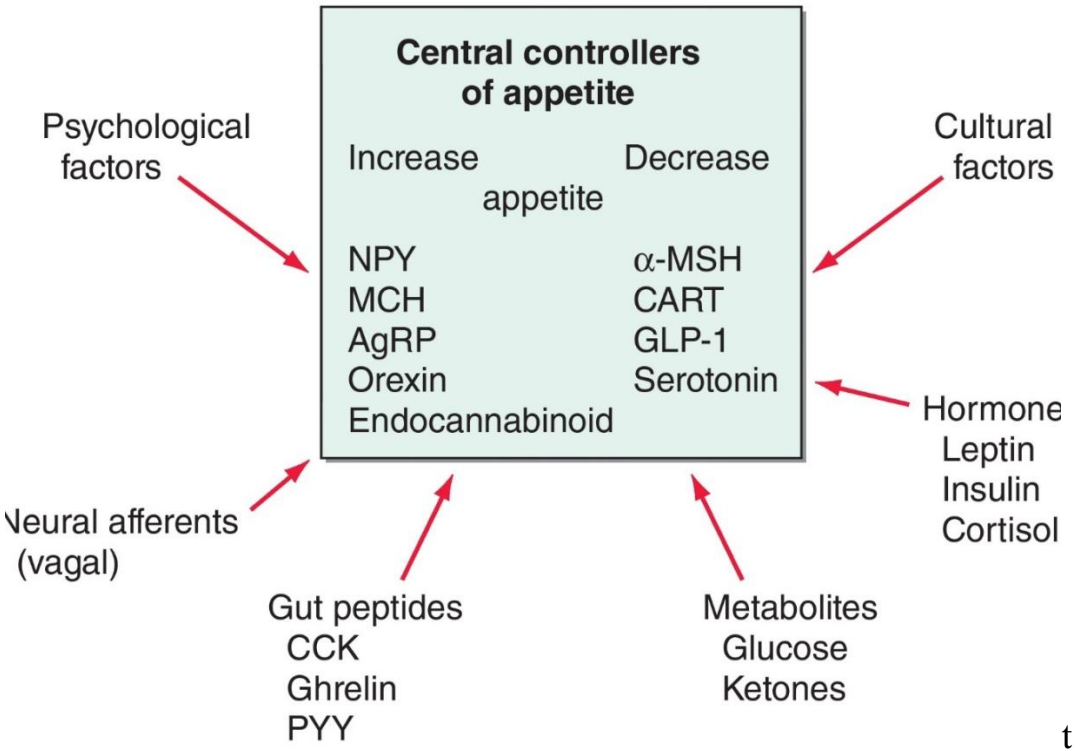
Metabolites – glucose can influence appetite, as seen by the effect of hypoglycemia to induce hunger; however, glucose is not normally a major regulator of appetite.

These diverse signals act by influencing the expression and release of various hypothalamic peptides (e.g., neuropeptide Y NPY, Agoutirelated peptide AgRP, α-melanocyte-stimulating hormone α MSH, and melanin-concentrating hormone MCH) that are integrated with serotonergic, catecholaminergic, endocannabinoid, and opioid signalling pathways.

Energy expenditure includes the following components:

- resting or basal metabolic rate;
- the energy cost of metabolizing and storing food;
- the thermic effect of exercise.

Adaptive thermogenesis occurs in brown adipose tissue (BAT). In contrast to white adipose tissue, which is used to store energy in the form of lipids, BAT expends stored energy as heat. A mitochondrial uncoupling protein (UCP-1) in BAT dissipates the hydrogen ion gradient in the oxidative respiration chain and releases energy as heat. The metabolic activity of BAT is increased by a central action of leptin, acting via sympathetic nervous system that heavily innervates this tissue. In rodents, BAT deficiency causes obesity and diabetes; stimulation of BAT with a specific adrenergic agonist (β3 agonist) protects against diabetes and obesity. BAT exists in humans (especially neonates), and although its physiologic role is not yet established, identification of functional BAT in many adults using positron emission tomography(PET) imaging has increased interest in the implications of the tissue for pathogenesis and therapy of obesity. Beige fat cells, recently described, resemble BAT cells in expressing UCP-1. They are scattered through white adipose tissue, and their thermogenic potential is uncertain.



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THE ADIPOCYTE AND ADIPOSE TISSUE

Adipose tissue is composed of the lipid-storing adipose cell & stromal/vascular compartment in which cells including preadipocytes and macrophages reside. Adipose mass increases by enlargement of adipose cells through lipid deposition, as well as by an increase in the number of adipocytes.

Obese adipose tissue is also characterized by increased numbers of infiltrating macrophages. The process by which adipose cells are derived from a mesenchymal preadipocyte involves an series of differentiation steps mediated specific transcription factors. One of the key transcription factors is *peroxisome proliferator -activated receptor γ* (PPARγ), a

nuclear receptor that binds the thiazolidinedione class of insulin-sensitizing drugs used in the treatment of type 2 diabetes.

It is also an endocrine cell that releases leptin, cytokines such as tumor necrosis factor(TNF)-α and interleukin (IL)-6, complement factors such as factorD (also known as *adipsin*), prothrombotic agents such as plasminogen activator inhibitor I, and a component of the blood pressure–regulating system, angiotensinogen.

Adiponectin, an abundant adipose- derived protein whose levels are reduced in obesity, enhances insulin sensitivity and lipid oxidation and has vascular-protective effects, whereas resistin and RBP4, whose levels are increased in obesity, may induce insulin resistance. These factors, and others not yet identified, play a role in the physiology of lipid homeostasis, insulin sensitivity, blood pressure control, coagulation, and vascular health, and are likely to contribute to obesity-related pathologies.

ETIOLOGY OF OBESITY

Molecular pathways regulating energy balance are the causes of obesity remain elusive. This reflects the fact that obesity is a heterogeneous group of disorders. At one level, the pathophysiology of obesity seems simple: a chronic excess of nutrient intake relative to the level of energy expenditure. However, due to the complexity of the

neuroendocrine and metabolic systems that regulate energy intake, storage, and expenditure, it has been difficult to quantitate all the relevant parameters (e.g., food intake and energy expenditure) over time in human subjects.

Specific Genetic Syndromes

Distinct mutations cause both hyperphagia and diminished energy expenditure, suggesting a physiologic link between these two parameters of energy homeostasis.

Identification of the *ob* gene mutation in genetically obese (ob/ob) mice represented a major breakthrough in the field. The ob/ob mouse develops severe obesity, insulin resistance, and hyperphagia, as well as efficient metabolism (e.g., it gets fat even when ingesting the same number of calories as lean litter mates). The product of the *ob* gene is the peptide leptin, a name derived from the Greek root *leptos*, meaning thin. Leptin is secreted by adipose cells and acts primarily through the hypothalamus. Its level of production provides an index of adipose energy stores. High leptin levels decrease food intake and increase energy expenditure. Another mouse mutant, db/db, which is resistant to leptin, has a mutation in the leptin receptor and develops a similar syndrome. The *ob* gene is present in humans where it is also expressed in fat.

- Mutations in the gene encoding proopiomelanocortin (POMC) cause severe obesity through failure to synthesize α-MSH, a key neuropeptide that inhibits appetite in the hypothalamus.

- Proenzyme convertase 1 (PC-1) mutations cause obesity by preventing synthesis of α-MSH from its precursor peptide, POMC.

- α-MSH bind s to the type 4 melanocortin receptor (MC4R), a key hypothalamicreceptor that inhibits eating. Heterozygous loss-of-function mutations of this receptor cause obesity.

- Loss of function of MRAP2, a protein required for normal MC4R signaling, has been found in rare cases of severe obesity.

- These six genetic defects define a pathway through which leptin (by stimulating POMC and increasing α-MSH) restricts food intake and limits weight.

The fat gene encodes carboxypeptidase E, a peptide-processing enzyme; mutation of this gene is thought to cause obesity by disrupting production of one or more neuropeptides. AgRP is coexpressed with NPY in arcuate nucleus neurons. AgRP antagonizes α-MSH action at MC4 receptors, and its overexpression induces obesity.

SYNDROMES ASSOCIATED WITH OBESITY(CONGENITAL)

- Prader-Willi syndrome
- Bardet-Biedl syndrome(BBS)

METABOLIC SYNDROME

The metabolic syndrome (syndrome X, insulin resistance syndrome) consists of a constellation of metabolic abnormalities that confer increased risk of cardiovascular disease (CVD) and diabetes mellitus. Evolution of the criteria for the metabolic syndrome since the original definition by the World Health Organization in 1998 reflects growing clinical evidence and analysis by a variety of consensus conferences and professional organizations. The major features of the metabolic syndrome include central obesity, hypertriglyceridemia, low levels of high-density lipoprotein (HDL) cholesterol, hyperglycemia, and hypertension.

NCEP: ATPIII (*a)* 2001 and Harmonizing Definition Criteria for the Metabolic Syndrome.

NCEP: ATPIII 2001

Three or more of the following:

- Central obesity: waist circumference >102 cm (M), >88 cm(F)

- Hypertriglyceridemia: triglyceride level ≥150 mg/dL or specific medication
- Low HDL cholesterol: <40 mg/dL and <50 mg/dL for men and women, respectively, or specific medication
- Hypertension: blood pressure ≥130 mmHg systolic or ≥85 mmHg diastolic or specific medication.
- Fasting plasma glucose level ≥100 mg/dL or specific medication or previously diagnosed type 2 diabetes.

Harmonizing Definition

Three of the following:

• Waist circumference (cm)

Men	Women	Ethnicity
≥94	≥80	Europid, sub-Saharan African, Eastern.
≥90	≥80	South Asian, Chinese, South and Central America.
≥85	≥90	Japanese

- Fasting triglyceride level >150 mg/dL or specific medication.
- HDL cholesterol level <40 mg/dL and <50 mg/dL for men and women, respectively, or specific medication.

- Blood pressure >130 mm systolic or >85 mm diastolic or previous diagnosis or specific medication.
- Fasting plasma glucose level ≥100 mg/dL (alternative indication: drug treatment of elevated glucose levels).
- *a* - National Cholesterol Education Program and Adult Treatment Panel III.

PATHOLOGIC CONSEQUENCES OF OBESITY

Obesity has major adverse effects on health. Obesity is associated with an increase in mortality , with a 50–100% increased risk of death from all causes compared to normal -weight individuals , mostly due to cardiovascular causes.

Obesity and overweight together are the second leading cause of preventable death in the United States, accounting for 300,000 deaths per year. Mortality rates rise as obesity increases, particularly when obesity is associated with increased intraabdominal fat.

Life expectancy of a moderately obese individual could be shortened by 2–5 years, and a 20- to 30-year-old male with a BMI >45 may lose 13 years of life. It is likely that the degree to which obesity affects particular organ systems is influenced by susceptibility genes that vary in the population. Insulin Resistance and Type 2 Diabetes Mellitus Hyperinsulinemia and insulin resistance are pervasive features of

obesity, increasing with weight gain and diminishing with weight loss. Insulin resistance is more strongly linked to intraabdominal fat than to fat in other depots .

Molecular links between obesity and insulin resistance in fat, muscle, and liver have been sought for many years. Major factors include:

- insulin itself, by inducing receptor downregulation;
- free fatty acids that are increased and capable of impairing insulin action;
- intracellular lipid accumulation; and
- Several circulating peptides produced by adipocytes, including the cytokines TNF-α and IL-6, RBP4, and the "adipokines" adiponectin and resistin, which have altered expression in obese adipocytes and can modify insulin action.
- Additional mechanisms are obesity-linked inflammation, includinginfiltration of macrophages into tissues including fat, and induction of the endoplasmic reticulum stress response, which can bring about resistance to insulin action in cells.
- Despite the prevalence of insulin resistance, most obese individuals do not develop diabetes, suggesting that diabetes

requires an interaction between obesity-induced insulin resistance and other factor s such as impaired insulin secretion. Obesity, however, is a major risk factor for diabetes, and as many as 80% of patients with type 2 diabetes mellitus are obese.Weight loss and exercise , even of modest degree, increase insulin sensitivity and often improve glucose control in diabetes.

- Reproductive Disorders Disorders that affect the reproductive axis are associated with obesity in both men and women. Male hypogonadism is associated with increased adipose tissue, often distributed in pattern more typical of females. In men whose weight is >160% ideal body weight (IBW), plasma testosterone and sex hormone–binding globulin (SHBG) are often reduced, and estrogen levels (derived from conversion of adrenal androgens in 3 adipose tissue) are increased. Gynecomastia may be seen. However, masculinization, libido, potency, and spermatogenesis are preserved in most of these individuals. Free testosterone may be decreased in morbidly obese men whose weight is >200% IBW.

Obesity has long been associated with menstrual abnormalities in women, particularly in women with upper body obesity. Common findings are increased androgen production, decreased SHBG, and increased peripheral conversion of androgen to estrogen. Most obese

women with oligomenorrhea have polycystic ovarian syndrome (PCOS), with its associated anovulation and ovarian hyperandrogenism; 40% of women with PCOS are obese. Most nonobese women with PCOS are also insulin-resistant, suggesting that insulin resistance, hyperinsulinemia, or the combination of the two are causative or contribute to the ovarian pathophysiology in PCOS in both obese and lean individuals. Increasing evidence supports a role for adipokines in mediating a link between obesity and the reproductive dysfunction of PCOS. In obese women with PCOS, weight loss or treatment with insulin-sensitizing drugs often restores normal menses. The increased conversion of androstenedione to estrogen, which occurs to a greater degree in women with lower body obesity, may contribute to the creased incidence of uterine cancer in postmenopausal women with obesity.

Cardiovascular Disease the Framingham Study revealed that obesity was an independent risk factor for the 26-year incidence of cardiovascular disease in men and women (including coronary disease, stroke and congestive heart failure). The waist-to-hip ratio may be the best predictor of these risks. When the additional effects of hypertension and glucose intolerance associated with obesity are included, the adverse impact of obesity is even more evident. The effect of obesity on cardiovascular mortality in women may be seen at BMIs as low as 25. Obesity, especially abdominal obesity, is associated with an atherogenic lipid profile; with increased low-density lipoprotein cholesterol, very-

low-density lipoprotein, and triglyceride; and with decreased high-density lipoprotein cholesterol and decreased levels of the vascular protective adipokine adiponectin.

Obesity is also associated with hypertension. Measurement of blood pressure in the obese requires use of a larger cuff size to avoid artefactual increases. Obesity-induced hypertension is associated with increased peripheral resistance and cardiac output, increased sympathetic nervous system tone, increased salt sensitivity, and insulin-mediated salt retention; it is often responsive to modest weight loss.

Pulmonary Disease Obesity may be associated with a number of pulmonary abnormalities. These include reduced chest wall compliance, increased work of breathing, increased minute ventilation due to increased metabolic rate, and decreased functional residual capacity and expiratory reserve volume. Severe obesity may be associated with obstructive sleep apnea and the “obesity hypoventilation syndrome” with attenuated hypoxic and hypercapnic ventilatory responses. Sleep apnea can be obstructive (most common), central, or mixed and is associated with hypertension.

Weight loss (10–20 kg) can bring substantial improvement, as can major weight loss following gastric bypass or restrictive surgery. Continuous positive airway pressure has been used with some success.

Hepatobiliary Disease Obesity is frequently associated with non-alcoholic fatty liver disease (NAFLD), and this association represents one of the most common causes of liver disease in industrialized countries. The hepatic fatty infiltration of NAFLD progresses in a subset to inflammatory nonalcoholic steatohepatitis (NASH) and more rarely to cirrhosis and hepatocellular carcinoma. Steatosis typically improves following weight loss, secondary to diet or bariatric surgery. The mechanism for the association remains unclear.

Obesity is associated with enhanced biliary secretion of cholesterol, supersaturation of bile, and a higher incidence of gallstones, particularly cholesterol gallstones. A person 50% above IBW has about a six fold increased incidence of symptomatic gallstones. Paradoxically, fasting increases supersaturation of bile by decreasing the hospholipid component.Fasting- induced cholecystitis is a complication of extreme diets.

Cancer Obesity is associated with increased risk of several cancer Types and in addition can lead to poorer treatment outcomes and increased cancer mortality. Obesity in males is associated with higher mortality from cancer of the esophagus, colon, rectum, pancreas, liver,and prostate; obesity in female s is associated with higher mortality from cancer of the gallbladder, bile ducts, breasts, endometrium, cervix and ovaries. Some of the latter may be due to increased rates of

conversion of androstenedione to estrone in adipose tissue of obese individuals. Other possible mechanistic links may involve hormones, growth factors, and cytokines whose levels are linked to nutritional state, including insulin, leptin, adiponectin, and IGF-I, as well as activation of signaling pathways linked to both obesity and cancer.

Bone, Joint, and Cutaneous Disease Obesity is associated with an increased risk of osteoarthritis, no doubt partly due to the trauma of added weight bearing, but potentially linked as well to activation of inflammatory pathways that could promote synovial pathology. The prevalence of gout may also be increased.

One of the skin problems associated with obesity is acanthosis nigricans, manifested by darkening and thickening of the skinfolds on the neck, elbows and dorsal interphalangeal spaces. Acanthosis reflects the severity of underlying insulin resistance and diminishes with weight loss.Friability of skin may be ncreased, especially in skinfolds, enhancing the risk of fungal and yeast infections. Finally, venous stasis is increased in the obese.

CLINICAL AND LABORATORY ASSESSMENT OF OBESE

Methods to assess body composition are numerous and also difficult to organize systematically. The most accurate methods in assessing body fat mass include underwater body density measurement,

body fat content estimation by dual energy X-ray absorptiometer (DEXA), magnetic resonance imaging (MRI), and computerized tomography (CT). These methods are time-consuming and require expensive equipment and thus are not feasible for large epidemiological studies. In epidemiological studies overweight, overall obesity, and abdominal obesity are typically measured by using ratios of body weight and height or body circumferences, such as Body Mass Index (BMI), waist circumference, and Waist Hip Ratio (WHR). In epidemiological studies overweight, overall obesity, and abdominal obesity are typically measured by using ratios of body weight and height or body circumferences, such as BMI, waist circumference, and WHR. BMI, WHR, and waist circumference are continuous variables and when used to define overweight or obesity, their cut-off points are arbitrary.

BODY MASS INDEX (BMI)

The BMI of a person is the number obtained by dividing his weight (in Kilograms) by the square of his height (in meters):

$$BMI = \frac{\text{(Weight in Kilograms)}}{\text{(Height in Meters)}2}$$

BMI is highly correlated with body weight and is a surrogate measure of total body fat content but is also affected by muscle

mass.World Health Organization (WHO, 2000, p. 8-9) and by the Expert Panel on theIdentification, Evaluation, and Treatment of Overweight and Obesity in Adults (1998) are based on an increased risk of morbidity and mortality in different populations.

BMI <18.5 kg/m2 is defined as underweight

18.5-24.9 kg/m2 as normal weight

25.0-29.9 kg/m2 as overweight

And > 30 kg/m2 is considered as obesity.

Obesity can be further stratified into

- Moderate obesity (BMI 30-34.9 kg/m2)
- Severe obesity (35-39.9 kgm2)
- Very severe obesity (≥40 kg/m2)

In adult Asians, a person is considered underweight if his BMI is <18.5 kg/m²; he is considered normal if his BMI is 18.5 to 22.9 kg/m²; he is considered overweight if his BMI is > 23 kg/m²; he is considered obese if his BMI is > 25 kg/m².

WAIST CIRCUMFERENCE (WC) *

Adipose tissue (fat) distributed centrally within the abdomen and among the viscera is also a predictor of cardiovascular, Cerebrovascular, and metabolic (diabetes & hyperlipidemia) diseases. The cut-off points of waist circumference and WHR are sex and population-specific. The World Health Organization has recommended the use of a cutoff point for waist circumference of 88 cm in women and 102 cm in menand for WHR of 0.85 in women and 1.0 in men to define an increased health risk (World Health Organization 2000, p. 9-11). The same cut-off points for waist circumference have been recommended by the Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults (1998) and the National Cholesterol Education Program (Ford et al. 2002).

Waist circumference appears particularly useful in the clinical setting, where both BMI and waist girth can be easily measured and followed in time (Despres et al. 2001). Waist circumference, however, is to some extent correlated with body height, and thus tall persons may falsely be categorized into the abdominally obese group. In Asian Indians Cut off values for Waist circumference were 85 and 80 cm for men and women.

Measure the waist circumference as recommended by the WorldHealth Organization:

1. The subject should be lightly dressed; measurement should not be made through thick or bulky clothing.

2. Position the subject upright with feet 25 – 30 cm apart and weight evenly distributed.

3. Sit yourself by the subject comfortable on a chair.

4. Fit a tape measure snugly around the abdominal girth without compressing soft tissue and measure the waist circumference to the nearest 0.1 cm in a horizontal plane midway between the inferior costal margin and the iliac crest.

WAIST-HIP RATIO (WHR)

The Waist-Hip Ratio of a person is obtained by dividing his "Waist circumference" by his "Hip circumference":

$$WHR = \frac{\text{(Waist Circumference in cm)}}{\text{(Hip circumference in cm)}}$$

Hip circumference can be measured around the pelvis at the point of maximal protrusion of the buttocks.

A WHR > 1.0 in European men or 0.85 in European women is associated with increased risk of ischemic heart disease, stroke, and diabetes. In Asian Indians Cutoff values for WHRs were 0.88 and 0.81,respectively for males and females respectively ((Journal of Association of Physicians of India 2007, vol. 55).

WHR is considered to be a ratio between fat stored centrally Inside the abdomen (waist circumference) and fat stored peripherally (hip circumference). Waist girth and WHR are better measures of Intraabdominal fat and probably also of total fat than BMI when Validated against computer tomography or magnetic resonance imaging. Although the current recommendation seems to favor the use of waist circumference in assessing abdominal obesity, WHR remains Suitable method for research purposes (World Health Organization \ 2000,p.10).

WHR has been criticized due to its inability to classify obesity In follow-up studies, particularly in women, if the subjects gain weight in the waist and hip areas simultaneously (Despres et al. 2001). Furthermore, it is difficult to interpret whether a large WHR is attributable to a large waist girth or to narrow hips. Previous studies suggest that both narrow waist and large hips may protect against CVD and for this reason, it is recommended that waist and hip girths should be measured (Lissner et al. 2001; Seidell et al. 2001b).

Both BMI and WHR are confounded by sex, age, and ethnic background. Also the distribution of overweight and obesity is different across populations (World Health Organization 2000).In general, men tend to have higher amounts of visceral adipose tissue than women, particularly pre- menopausal women (Lemieux et al.1993), older persons have larger waist circumferences than younger ones (Molarius and Seidell 1998), and morbidity risks at the same level of overweight vary across different ethnic populations (Seidell et al.2001a). Therefore, instead of globally accepted cut-off points for obesity, there need to be age, sex, and race-specific categories for overweight and obesity.

It is important to understand that BMI, waist circumference, and WHR estimate fat mass at different locations, reflect different etiological perspectives, and thus do not assess identical phenomena.

SKIN FOLD MEASUREMENT

An increase in weight in relation to height does not always Mean fatness or obesity if the increase in weight is due to muscle mass as in body- builders. Degree of fatness (adiposity) correlates better with the increased risks of diseases. Skin fold thickness is often used to measure this adiposity.

Measurement of skin fold thickness requires special calipers. Various areas of the body have been suggested as suitable for measuring

skin fold thickness, including back of the arm over the triceps, below the scapulae at the back, and at the supra-iliac region of the anterior abdominal wall.

Advantages

- Easy to use once skill has been mastered
- Does not require much time
- Non-invasive method

Disadvantages

- Technical sources of error
- Mostly concerned with subcutaneous fat (under the skin)
- May not be an ideal measurement for those who are obese and very lean

BIOELECTRICAL IMPEDANCE

Bioelectrical impedance measures the body's electrical impedance (resistance) to a small electric current. This impedance measurement can be analyzed to provide an estimate of the adiposity (amount of fat tissue in the body) of a person.

It is recommended the following guidelines be followed :

• Abstain from eating and drinking within 4 hours of the test

• Avoid exercising within 12 hours of the test

• Void (urinate) completely prior to testing

• Do not drink alcohol within 48 hours of the test

• Avoid taking diuretics prior to testing

However, accuracy is dependant upon several client-based variables. Wrong interpretation of adiposity through measurement of bioelectrical impedance can result due to the presence of fluid in the limbs (edema), fluid in the abdominal cavity (ascites), a full urinary bladder, or excessive sweating.

Advantages

• Requires little or no technical knowledge of the operator or the client

• Testing itself takes less than a minute

• The unit can be easily transported from place to place

• Requires only an electrical outlet and the machine itself

Disadvantages

- This method has a higher standard error range than most people desire.
- Tends to consistently overestimate lean people and underestimate obese people.
- The accuracy BIA does have is very dependant on multiple variables which may be hard to control for some people. There is doubt whether bioelectrical impedance measurement offers any advantage over BMI, WC, or WHR measurement.
- Its need of specialized equipment is another disadvantage.

DUAL ENERGY X-RAY ABSORPTIOMETRY (DEXA)

It is based on the three component model of body composition. DEXA uses two X-ray energies to measure body fat, muscle, and bone mineral. When having the scan done, one must lay still in the supine position on what looks like an x-ray table. It takes approximately twelve minutes for the computer software to reduce an image of the tissues. The results may be viewed as whole body estimates of body fat, muscle, and bone mineral as well as regional body estimates.

Advantages

• Radiation exposure is low

• DEXA is quick

• One does not have to wear a bathing suit or skimpy clothing

• There is no special preparation on the part of the participant

Disadvantages

- DEXA is costly.
- DEXA takes bone mineral content into consideration when estimating body fat and muscle; therefore it is considered to be more accurate and valid than a two component model of body composition such as underwater weighing.

MAGNETIC RESONANCE IMAGING:-

Cross-sectional abdominal images obtained with MRI, allow for separation of the subcutaneous adipose tissue (SAT) from the visceral adipose tissue (VAT) compartment. Several studies have examined the relationship between the VAT and SAT compartments for adults and children. Equations have been developed for the calculation of the VAT or SAT area based on anthropometric measurements, e.g., waist circumference or skinfolds. Although these equations may predict the

average VAT for a population, they have limited accuracy for the individual. A significant advantage of MRI for body composition studies is its potential for monitoring changes in the VAT and SAT compartments separately, information which is presently not available with any alternate in vivo techniques except for CT. The MRI-derived estimates for abdominal SAT and VAT will serve as a reference measure of adiposity for testing the efficacy of future therapies for obesity.

COMPUTED TOMOGRAPHY

This basic anatomical image is similar to that obtained using MRI, except it contains additional information for the tissue's true density at each pixel. This information coupled with the anatomical location of the pixel within the image can be used to identify it as adipose, muscle, skin, viscera, or bone tissue. Reconstruction of total body mass and separate organ masses based on scans along the length of the body at 10-cm intervals has been shown to have excellent accuracy (<1% error) and precision (<1%). These reconstructed CT images, and those for MRI, can be assigned to level 4 or tissue systems level of the multicompartment model. The CT images can also be used to separate the total adipose tissue mass into its subcutaneous and visceral components, or the lean tissues into skeletal muscle and visceral or organ mass.

NEAR INFRARED INTERACTANCE (NIR)

This method of assessing body fat is based on the principles of light absorption, reflectance, and near infrared spectroscopy. To estimate body composition, a computerized spectrophotometer that has a scan and probe are used. The probe is placed onto a selected body site such as the biceps; it emits an infrared light which passes through both fat and muscle and is reflected back to the probe.

Subject data such as height, weight, sex, age, frame size and activity level are taken into consideration. Density measurements are obtained and incorporated into the manufacturer's prediction equations. A digital read out including percentage body fat and lean tissue are displayed.

This method of assessing body fat is not the most accurate. In a study by Mclean et al (1992), it was found that skinfolds more accurately predicted body fat than NIR when underwater weighing was used as the criterion measure. In their study, NIR underestimated body fat by more than 4% in subjects greater than 30% fat and overestimated body fat by 4% in subjects less than 8% fat.

Advantages

- Safe
- Non-invasive
- Fast
- Convenient.

Disadvantages

- Not one of the most accurate techniques used to assess body fat composition.

UNDERWATER WEIGHING (HYDROSTATIC WEIGHING)

This method uses Archimedes principle which states that when a body is submerged in water, there is a buoyant counter force equal to the weight of the water which is displaced because bone and muscle are denser than water, a person with a larger percentage of fat free mass will weigh more in the water and have a lower percent body fat. Conversely, fat floats. Therefore, a large amount of fat mass will make the body lighter in the water and have a higher percent body fat. If each test is performed correctly according to the recommended guidelines, there is a +/- 1.5% error.

Advantages

- This method is currently considered the "gold standard" in percent body fat measurement.

- Repeat measures usually prove consistent, and can be used to chart progress.

Disadvantages:

- This method usually requires a lot of equipment and space.
- Testing is time consuming and involved.
- Requires in-depth knowledge to administer the tests and compute the calculations.
- Being submerged under water may be difficult and produce anxiety for some.

VISCERAL ADIPOSITY INDEX

The BMI is wrongly considered a satisfactory predictor of the percentage of body fat, and it is known that it shows a curvilinear and not a linear association with the body fat percentage in both men and women. Beyond the criticisms recently leveled towards the normalization of weight by the square of the height many factors affect the relationship between BMI and body fat percentage, such as gender, race, high muscle mass(e.g., subjects who practice body building), and changes in hydration status (in particular subjects having retention of extracellular fluids may lead to significant mistakes in interpretation about BMI). In older people, significant changes also occur in both BMI

numerator and denominator. Also, in 2005 in JAMA Katherine Flegal published one of the first studies to analyze in detail the correlation between BMI and all causes of mortality on a large series.This study, based on National Health and Nutrition Examination Survey (NHANES) data, showed that BMI is not a good predictor of mortality risk. A subsequent meta-analysis published in *Lancet* in 2006, evaluating 40 epidemiological studies for a mean follow up of 3.8 years, confirmed Flegal's data. These data led in 2006 to an editorial published in Lancet, provocatively titled "Should we continue to use BMI as a cardiovascular risk factor?" which states that "the BMI may now be withdrawn permanently as a clinical or epidemiological tool for evaluation of cardiovascular risk in both primary and secondary prevention."

In fact the BMI does not appear in any of the versions of the algorithm of Framingham, one of the strongest tools for defining coronary and cardiovascular risk. Indeed, in the Framingham study it was assumed that there could be "metabolically healthy obese subjects."

An important contribution to evaluation of the influence of obesity on cardiovascular risk is the Interheart Study, which shows inconvertible evidence that abdominal obesity makes a higher contribution than BMI to the probability of these events. The following year, focusing attention on the relation between obesity and heart attack risk, Yusuf et al.published a study defining the results more accurately, showing that

the association between abdominal adiposity and coronary heart disease risk is highly significant in all geographical areas in which Interheart Study data were collected.

These data incontrovertibly seem to support the usefulness of the evaluation of waist circumference (WC) in cardiovascular risk stratification, having been considered the most valid index of regional distribution of adipose tissue. Measurement of body circumferences, although it is a valid method, requires an accurate performance method in order to provide reproducible information. Several studies have shown that WC is strongly related to visceral fat and abdominal adiposity, more than BMI and waist/hip ratio. The only limitation of WC is inaccurate distinction between visceral and subcutaneous adipose tissue in the abdominal region.

2. A New Method for Evaluation of Adipose Distribution and Function (Visceral Adiposity Index)

The Visceral Adiposity Index (VAI) is an empirical mathematical model, gender-specific, based on simple anthropometric (BMI and WC) and functional parameters (triglycerides (TG) and HDL cholesterol (HDL)), and indicative of fat distribution and function. It is an empirical-mathematical model that does not originate from theoretical assumptions, but from observation in a healthy normal/overweight population of a linear relationship between BMI and CV.

At first a model of adipose distribution (MOAD) was created based on this linear equation (which shows a strong correlation with visceral fat mass determined by MRI. Subsequently MOAD was corrected for triglyceride and HDL cholesterol levels, determining the VAI:

Females:

VAI = (WC) / 36.58 + (1.89*BMI) * (TG/0.81) * (1.52/HDL)

Males:

VAI = (WC) / 39.68 + (1.88*BMI) * (TG/1.03) * (1.31/HDL)

where WC is expressed in cm, BMI in Kg/m2, TG in mg/dL,

and HDL in mg/dL. Assuming VAI = 1 in healthy non obese subjects with normal adipose distribution and normal TG and HDL levels.

The VAI has shown a strong positive correlation with peripheral glucose utilization during euglycemic hyperinsulinemic clamp and seems to be independently associated with cardio- and cerebrovascular events. In the last three years, it has been reported on more than 30 publications, in which the capability of the VAI to express a possible “adipose tissue dysfunction and the cardiometabolic risk associated to it have been evaluated.

3. VAI in the General Population as a Marker of Cardiometabolic Risk

The main aim of our research field on the VAI has been to identify a simple clinical marker of adipose tissue dysfunction (indirectly reflecting cardiometabolic risk), before it develops into an overt metabolic syndrome and/or a cardiovascular complication. Unfortunately, today there are still no longterm prospective studies that allow us to evaluate the predictive power of the VAI regarding cardiovascular risk.

Our first study on the VAI , in a population of 1,498 Caucasian primary care patients, already showed that there was a strong independent association with both cardiovascular (odd ratio (95% CI): 2.45 (1.52–3.95)) and cerebrovascular events (odd ratio (95% CI): 1.63 (1.06–2.50)); in the same study, a receiver operating characteristic (ROC) analysis proved greater sensitivity and specificity of VAI, compared to its individual components (WC, BMI, HDL, and TG) with regard to cardiovascular and cerebrovascular events.

In another ROC analysis on 1518 Peruvian adults, in which various measures of adiposity were evaluated, VAI, WC, and waist-height ratio (WHtR) were the best predictors of the individual components of the metabolic syndrome . In particular, the VAI showed good predictive power regarding the visceral adiposity-related risk of

type 2 diabetes and hypertension . Although in some studies the increase in VAI was significantly associated with a significant reduction in insulin sensitivity ,VAI is not to be seen as an index of insulin sensitivity, but as an indicator of altered adipose function which is associated with insulin resistance.

Few studies connected in different population did not show any superiority of VAI. Hence these data suggest the need for further prospective studies which take into account the greater variability in the years of the VAI, compared to other simple anthropometric measures.

MATERIALS AND METHODS

PLACE OF STUDY:

Chengalpattu Medical College & Hospital, Chengalpattu

DURATION OF STUDY:

6 months (Jan 2016 – June 2016)

STUDY DESIGN:

Case control cross sectional study.

POPULATION STIDIED:

Cases - 100 and Controls – 100

STUDY GROUP:

Patients admitted with a diagnosis of Acute Coronary Syndrome as diagnosed by symptoms, ST segment elevation or depression, T wave changes, in contiguous leads and who presented to the hospital within 24 hours of onset of symptoms and who were willing to give informed consent. The control population included patients presenting to the hospital with any other diagnosis apart from myocardial infarction, unstable angina or non ST elevation such as Dilated Cardiomyopathy, Hypertension with atypical chest pain.

INCLUSION CRITERIA:

FOR CASES:

- Patients admitted with a diagnosis of Acute ST Elevation Myocardial Infarction as diagnosed by symptoms, ST segment elevation in contiguous leads and echo correlation.
- Patients admitted with NON ST Elevation Myocardial Infraction, Unstable angina as diagnosed by symptoms, ST segment and T wave changes.

FOR CONTROLS:

- Patients with DCMP or CAD presenting with atypical chest pain.
- Patients with hypertension atypical chest pain.
- Patients with diabetes atypical chest pain.
- Patients with non cardiac chest pain (NACP).

EXCLUSION CRITERIA:

- Patients presenting with chest pain like
- Patients with dilated cardiomyopathy,
- Reinfraction in patients who gave already undergone CABG or angioplasty.

- Pericarditis.
- Patient who did not give consent for this study.

METHODS

Proper approval for this study was obtained from Institutional ethical committee of Chengalpattu Medical College. Written informed consent was obtained from each participant prior to inclusion in this study. Anthropometric measurements have been done by standard techniques. To ensure correct measurement of height subjects were asked to straighten their back and observer adjusted the head of the subject in Frankfort plane. Weight was measured by electronic weighing scale.

BMI was calculated in Kg/m2 from height and weight. BMI was used as a measure of obesity and subjects were classified as (in adult Asians), a person is considered

- Underweight - BMI <18.5 kg/m²;
- Normal - BMI 18.5 to 22.9 kg/m²;
- Overweight - BMI > 23 kg/m²;
- Obese - BMI > 25 kg/m².

Waist circumference was assessed with tape measure, at the trunk's narrowest portion, between the ribs and the iliac crest at the moment of exhalation, with an unclothed waist,

- Abdominal obesity (waist circumference >90 in men and >80 cm in women).

Visceral Adiposity Index

Females:

- VAI = (WC) / 36.58 + (1.89*BMI) * (TG/0.81) * (1.52/HDL)

Males:

- VAI = (WC) / 39.68 + (1.88*BMI) * (TG/1.03) * (1.31/HDL)

where WC is expressed in cm, BMI in Kg/m2, TG in mg/dL,and HDL in mg/dL. Assuming VAI = 1 in healthy non obese subjects with normal adipose distribution and normal TG and HDL levels.

Biochemical Measures:

Fasting samples of subjects were obtained for triglycerides and HDL. To know the diabetic status fasting blood sugar and post prandial blood sugar were obtained. BP was measured using standard adult BP cuff in right upper arm in sitting position in mm of Hg.

Analysis:

Analysis has been done by comparing the groups using T – test and Chi-Square test depending upon the type of variable. Correlation is assessed by Pearson's method.

RESULTS AND ANALYSIS

There are 200 participants in this study. The information was collected prospectively and the data were entered into MS-Excel sheet. The collected data were analysed with IBM. SPSS statistics software 23.0 version. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the unpaired sample t-test was used. To find the significance in categorical data Chi-Square test was used. In both the above statistical tools the probability value .05 is considered as significant level.

Table : GENDER DISTRIBUTION OF PARTICIPANTS

Sex	Participants		Total
	Experimental	Control	
Female	39	45	84
Male	61	55	116
Total	100	100	200

This table shows that 200 people participated in our study, out of these 84 were females and 116 were males constituting about 42% and 58% respectively.

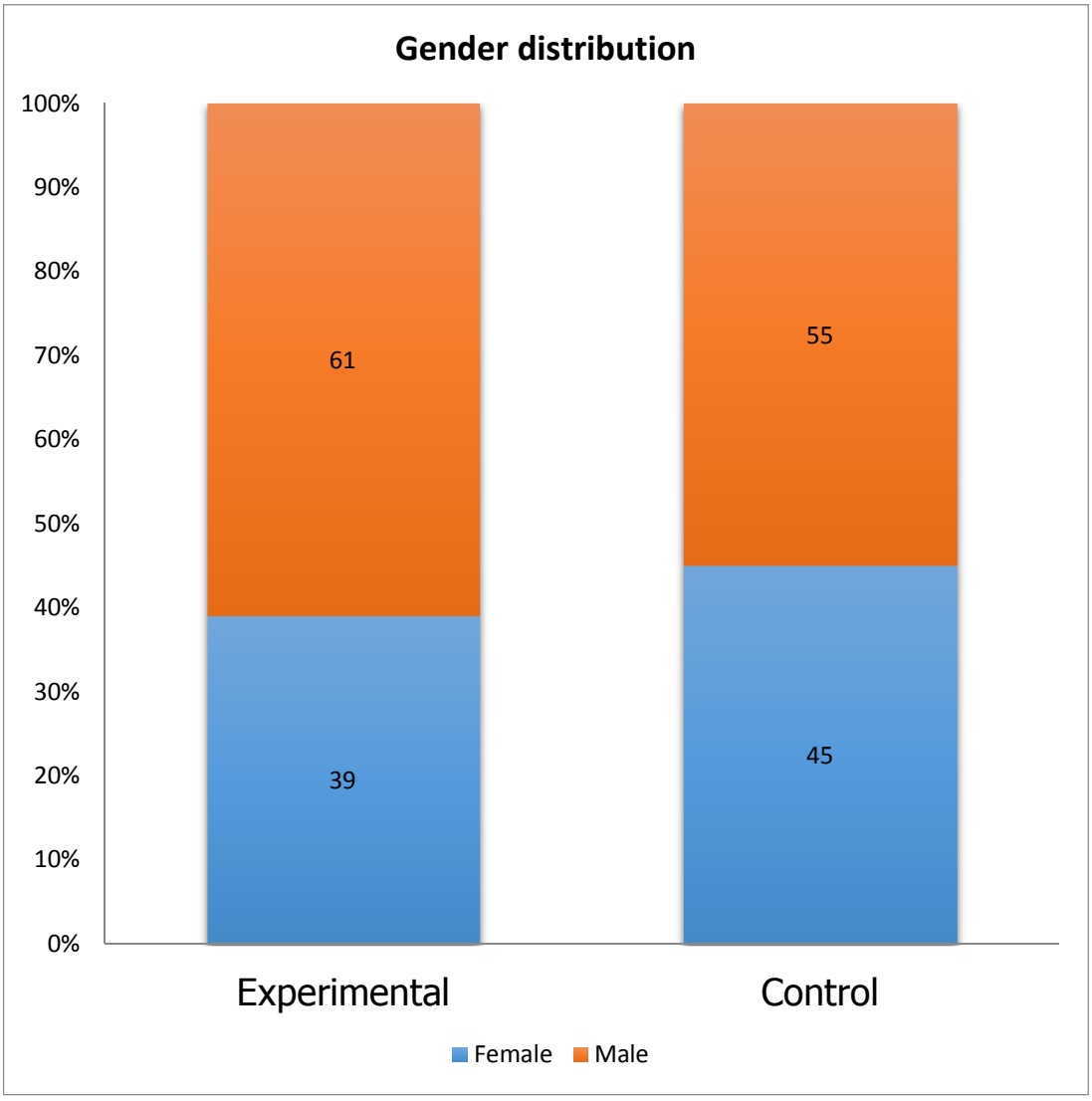
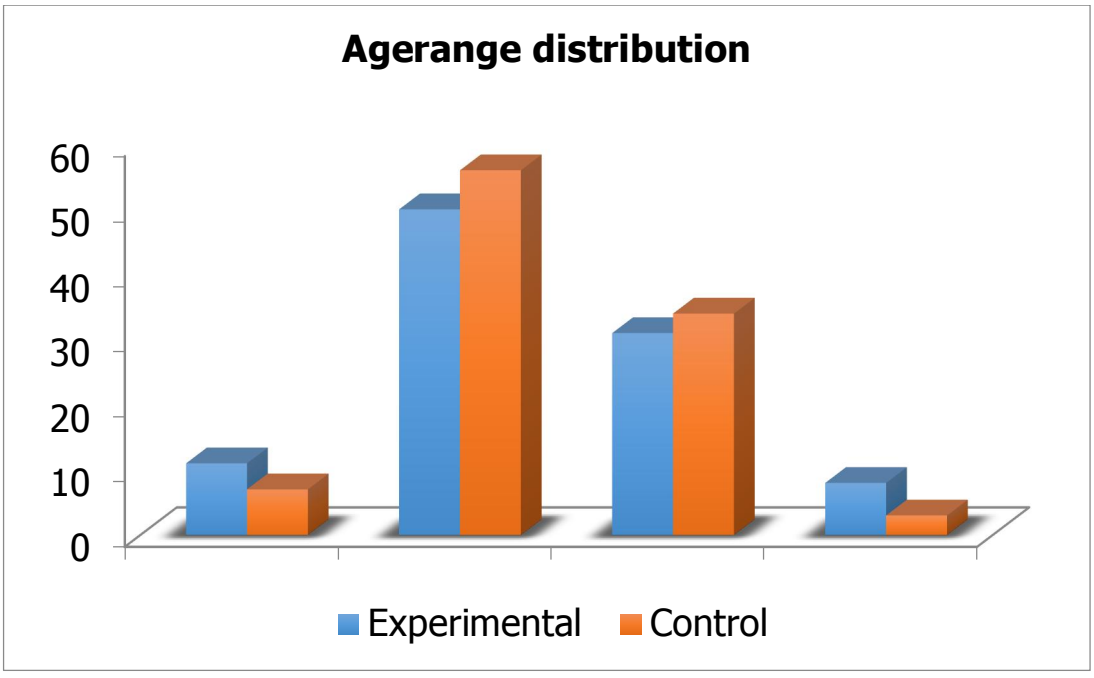

Gender distribution
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
61
55
39
45
Experimental
Control
Female
Male

Table : Age Distribution of Participants

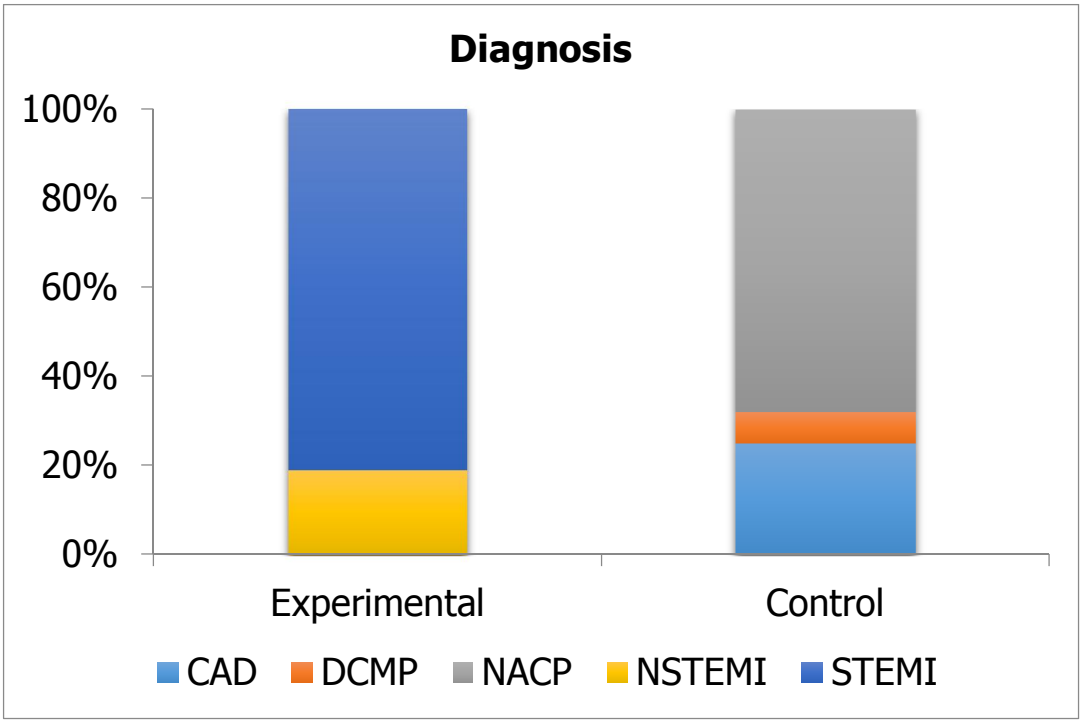
Age	Participants		Total
	Experimental	Control	
Upto 40 yrs	11	7	18
41 - 60 yrs	50	56	106
61 - 80 yrs	31	34	65
Above 80 yrs	8	3	11
Total	100	100	200



This table shows that participants were mostly from the age group of 40 to 80 with majority from 41 to 60 years. Few people were also below 40 and above 80.

Table: Diagnosis

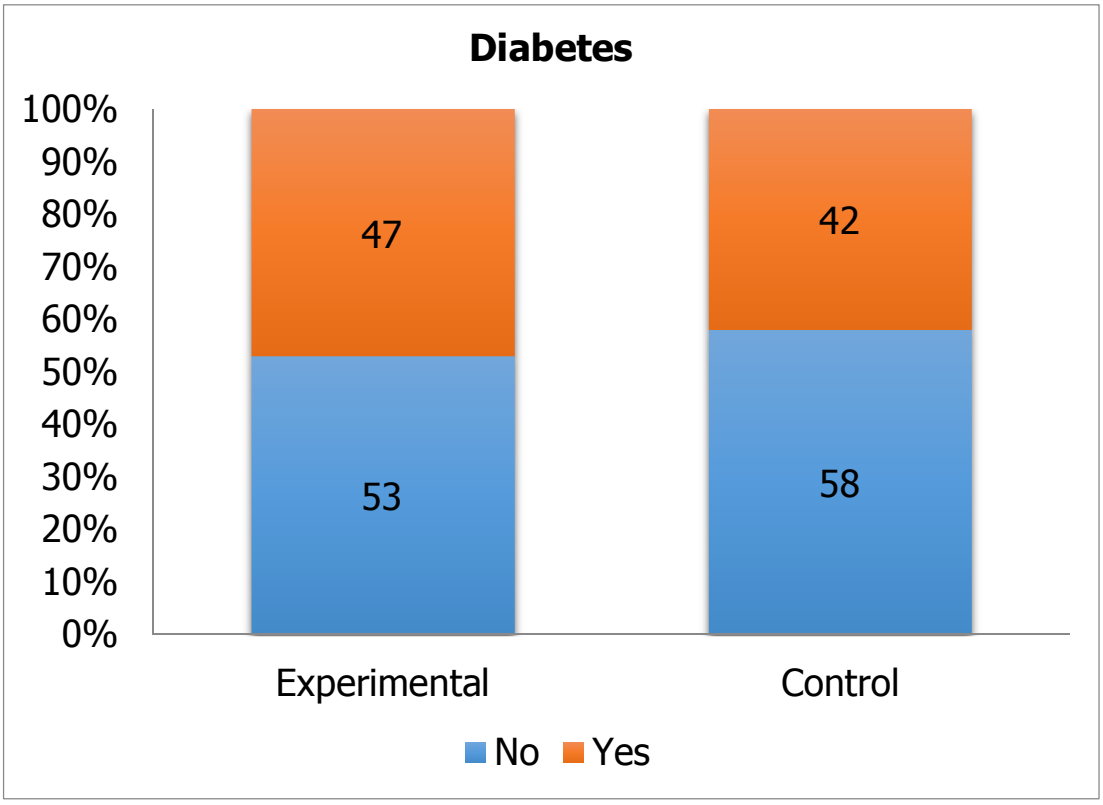
Diagnosis	Participants		Total
	Experimental	Control	
CAD	0	25	25
DCMP	0	7	7
NACP	0	68	68
NSTEMI	19	0	19
STEMI	81	0	81
Total	100	100	200



Out of 100 experimental participants 19 were diagnosed to have NSTEMI and 81 were from STEMI. Matched were selected randomly from those admitted with chest pain due to other causes.

Table: Prevalence of Diabetes in Participants

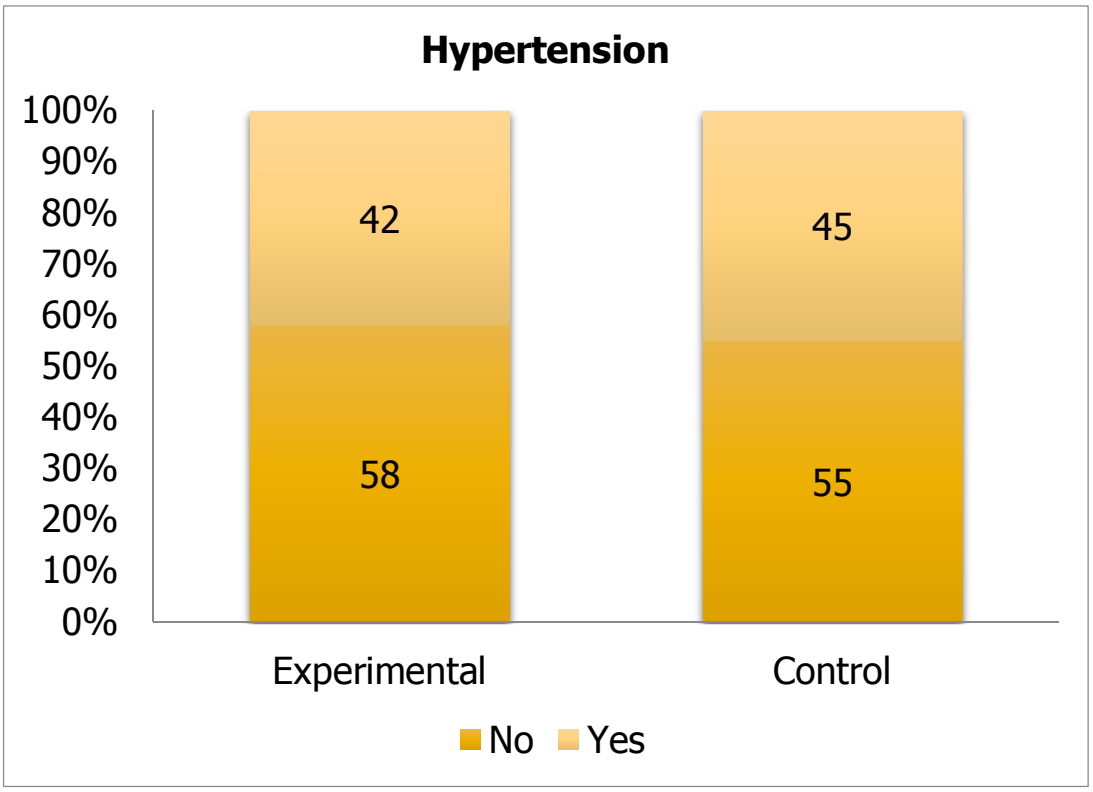
Diabetes	Participants		Total
	Experimental	Control	
No	53	58	111
Yes	47	42	89
Total	100	100	200



Out of 200 participants 89 were with diabetes. Amongst them 47 were from experimental and 42 from control. Hence controls and cases are matched.

Table: Hypertension

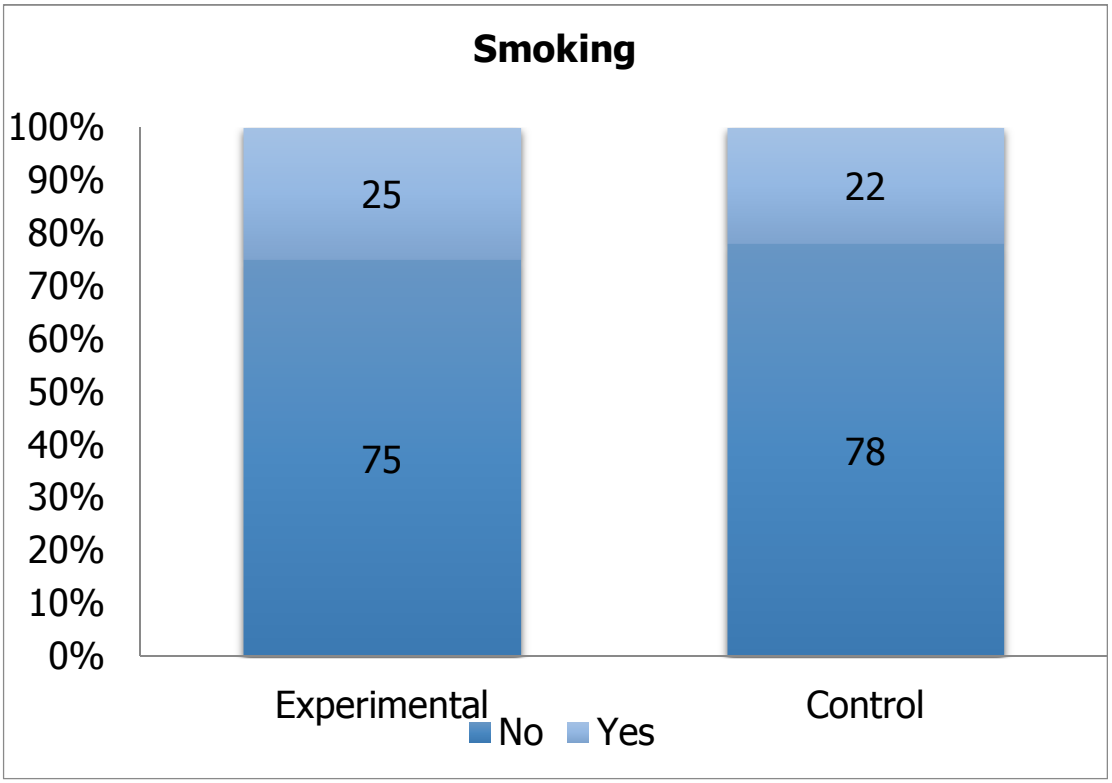
Hypertension	Participants		Total
	Experimental	Control	
No	58	55	113
Yes	42	45	87
Total	100	100	200



Out of 200 participants 87 were with Hypertension. Amongst them 42 were from experimental and 45 from control. Hence controls and cases are matched.

Table: Smoking

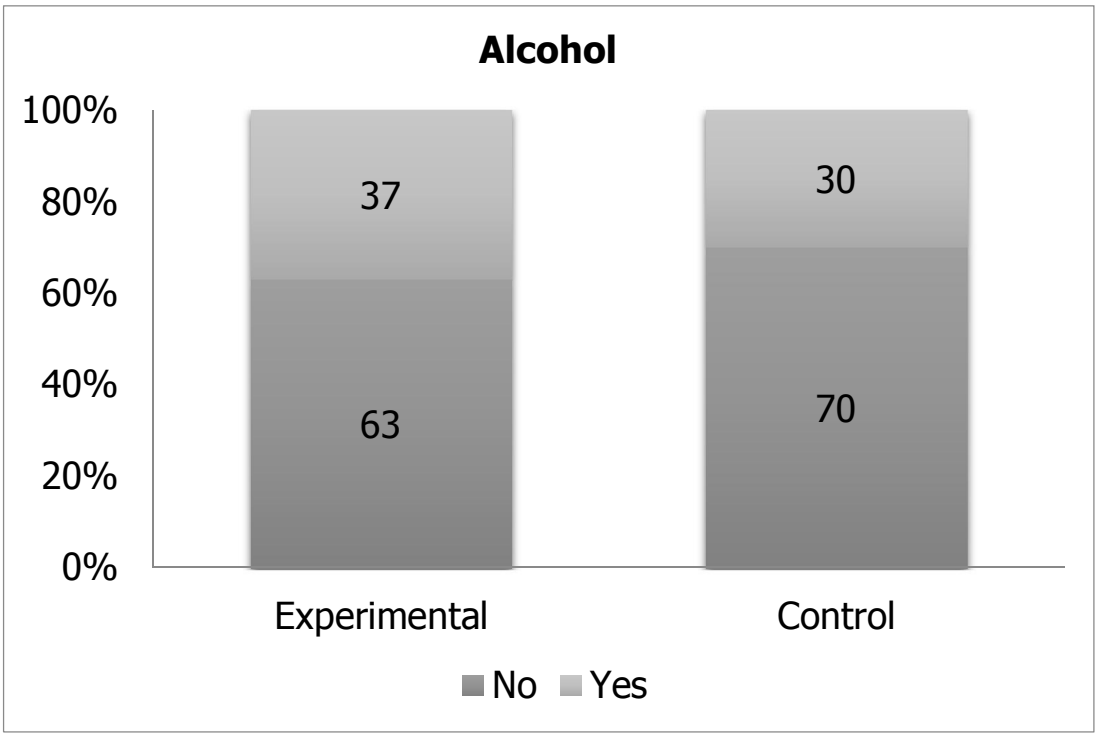
Smoking	Participants		Total
	Experimental	Control	
No	75	78	153
Yes	25	22	47
Total	100	100	200



Out of 200 participants 47 were smokers. Amongst them 25 were from experimental and 22 from control. Hence controls and cases are matched.

Table: Alcohol

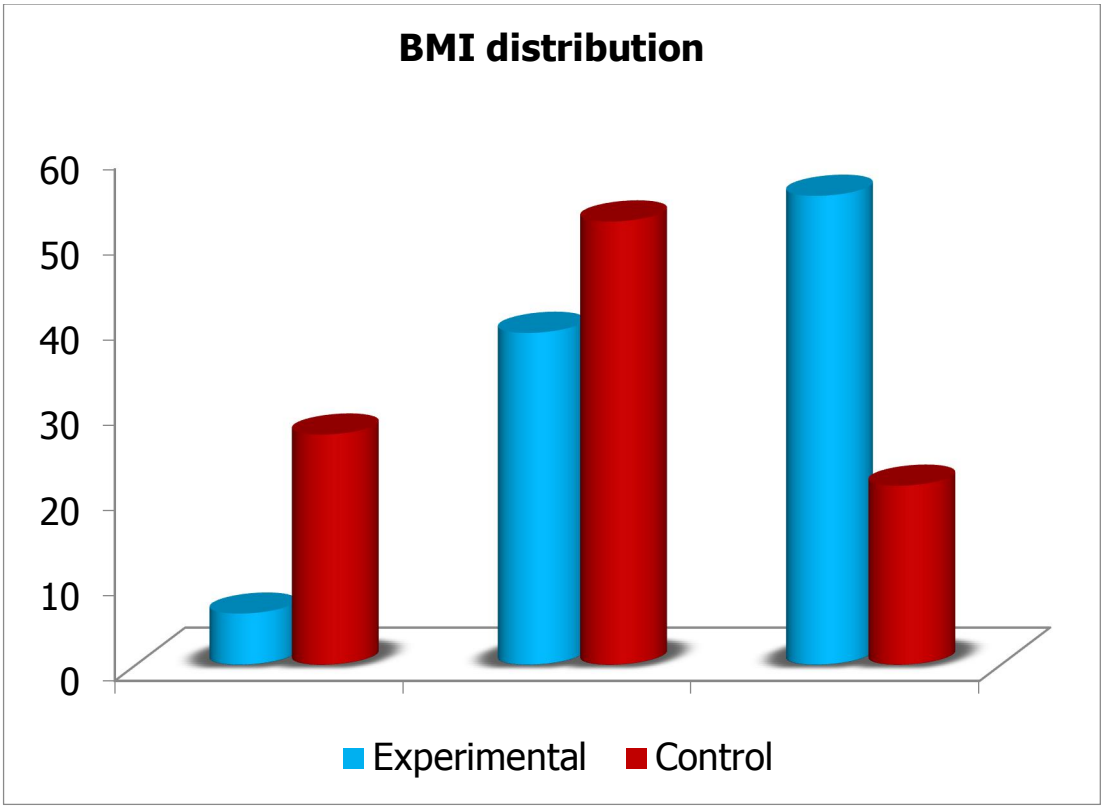
Alcohol	Participants		Total
	Experimental	Control	
No	63	70	133
Yes	37	30	67
Total	100	100	200



Out of 200 participants 67 were alcoholics. Amongst them 37 were from experimental and 30 from control. Hence controls and cases are matched.

Table: BMI Distribution

BMI range	Participants		Total
	Experimental	Control	
Normal	6	27	33
Over weight	39	52	91
Obesity	55	21	76
Total	100	100	200

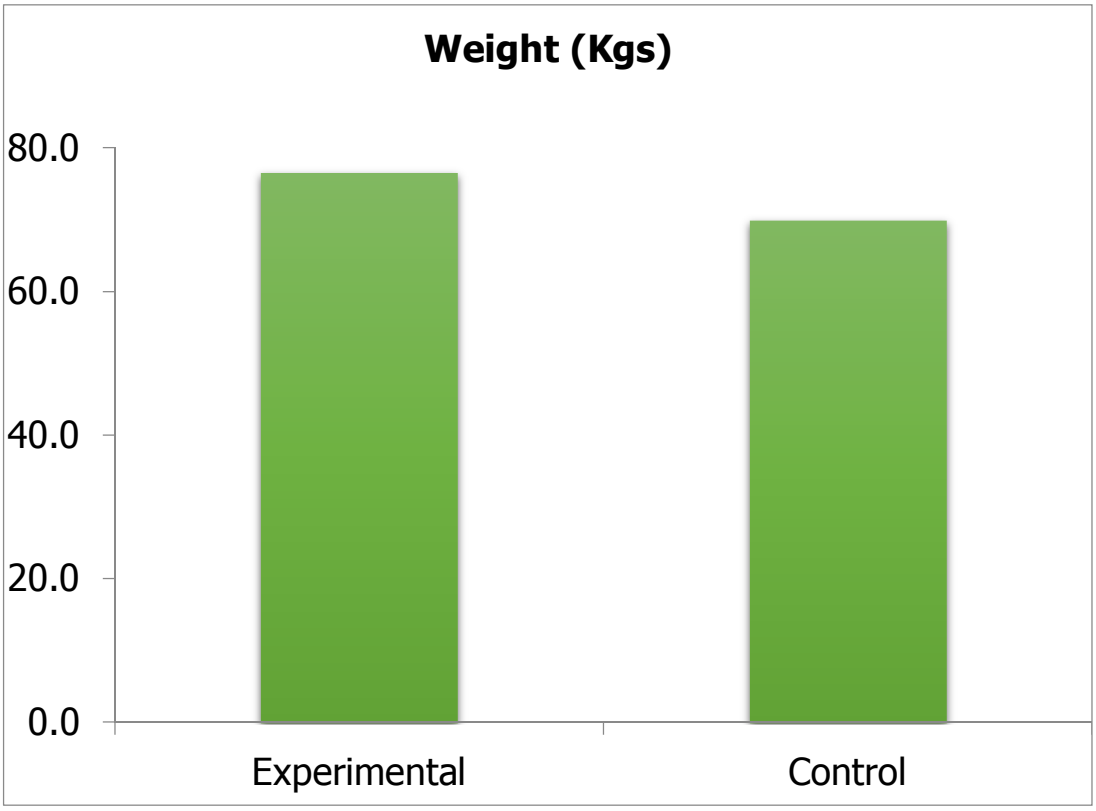


This graph shows 91 were overweight and 76 were obese out of 200 participants. Thus signifying the importance of obesity.

T-Test

Weight:

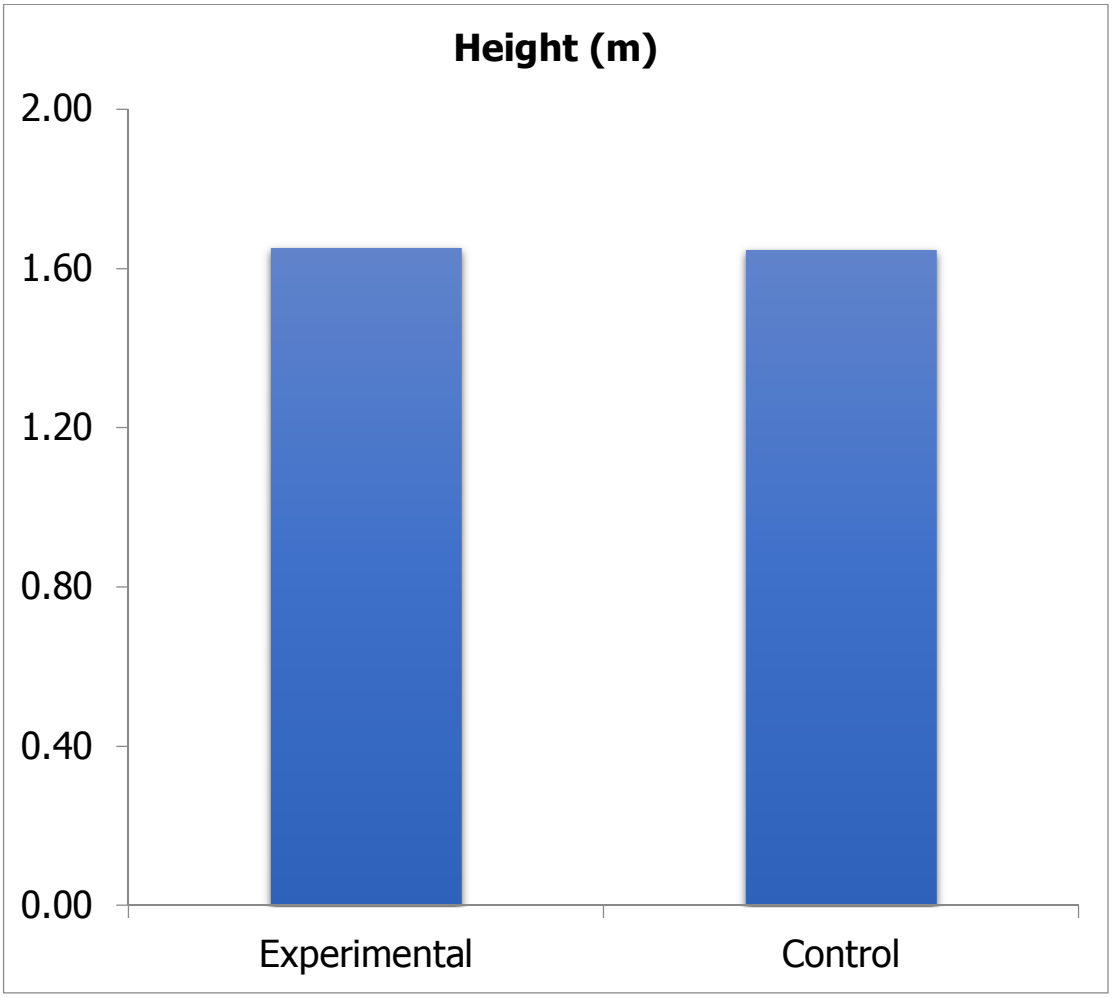
Weight (Kgs)					
Participants	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**p**
Experimental	100	76.47	12.252	1.225	0.0001
Control	100	69.89	11.757	1.176	



This table shows that experimental population are obese than controls with mean weight of the experiments being 76.47 and 69.89 Kgs for controls. This shows significance by p = 0.0001

Height:

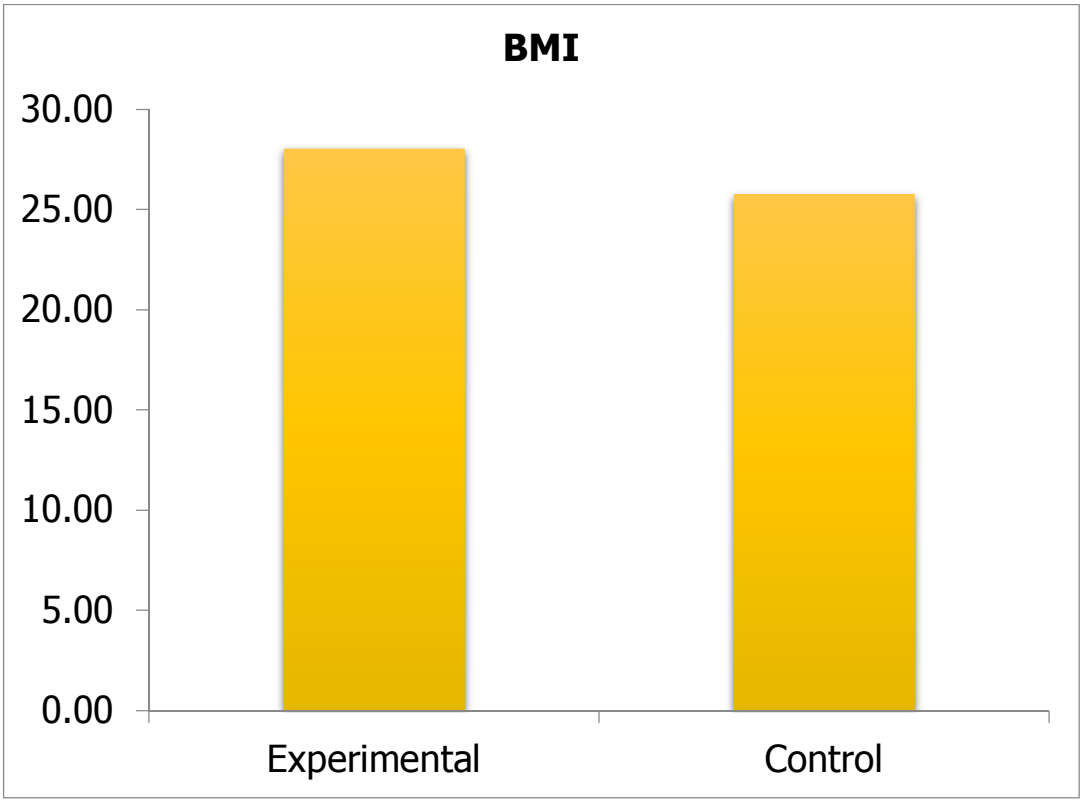
Height (m)					
Participants	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**p**
Experimental	100	1.6495	.08599	.00860	0.801
Control	100	1.6465	.08249	.00825	



This chart shows that experiments and controls are matched by height and there is no significant difference found by p = 0.801.

BMI:

BMI					
Participants	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**p**
Experimental	100	28.0300	3.46044	.34604	0.0001
Control	100	25.7340	3.56283	.35628	



This chart shows that average BMI of the experimental population was 28.03 and 25.73 for controls. T – Test for equality of means was performed between these population which gave p value = 0.0001 and thus found a significance.

BMI with GENDER:

Experimental Participants

Experimental Participants			Sex		Total
			Female	Male	
BMI range	Normal	Count	2	4	6
		% within Sex	5.1%	6.6%	6.0%
	Over weight	Count	12	27	39
		% within Sex	30.8%	44.3%	39.0%
	Obesity	Count	25	30	55
		% within Sex	64.1%	49.2%	55.0%
Total		Count	39	61	100
		% within Sex	100.0%	100.0%	100.0%

This table shows the distribution of BMI among experimental population of which 39% were overweight and 55% were obese. Only 6% of population being normal weight.

Control Participants:

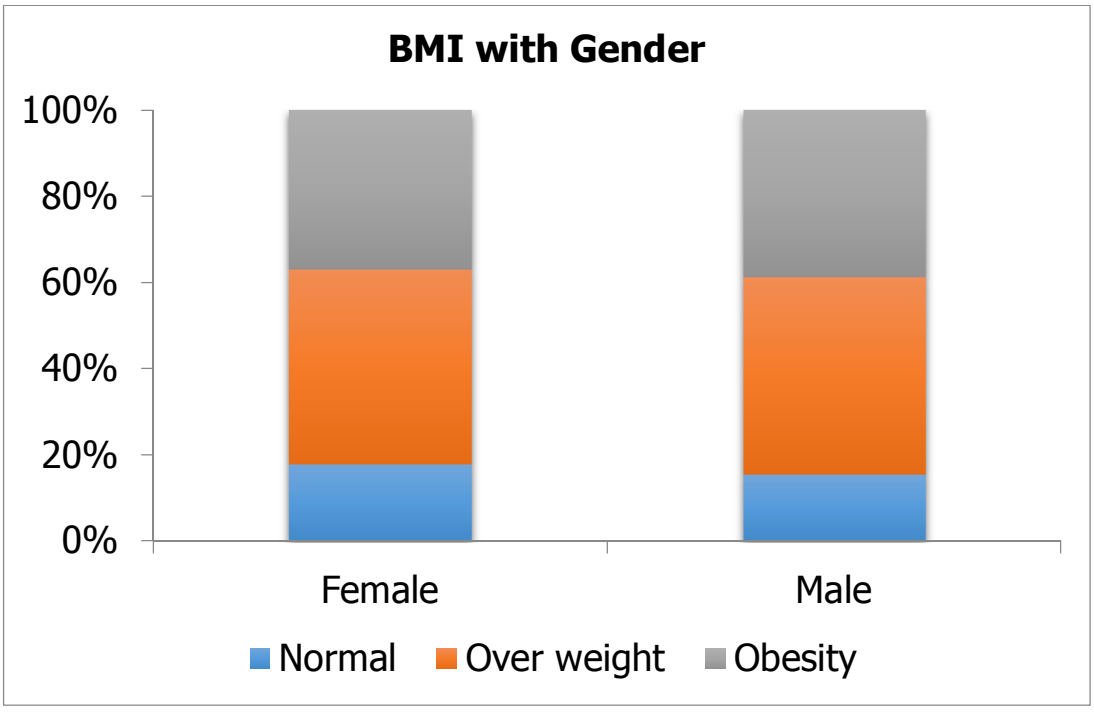
Control Participants			Sex		Total
			Female	Male	
BMI range	Normal	Count	13	14	27
		% within Sex	28.9%	25.5%	27.0%
	Over weight	Count	26	26	52
		% within Sex	57.8%	47.3%	52.0%
	Obesity	Count	6	15	21
		% within Sex	13.3%	27.3%	21.0%
Total		Count	45	55	100
		% within Sex	100.0%	100.0%	100.0%

This table shows the distribution of BMI among control population of which 52% were overweight and 21% were obese. 27% of population being normal weight.

Total Participants with BMI range:

Total Participants			Sex		Total
			Female	Male	
BMI range	Normal	Count	15	18	33
		% within Sex	17.9%	15.5%	16.5%
	Over weight	Count	38	53	91
		% within Sex	45.2%	45.7%	45.5%
	Obesity	Count	31	45	76
		% within Sex	36.9%	38.8%	38.0%
Total		Count	84	116	200
		% within Sex	100.0%	100.0%	100.0%

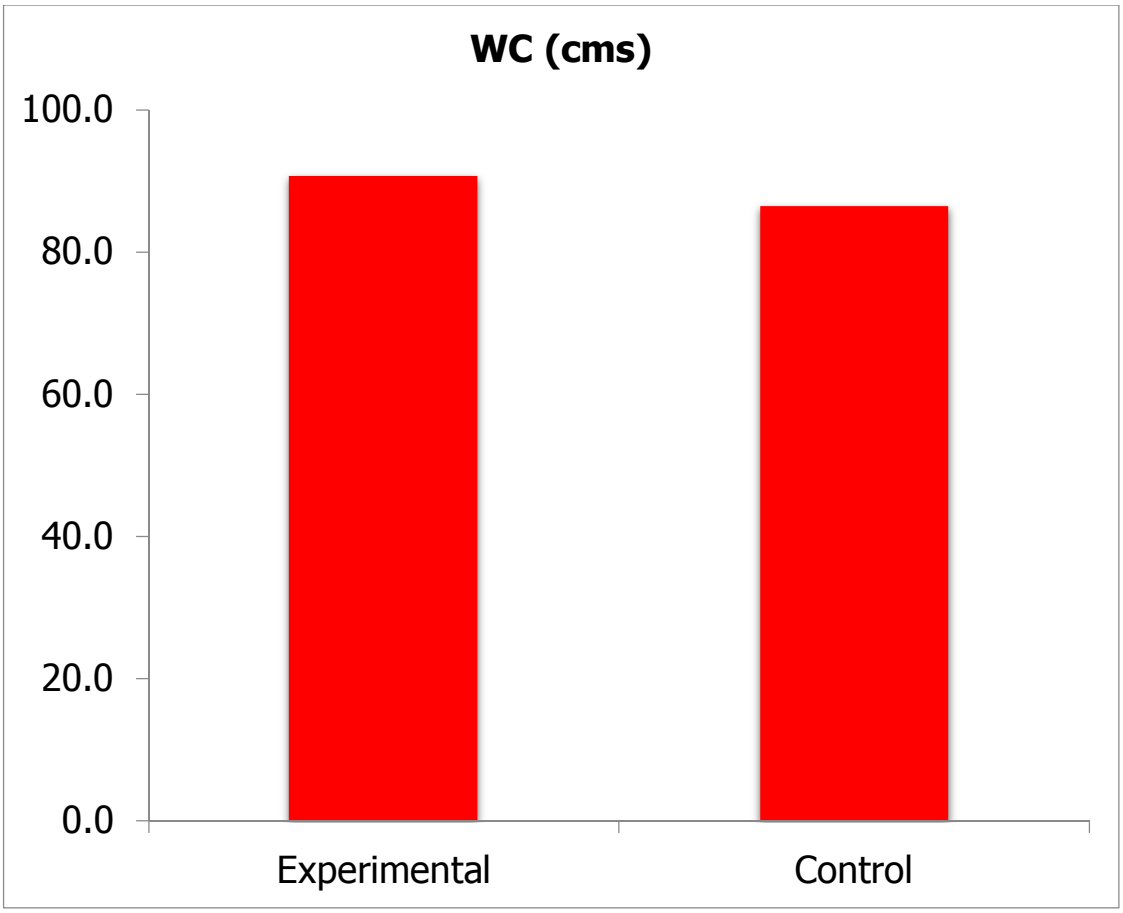
This table shows the distribution of BMI among study population of which 45.5% were overweight and 38% were obese. Only 16.5% of population being normal weight.



This bar diagram shows the distribution of BMI with gender with 18% of female and 15.5% of male being normal. 45.2% of females and 45.7% of males were overweight. 37% of females and 38.8% of males were obese.

WC:

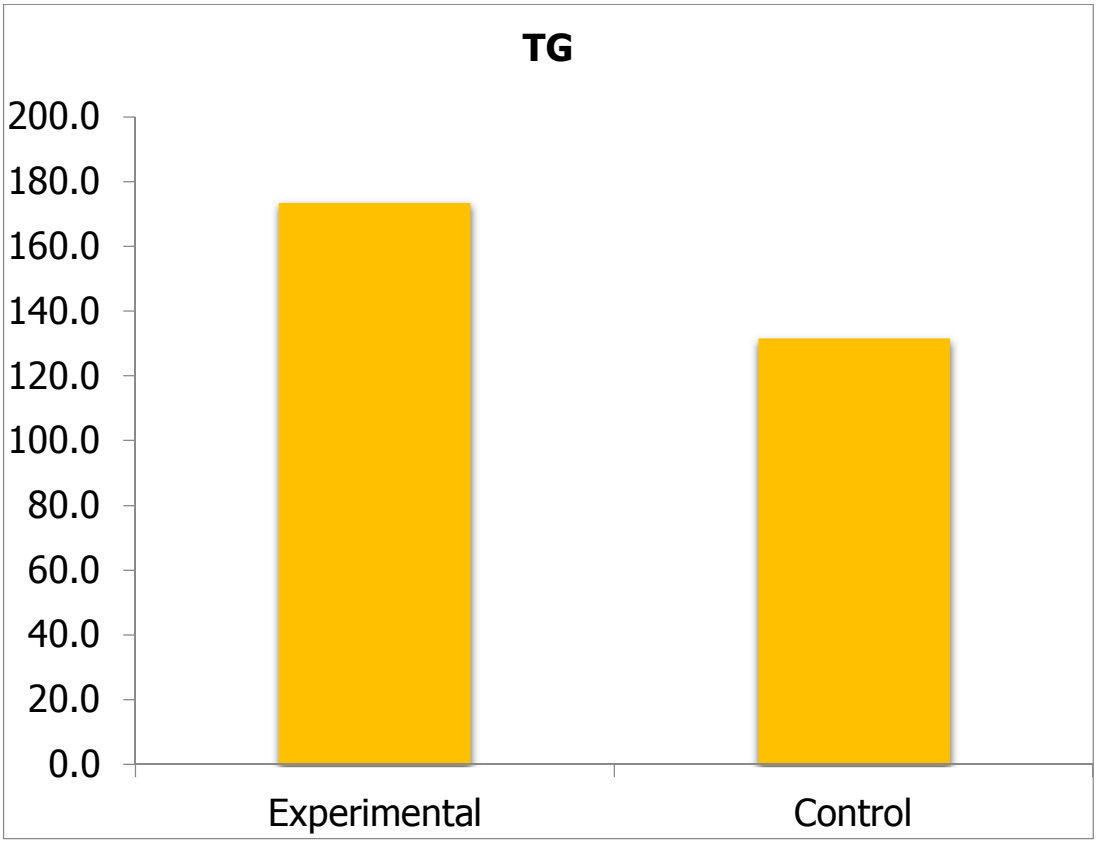
WC (cms)					
Participants	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**p**
Experimental	100	90.73	11.836	1.184	0.0001
Control	100	86.49	11.105	1.111	



This table shows that the mean of WC in experimental population is 90.73 and 86.49 for controls. T – Test for equality of means found significance with p = 0.0001

TG:

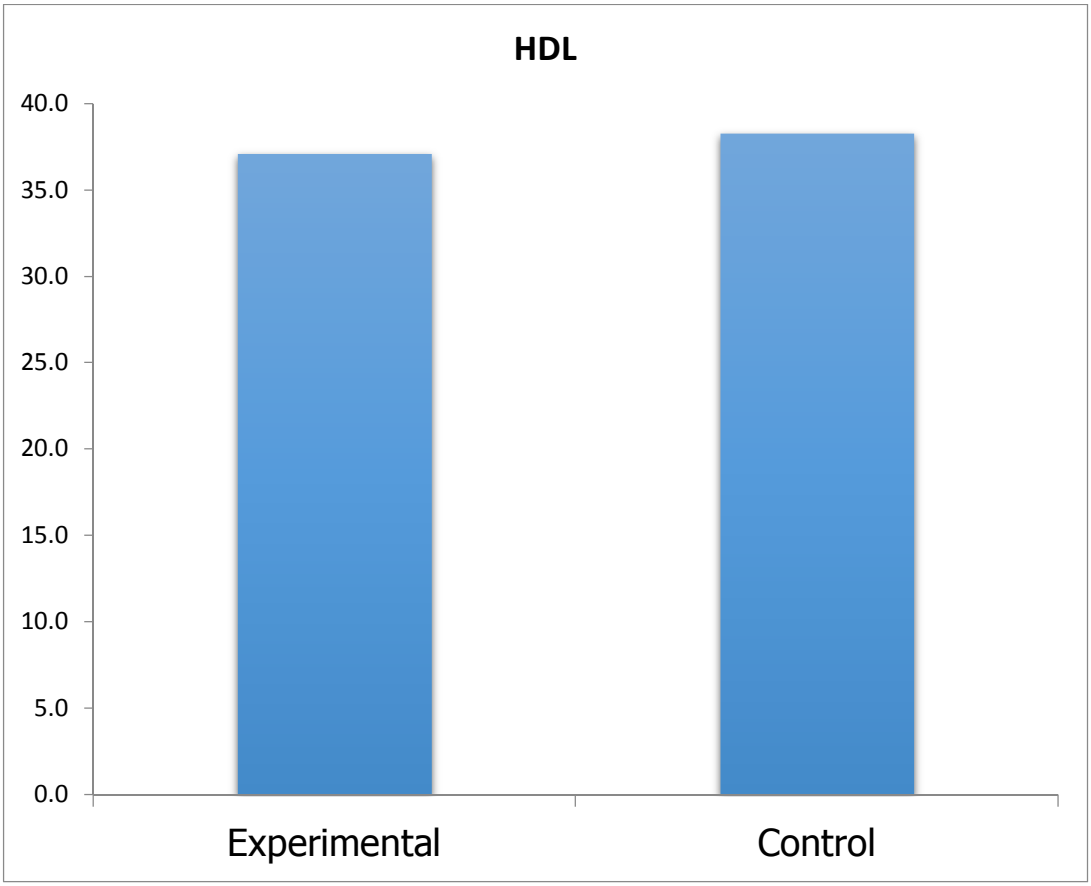
TG					
Participants	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**p**
Experimental	100	173.26	64.055	6.405	0.0001
Control	100	131.45	44.358	4.436	



This table shows that the mean of TG in experimental population is 173.26 and 131.45 for controls. T – Test for equality of means found significance with p = 0.0001

HDL:

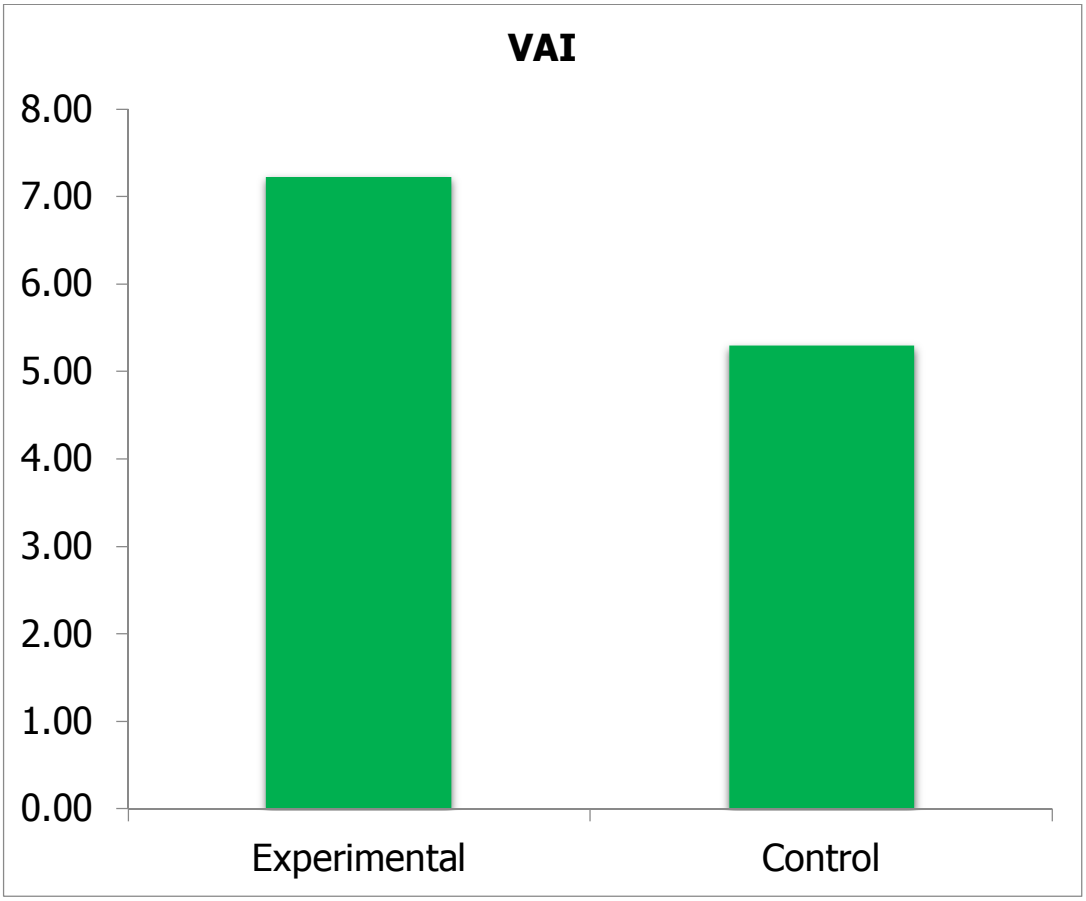
HDL					
Participants	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**p**
Experimental	100	37.08	3.234	.323	0.005
Control	100	38.27	2.719	.272	



This table shows that the mean of HDL in experimental population is 37.08 and 38.27 for controls. T – Test for equality of means found significance with p = 0.005

VAI:

VAI					
Participants	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**p**
Experimental	100	7.2159	3.54018	.35402	0.0001
Control	100	5.2949	2.23323	.22332	



This table shows that the mean of VAI in experimental population is 7.2159 and 5.2949 for controls. T – Test for equality of means found significance with p = 0.0001

VAI with Age Range

Age range = Up to 40 years.

Participants		N	Mean	Std. Deviation	Std. Error Mean
VAI	Experimental	11	4.6200	2.07083	.62438
	Control	7	3.8943	.70684	.26716

Age range = 41 - 60 years

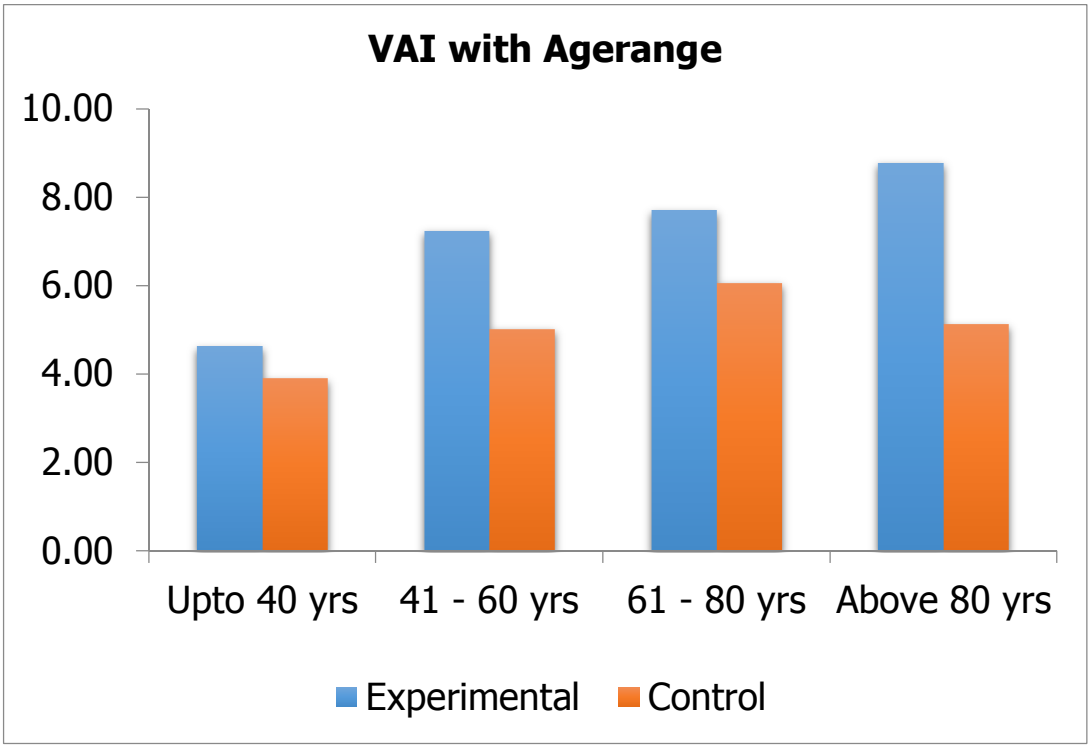
Participants		N	Mean	Std. Deviation	Std. Error Mean
VAI	Experimental	50	7.2354	3.44812	.48764
	Control	56	5.0150	1.94696	.26017

Age range = 61 - 80 years

Participants		N	Mean	Std. Deviation	Std. Error Mean
VAI	Experimental	31	7.7052	3.45338	.62024
	Control	34	6.0591	2.64384	.45342

Age range = above 80 years

Participants		N	Mean	Std. Deviation	Std. Error Mean
VAI	Experimental	8	8.7675	4.73120	1.67273
	Control	3	5.1267	2.60101	1.50169



This bar diagram shows the age wise categorisation of VAI among cases and controls with clear cut higher level of VAI among experiments with a,

- Mean value of 4.62 and 3.89 in age group less than 40, among cases and controls respectively.
- Mean value of 7.23 and 5.01 in age group 41-60, among cases and controls respectively.
- Mean value of 7.7 and 6.05 in age group 61-80, among cases and controls respectively.
- Mean value of 8.76 and 5.12 in age group above 80, among cases and controls respectively.

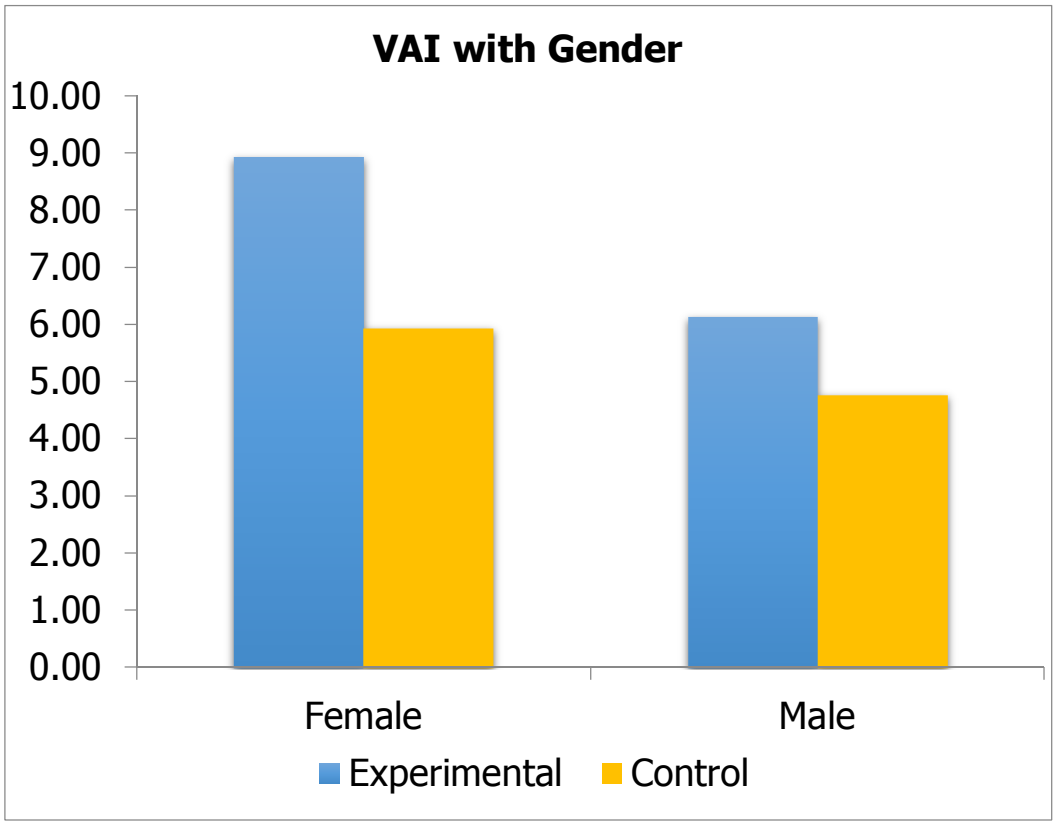
VAI with GENDER:

FEMALES:

Participants		N	Mean	Std. Deviation	Std. Error Mean
VAI	Experimental	39	8.9203	3.59645	.57589
	Control	45	5.9356	2.38756	.35592

MALES:

Participants		N	Mean	Std. Deviation	Std. Error Mean
VAI	Experimental	61	6.1262	3.06466	.39239
	Control	55	4.7707	1.96893	.26549



This bar diagram shows the mean value of VAI among females to be 8.92 and 5.93 among cases and controls respectively and the mean value of VAI among males to be 6.12 and 4.7 among cases and controls respectively. This also shows that females need a higher value for cardiovascular risk than males.

DISCUSSION

Prevalence of Obesity:

In our study of samples 200, 38% are obese and 45% are overweight. This study shows that problem of obesity in India is increasing. The study results conducted by C.S.Yajnik on obesity in India and National family health survey 2005 – 2006 showed that obesity has reached a epidemic proportion in India. Our study also show similar reslt. Recent trend in the westernisation of diet, intake of high caloric processed food and junk foods amongst middle class people combined with sedentary lifestyle has led to this dramatic increase in obesity.

BMI

Only 5% of cases and 25% of controls are within normal weight. 15% of cases and 21% of controls are coming under the category of overweight but 80% of caes are obese and 54% of controls are obese. This data show that an increasing trend towards the obesity and also high prevalence of coronary vascular event among these cases. This study shows a direct relation between the occurrence of obesity and increase in acute coronary syndrome events and thus increased mortality among these patients. The body weight increases with age and obeity is most prevalent among 40 to 70 years of age. This has also been reported by Kapoor and Tyagi and Tandon in studies conducted in India.

Increase in body weight in midlle age may be due to the accumulation of fat with age as thee subjects have larger appetite leading to increased energy intake, fat rich diet and relatively less energy expenditure due to family and professional commitments and sedentary life style.

Diabetes and Acute coronary syndrome:

In our study 47% of cases are Diabetics and this also shows the association of diabetes and acute coronary syndrome. This study shows the importance of metabolic syndrome in predicting the occurrence of acute coronary vascular events. A Study on association betwwen metabolic syndrome and acute coronary syndrome by Virendra Dhakada and Madhu Panjwani also showed a similar result with prevalence of metabolic syndrome in 59% of patients with acute coronary syndrome with prevalence higher in males compared to females.

Our study also shows a similar relationship between higher prevalence of diabetes and acute coronary syndrome.

HYPERTENSION AND ACUTE CORONARY SYNDROME

In our study 42% among cases were Hypertensive. This clearly shows that increased relationship between hypertension and acute coronary syndrome. A study published in Japi by Raihanathul Misiriya, clinical spectrum of acute coronary syndrome experience from a major

center in kerala alo shows increased prevalence of acute coronary syndrome in patients with hypertension with a significant p value.

Hypertiglyceridemia and Acute coronary syndrome:

Our study shows the mean value of Triglycerides to be 173mg/dL whereas that of controls to be 131mg/dL. This shows the higher prevalence of hypertriglyceridemia among our study population and our study also shows a significant p value between the occurrences of acute coronary syndrome and hypertriglyceridemia. A similar result was shown by a study conducted by Maohammad Naeem Malik in patients admitted to CCU with acute coronary syndrome. This study showed an occurrence of hypertriglyceridemia in 83% patients admitted to CCU with acute coronary syndrome.

HDL and Acute coronary syndrome:

HDL was low among cases with a significant p value of less than 0.005. A similar was also showed by INTERHEART study with low HDL and high triglycerides among south Asian population. Among men and women high triglycerides by HDL ratio is a powerful independent predictor of metabolic syndrome cardiovascular events and overall mortality also the study show that prevalence of high lipoprotein A in the presence of low HDL accounts for malignant heart disease in young Asian Indians. So this shows the importance of HDL raising therapy in

reducing the occurrence of acute coronary syndrome and the adoption of healthy diet.

VAI and occurrence of acute coronary syndrome:

VAI is a recently devised gender specific formula calculated using BMI, Waist circumference, Triglycerides and HDL. This formula mainly throws a light on occurrence of metabolic syndrome and thus obesity related mortality. This formula has been studied under various circumstances in relation to PCOD, NASH and NAFLD, cardiovascular diseases but studies in relationship to cardiovascular disease is limited. A study by Amato et al in identifying cutoff points of visceral adipose dysfunction associated with cardio metabolic risk in Caucasian population showed that dysfunctional visceral fat rather than overall obesity was a key component of constellation of metabolic abnormality related to energy surplus sedentary lifestyle and cardio metabolic risk. To identify the visceral fat MRI is the most specific and sensitive test but due to cost of the procedure, it is not routinely performed and not recommended in clinical practice for this purpose.

Visceral Adiposity Index is a mathematical model that uses both functional parameters like TG, HDL and anthropermetic measures like BMI and waist circumference. This index can be considered a simple marker of visceral adipose dysfunction and showed a strong association with both the rate of peripheral glucose utilisation in the hyper

insulinemic state and visceral adipose tissue as measured with MRI. It also showed a strong independent association with cardiovascular and cerebrovascular events and showed better predictive power for visceral adipose tissue dysfunction and its related mortality than the individual components like BMI, Waist circumference and TGs.

Furthermore, over the last decade there has been increasing evidence regarding the endocrine function of the adipose tissue changes in the secretory function of adipocytes and macrophages, along with low grade chronic inflammation are associated with insulin resistance, dyslipidemia, diabetes, vascular disease contributing to the clinical effects of obesity.

Visceral adipose dysfunction showed a significant trend of increased prevalence of cardiovascular event. Severe visceral adipose dysfunction proved to be independently associated with cardiovascular events together with age, male gender, smoking at the time of events when compared with other conventional risk factors alone.

For VAI, which represents a global calculator of non glycemic and non-hemodynamic components of the Met S , while on one hand the variables are treated as continuous variables, on the other two important aspects are taken into consideration, i.e. BMI and gender. In fact, women have, on average, more subcutaneous fat and less visceral fat than men. Although VAI cannot be claimed per se as a diagnostic tool for cardio

vascular and cerebrovascular events, since it includes physical (BMI and WC) and metabolic (T G and H DL) parameters, it may, however, indirectly reflect other non-classical risk factors, i.e. altered production of adipocytokines, increased lipolytic activity and plasma- free fatty acids. In fact, visceral obesity and "High-Triglyceride/Low-HDL-Cholesterol Dyslipidemia" were proposed by Unger et al. who suggested that a state of relative hypoleptinemia can be observed in sub jects with visceral obesity compared with generalized obesity. This condition, which we ourselves consider useful to define visceral fat dysfunction, when associated

with physiological age-linked leptin resistance, leads to pancreatic lipotoxicity with subsequent beta-cell apoptosis and diabetes on set, muscle insulin resistance, liver insulin resistance and NAFL D, lipotoxic cardiomyopathy and generalized endothelial dysfunction.

Our study shows that a definitely high level of VAI among cases with mean value of 7.1 among cases and 5.2 among controls with a p value of 0.0001.

CONCLUSION

Our study was conducted in a sample of population which comprised of patients admitted in Chengelpattu Medical College and Hospital above the age of 20 with complaints of chest pain in the department of General Medicine, Chengelpattu reports that findings of increasing VAI is strongly associated with increased risk of cardio vascular events. Larger epidemiological studies in future may help further elucidate these relationships.

By understanding the correlation between VAI and the acute coronary syndrome, we will be able to better stratify the cardio vascular risk in general population and device strategies for primary prevention accordingly. Since human productivity and cardio vascular morbidity are directly related with obesity being the root cause of many health issues. This study can throw light in formulating a public health policy in preventing obesity and metabolic syndrome.

In conclusion, given the simplicity of WC and BMI measurement and TG and HDL assessment, and the identification of reference cut-off points in a population, we suggest that VAI would be an easy tool for the assessment of VAD, and might be useful in daily clinical practice and in population studies for the assessment of cardio metabolic risk associated with visceral obesity.

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50. Indian J Med Res 117, April 2003, pp 170-179 – Receiver operating characteristics curve analysis of body fat & body mass index in dyslipidaemic Asian Indians, A. Misra, R.M. Pandey*, S.Sinha, R. Guleria, V. Sridhar & V. Dudeja.

PROFORMA

PATIENT DETAILS:

NAME	
AGE	
SEX	

DATE,MONTH,YEAR OF ADMISSION	
WARD NO	
IP NO	

HISTORY:

COMPLAINT OF	
SYMPTOM ONSET	

PAST HISTORY / TREATMENT HISTORY:

DIABETIS MELLITUS	
SYSTEMIC HYPERTENSION	
H/O CVA	

PERSONAL HISTORY:

SMOKING	
CHRONIC ALCOHOLIC	

INVESTIGATIONS:

ECG (STEMI / NSTEMI)	
LIPID PROFILE:TG HDL-C	

MEASUREMENTS:

WEIGHT	
HEIGHT	
BODY MASS INDEX	
WAIST CIRCUMFERANCE	
VAI	

KEY TO MASTER CHART

STEMI	ST segment elevation myocardial infarction.
NSTEMI	Non ST segment elevation MI.
CAD	Coronary Artery Disease.
DCMP	Dilated Cardiomyopathy.
BMI	Body Mass Index.
WC	Waist Circumference.
TG	Triglycerides.
HDL	High Density Lipoprotein.
VAI	Visceral Adiposity Index.

MASTER CHART

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
1	Murugan	58	M	STEMI	No	No	Yes	Yes	70	1.72	23.7	88	150	39	5.11
2	Ellapan	67	M	STEMI	Yes	No	No	Yes	102	1.68	36.1	110	230	32	9.34
3	Maheswari	62	F	NSTEMI	Yes	Yes	No	No	92	1.65	33.8	102	302	35	16.44
4	Lakshmi	68	F	STEMI	No	Yes	No	No	70	1.52	30.3	95	198	39	9.64
5	Lalitha	52	F	STEMI	Yes	Yes	No	No	64	1.51	28.1	88	201	41	9.03
6	Narayanan	72	M	STEMI	No	Yes	Yes	Yes	62	1.58	24.8	90	130	38	4.53
7	Ramasamy	75	M	NSTEMI	No	No	No	No	68	1.6	26.6	85	110	40	3.31
8	Ranganathan	70	M	STEMI	Yes	Yes	No	No	110	1.7	38.1	112	240	31	9.91
9	Premalatha	57	F	STEMI	Yes	No	No	No	70	1.59	27.7	92	159	35	8.82
10	Meera	68	F	STEMI	No	Yes	No	No	72	1.55	30.0	98	192	33	11.47
11	Fathima	55	F	NSTEMI	No	No	No	No	75	1.58	30.0	99	202	36	11.16
12	Gopinath	48	M	STEMI	Yes	No	Yes	Yes	105	1.84	31.0	115	250	34	10.97
13	Pandian	55	M	NSTEMI	No	No	Yes	Yes	68	1.6	26.6	82	106	38	3.24
14	Abib	58	M	STEMI	Yes	Yes	Yes	Yes	96	1.8	29.6	98	248	35	9.25
15	Parvathy	60	F	STEMI	Yes	Yes	No	No	71	1.54	29.9	92	156	38	7.6
16	Balaji	52	M	STEMI	Yes	Yes	No	No	70	1.68	24.8	80	115	39	3.47
17	Vinayagam	65	M	STEMI	Yes	No	No	Yes	84	1.75	27.4	82	178	36	5.65
18	Rafeeq	28	M	STEMI	No	No	Yes	Yes	70	1.75	22.9	75	102	40	2.94
19	Munusamy	78	M	STEMI	No	No	No	No	80	1.7	27.7	90	178	39	5.69
20	Kesavan	68	M	STEMI	Yes	Yes	Yes	Yes	90	1.68	31.9	105	350	31	15.13
21	Aarif	55	M	NSTEMI	Yes	No	No	No	100	1.85	29.2	110	210	38	8.17
22	Sarojini	85	F	NSTEMI	No	No	No	No	70	1.52	30.3	95	148	39	7.2
23	Dhanam	85	F	STEMI	No	No	No	No	72	1.56	29.6	96	200	36	10.82
24	Padmavathy	82	F	STEMI	Yes	Yes	No	No	68	1.55	28.3	85	138	35	6.98
25	Sukumaran	87	M	STEMI	No	Yes	No	No	77	1.7	26.6	82	150	38	4.58

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
26	Muthu	62	M	STEMI	Yes	Yes	No	No	80	1.68	28.3	85	160	37	5.02
27	Rangasamy	38	M	STEMI	No	No	Yes	Yes	70	1.7	24.2	75	138	35	4.41
28	Annamalai	48	M	STEMI	Yes	Yes	No	No	75	1.65	27.5	80	190	39	5.41
29	Joseph	60	M	STEMI	Yes	No	Yes	No	80	1.7	27.7	75	120	42	2.97
30	Valli	51	F	NSTEMI	Yes	Yes	No	No	62	1.55	25.8	82	105	43	4.4
31	Arockiyaraj	30	M	STEMI	No	No	Yes	Yes	70	1.71	23.9	74	99	35	3.14
32	Sujatha	68	F	NSTEMI	Yes	Yes	No	No	90	1.68	31.9	102	135	38	7.02
33	Mary	55	F	STEMI	No	Yes	No	No	80	1.62	30.5	98	202	36	10.95
34	Kanagavalli	70	F	STEMI	No	No	No	No	75	1.6	29.3	85	180	32	9.75
35	Moorthi	45	M	STEMI	No	Yes	Yes	Yes	85	1.8	26.2	90	102	37	3.54
36	Siva	55	M	NSTEMI	No	No	No	No	90	1.67	32.3	105	241	33	9.71
37	Dhanalakshi	85	F	STEMI	Yes	Yes	No	No	92	1.62	35.1	110	268	31	17.35
38	Pathiammal	57	F	STEMI	No	No	No	No	68	1.55	28.3	87	198	39	9.2
39	Pushpa	63	F	STEMI	Yes	No	No	No	58	1.52	25.1	78	157	42	6.51
40	Chittra	51	F	STEMI	No	No	No	No	82	1.58	32.8	98	330	39	15.72
41	Manigandan	35	M	STEMI	No	No	Yes	Yes	85	1.68	30.1	100	141	32	5.81
42	Nagaraj	48	M	STEMI	Yes	No	No	No	80	1.72	27.0	89	101	38	3.32
43	Vettaiyan	34	M	STEMI	No	No	Yes	Yes	85	1.78	26.8	86	99	39	3.08
44	Karunakaran	90	M	STEMI	Yes	Yes	No	No	76	1.65	27.9	83	178	31	6.57
45	Antony	51	M	STEMI	No	Yes	Yes	Yes	85	1.85	24.8	96	230	32	10.16
46	Victor	72	M	STEMI	Yes	Yes	No	No	78	1.65	28.7	90	140	34	5.03
47	Farooq	65	M	NSTEMI	No	Yes	No	No	95	1.7	32.9	110	240	39	8.48
48	Ayesha	58	F	STEMI	Yes	Yes	No	No	85	1.53	36.3	115	301	38	16.24
49	Mohan	55	M	STEMI	No	No	No	No	73	1.58	29.2	100	120	35	4.6
50	Suguna	58	F	STEMI	Yes	No	No	No	80	1.65	29.4	90	152	39	7.14
51	Kamalamal	75	F	STEMI	No	No	No	No	60	1.51	26.3	80	123	32	6.68

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
52	Mumtaj	51	F	NSTEMI	Yes	No	No	No	65	1.52	28.1	81	280	36	13.17
53	Ramesh	38	M	STEMI	No	No	Yes	Yes	80	1.75	26.1	85	97	37	3.19
54	Arumugam	66	M	STEMI	Yes	Yes	No	Yes	84	1.71	28.7	88	161	38	5.06
55	Chinnaya	72	M	STEMI	No	Yes	No	Yes	68	1.7	23.5	82	98	42	2.89
56	Periyasamy	83	M	STEMI	No	No	No	No	65	1.68	23.0	80	105	39	3.3
57	Dhanraj	51	M	NSTEMI	No	Yes	Yes	No	72	1.65	26.4	83	130	35	4.38
58	Latha	71	F	STEMI	Yes	No	No	No	70	1.52	30.3	90	145	42	6.21
59	Kalyani	38	F	NSTEMI	No	Yes	No	No	60	1.51	26.3	75	110	41	4.37
60	Bhuvana	47	F	STEMI	No	No	No	No	54	1.51	23.7	73	88	40	3.7
61	Kaasim	52	M	STEMI	No	Yes	No	Yes	76	1.6	29.7	90	168	39	5.16
62	Robert	61	M	STEMI	Yes	Yes	Yes	Yes	82	1.61	31.6	92	190	42	5.33
63	Marcus	75	M	STEMI	No	No	No	Yes	60	1.72	20.3	70	97	32	3.46
64	Vijayalakhmi	58	F	NSTEMI	Yes	Yes	No	Yes	56	1.52	24.2	75	198	36	9.39
65	Shyamala	48	F	STEMI	No	No	No	No	80	1.65	29.4	90	135	39	6.34
66	Usha Rani	53	F	STEMI	Yes	No	No	No	65	1.66	23.6	80	200	42	8.8
67	Hemalatha	43	F	STEMI	No	No	No	No	70	1.55	29.1	85	250	36	12.08
68	Padmanabhan	57	M	STEMI	Yes	Yes	Yes	Yes	92	1.73	30.7	97	93	37	3.18
69	Adhilakshmi	47	F	NSTEMI	Yes	No	No	No	55	1.53	23.5	75	153	42	6.33
70	Ananth	66	M	STEMI	No	No	Yes	Yes	67	1.66	24.3	85	178	35	6.43
71	Kannan	42	M	NSTEMI	No	Yes	No	No	80	1.72	27.0	90	390	36	13.69
72	Moorthi	47	M	STEMI	No	No	Yes	Yes	82	1.75	26.8	92	201	41	6.37
73	Vasudevan	30	M	STEMI	No	No	Yes	Yes	70	1.72	23.7	84	90	39	2.92
74	Nazir	75	M	STEMI	Yes	Yes	No	No	90	1.68	31.9	105	253	34	9.97
75	Mohammad Ali	81	M	STEMI	Yes	No	No	Yes	95	1.71	32.5	115	285	31	13.34
76	Rizwan	43	M	STEMI	No	Yes	No	No	81	1.68	28.7	98	120	39	4.09
77	Kamakshi	59	F	STEMI	Yes	Yes	No	No	75	1.65	27.5	90	151	42	6.84

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
78	Periyanaygi	61	F	STEMI	No	Yes	No	No	55	1.52	23.8	73	103	39	4.43
79	Pattamal	76	F	STEMI	No	No	No	No	58	1.51	25.4	76	200	35	9.62
80	Velraj	51	M	NSTEMI	Yes	No	No	No	71	1.62	27.1	85	180	40	5.37
81	Kavitha	40	F	NSTEMI	Yes	No	No	No	85	1.73	28.4	90	215	41	9.81
82	Cyril Mathew	28	M	STEMI	No	No	No	Yes	80	1.79	25.0	86	132	32	5.2
83	Govindaraj	55	M	STEMI	Yes	No	No	Yes	74	1.68	26.2	84	121	39	3.72
84	Thandabani	63	M	STEMI	No	Yes	No	No	100	1.7	34.6	120	280	35	11.65
85	Valliammal	59	F	STEMI	No	No	No	No	60	1.65	22.0	76	105	42	4.55
86	Parimala	53	F	STEMI	Yes	No	No	No	52	1.56	21.4	72	89	43	3.63
87	Kumudha	48	F	STEMI	No	Yes	No	No	61	1.51	26.8	82	141	38	6.55
88	Sundaram	57	M	STEMI	No	No	No	Yes	68	1.63	25.6	88	168	37	5.78
89	Manoj	38	M	STEMI	No	No	No	Yes	72	1.69	25.2	82	179	36	5.95
90	Krishnasamy	66	M	STEMI	Yes	Yes	No	Yes	81	1.68	28.7	95	122	39	4.03
91	Murali	59	M	STEMI	No	No	No	No	63	1.75	20.6	75	96	40	2.92
92	Sampath	54	M	NSTEMI	Yes	No	No	Yes	71	1.65	26.1	86	141	36	4.82
93	Vijayashankar	59	M	STEMI	Yes	Yes	No	No	85	1.66	30.8	105	192	32	8.2
94	Raja	66	M	STEMI	Yes	No	No	No	88	1.7	30.4	109	213	37	8.23
95	Syed Ali	49	M	STEMI	No	No	Yes	Yes	96	1.75	31.3	115	290	35	12.28
96	Kannamal	71	F	STEMI	Yes	Yes	No	No	90	1.68	31.9	106	211	32	13.54
97	Muniammal	75	F	STEMI	No	No	No	No	73	1.6	28.5	97	159	38	8.41
98	Raju	51	M	STEMI	Yes	No	Yes	Yes	80	1.7	27.7	99	139	36	5.3
99	Chinnaraj	52	M	STEMI	Yes	No	Yes	Yes	85	1.65	31.2	105	156	33	6.41
100	Ganesan	59	M	STEMI	No	No	Yes	Yes	90	1.72	30.4	108	235	39	8.54
101	Rajesh	51	M	NACP	No	No	Yes	Yes	74	1.75	24.2	82	101	42	2.94
102	Geetha	48	F	NACP	No	No	No	No	60	1.62	22.9	80	97	43	4.24
103	Lakshmiammal	55	F	NACP	No	Yes	No	No	58	1.51	25.4	77	151	41	6.28

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
104	Kanmani	45	F	NACP	No	No	No	No	60	1.58	24.0	78	108	42	4.58
105	Pushparaj	57	M	CAD	Yes	Yes	No	No	81	1.72	27.4	98	121	37	4.47
106	Sangavi	51	F	NACP	No	No	No	No	66	1.68	23.4	79	144	40	6.6
107	Venkatesan	45	M	NACP	No	No	No	Yes	70	1.65	25.7	88	201	40	6.38
108	Perumal	75	M	DCMP	Yes	Yes	No	No	76	1.66	27.6	96	102	41	3.31
109	Karthikeyan	37	M	NACP	No	No	Yes	Yes	72	1.79	22.5	80	97	40	3.01
110	Poornima	49	F	NACP	No	No	No	No	58	1.62	22.1	73	99	42	4.12
111	Annamalai	59	M	CAD	Yes	Yes	No	Yes	86	1.72	29.1	99	131	36	4.85
112	Vanaja	51	F	NACP	No	No	No	No	59	1.61	22.8	79	123	45	5.09
113	Partheepan	55	M	NACP	Yes	Yes	No	No	70	1.73	23.4	82	103	36	3.56
114	Magesh	45	M	NACP	No	No	No	No	79	1.83	23.6	88	115	41	3.73
115	Faranha	61	F	NACP	No	Yes	No	No	53	1.55	22.1	71	108	38	4.83
116	Prasanna	56	M	CAD	Yes	No	No	No	68	1.61	26.2	88	141	39	4.54
117	Faizal	59	M	CAD	Yes	Yes	No	Yes	90	1.67	32.3	110	189	38	6.93
118	Devaki	65	F	CAD	Yes	No	No	No	95	1.68	33.7	105	201	36	10.97
119	Ranganayaki	63	F	NACP	No	No	No	No	55	1.51	24.1	75	97	42	3.95
120	Malathi	52	F	NACP	No	Yes	No	No	58	1.57	23.5	76	106	41	4.54
121	Muthuraman	62	M	CAD	No	Yes	No	Yes	91	1.65	33.4	110	250	39	8.74
122	Chakrapani	66	M	NACP	No	Yes	Yes	No	61	1.66	22.1	82	110	36	3.91
123	Manickam	38	M	NACP	No	No	No	Yes	73	1.81	22.3	82	97	40	3.1
124	Jegannathan	48	M	NACP	No	Yes	No	No	82	1.75	26.8	88	115	38	3.76
125	Sivanesan	57	M	CAD	No	Yes	No	No	108	1.75	35.3	115	270	35	10.64
126	Manjula	68	F	CAD	Yes	No	No	No	58	1.51	25.4	80	114	42	4.81
127	Pangajam	53	F	NACP	No	No	No	No	50	1.55	20.8	72	98	43	4.05
128	Sapthagiri	59	M	NACP	Yes	No	Yes	Yes	65	1.6	25.4	83	108	36	3.62
129	Meiyanandam	56	M	NACP	Yes	No	No	No	70	1.65	25.7	88	121	38	4.04

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
130	Vijayammal	62	F	CAD	No	Yes	No	No	63	1.59	24.9	83	99	39	4.72
131	Narasimhan	61	M	NACP	No	Yes	No	Yes	71	1.69	24.9	87	135	36	4.8
132	Seetha	58	F	NACP	Yes	No	No	No	82	1.58	32.8	102	251	36	13.52
133	Lurdusamy	59	M	CAD	No	Yes	Yes	No	68	1.65	25.0	85	121	37	4.08
134	Saravanan	30	M	NACP	No	No	Yes	No	75	1.75	24.5	86	103	35	3.75
135	Amirthavalli	52	F	NACP	Yes	No	No	No	60	1.62	22.9	73	125	39	5.5
136	Selvaraj	55	M	NACP	Yes	No	No	Yes	71	1.66	25.8	86	139	37	4.66
137	Vadivelu	53	M	NACP	No	No	Yes	Yes	80	1.75	26.1	90	105	39	3.47
138	Monika	41	F	NACP	No	No	No	No	51	1.5	22.7	73	135	41	5.67
139	Banumathy	56	F	CAD	Yes	Yes	No	No	80	1.65	29.4	105	198	39	10.85
140	Lakshmi	52	F	NACP	No	No	No	No	56	1.53	23.9	83	127	43	5.62
141	Pathiammal	54	F	NACP	Yes	No	No	No	62	1.57	25.2	82	104	41	4.64
142	Murugappan	77	M	CAD	No	Yes	Yes	No	67	1.58	26.8	89	141	36	4.91
143	Vinayagamurthy	65	M	CAD	Yes	No	No	Yes	75	1.69	26.3	92	101	37	3.58
144	Kanniappan	69	M	CAD	Yes	Yes	No	Yes	90	1.68	31.9	106.5	271	38	9.69
145	Arumugam	81	M	CAD	No	Yes	Yes	No	65	1.7	22.5	82	103	36	3.64
146	Kannagi	79	F	NACP	No	No	No	No	61	1.57	24.7	84	139	37	7.1
147	Gunavathy	36	F	NACP	No	No	No	No	53	1.59	21.0	71	107	43	4.35
148	Kathirammal	85	F	CAD	No	Yes	No	No	72	1.65	26.4	89	135	32	8.13
149	Bagyalakshmi	39	F	NACP	No	No	No	No	59	1.61	22.8	77.5	109	40	4.97
150	Glory	41	F	NACP	Yes	No	No	No	65	1.55	27.1	85	111	39	5.17
151	Anandhan	62	M	NACP	No	Yes	Yes	Yes	68	1.76	22.0	82	97	39	3.2
152	Krishnamurthy	64	M	NACP	Yes	No	Yes	Yes	72	1.68	25.5	90	123	36	4.46
153	Ranganathan	68	M	CAD	Yes	Yes	No	No	98	1.67	35.1	115.5	291	35	11.55
154	Ramasamy	59	M	DCMP	Yes	No	No	No	78	1.68	27.6	98	108	39	3.76
155	Iyyasamy	47	M	NACP	No	No	Yes	Yes	65	1.68	23.0	80.5	99	32	3.81

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
156	Elango	55	M	NACP	Yes	Yes	Yes	No	87	1.82	26.3	95	114	39	3.96
157	Dhanam	58	F	NACP	No	Yes	No	No	54	1.54	22.8	76.5	110	43	4.61
158	Veeralakshmi	71	F	CAD	No	Yes	No	No	61	1.64	22.7	80	99	39	4.79
159	Anandhi	56	F	NACP	Yes	No	No	No	58	1.56	23.8	79.5	106	40	4.84
160	Indhrani	68	F	DCMP	Yes	No	No	No	75	1.58	30.0	103	171	35	10.11
161	Jothy	39	F	NACP	No	No	No	No	59	1.61	22.8	73	92	42	3.76
162	Shanmugam	48	M	NACP	No	Yes	Yes	Yes	75	1.68	26.6	89	147	34	5.45
163	Sundaramurthy	59	M	NACP	No	No	No	No	80	1.7	27.7	90	151	39	4.83
164	Murugavel	62	M	CAD	Yes	Yes	No	No	95	1.68	33.7	115.5	221	35	9
165	Velusamy	52	M	NACP	Yes	No	Yes	Yes	80	1.71	27.4	90	103	39	3.31
166	Pannerselvam	57	M	NACP	No	No	Yes	Yes	60	1.74	19.8	77.5	106	37	3.67
167	Pazhanisamy	62	M	NACP	No	No	Yes	Yes	70	1.68	24.8	87	136	36	4.84
168	Kanniappan	65	M	DCMP	Yes	Yes	No	No	91	1.65	33.4	106.5	178	35	6.71
169	Prabhakar	49	M	NACP	No	No	Yes	Yes	73	1.7	25.3	89	135	39	4.49
170	Sukumaran	85	M	CAD	No	Yes	No	No	80	1.67	28.7	100	101	38	3.61
171	Samikannu	75	M	CAD	No	Yes	No	No	65	1.75	21.2	80.5	121	39	3.99
172	Varalakhsmi	69	F	DCMP	Yes	No	No	No	71	1.65	26.1	83.5	112	40	5.1
173	Premavathy	62	F	DCMP	Yes	Yes	No	No	85	1.6	33.2	98	261	36	13.42
174	Indhurani	59	F	NACP	No	Yes	No	No	56	1.55	23.3	76	106	39	4.8
175	Fathima	68	F	NACP	No	No	No	No	64	1.53	27.3	83	123	41	5.29
176	Nazreen	53	F	NACP	Yes	No	No	No	74	1.49	33.3	106.5	182	39	9.36
177	Veerappan	67	M	CAD	No	Yes	No	No	72	1.78	22.7	82	97	37	3.31
178	Velu	63	M	NACP	No	No	No	Yes	76	1.67	27.3	88	101	36	3.45
179	Muthukrishnan	55	M	NACP	Yes	No	No	Yes	71	1.62	27.1	88.5	115	39	3.66
180	Aiyanar	59	M	NACP	No	Yes	Yes	Yes	82	1.84	24.2	93.5	98	34	4.02
181	Vasanthi	58	F	NACP	Yes	No	No	No	60	1.59	23.7	79	118	39	5.5

S.No	Name	Age	Sex	Diagnosis	Diabetes	Hyper tension	Smoking	Alcohol	Weight (Kgs)	Height (m)	BMI	WC (cms)	TG	HDL	VAI
182	Rajathi	77	F	CAD	No	Yes	No	No	61	1.5	27.1	88	141	37	7.16
183	Raaji	49	F	NACP	No	Yes	No	No	56	1.51	24.6	77.5	109	39	4.89
184	Adhimulam	44	M	NACP	Yes	No	No	Yes	58	1.61	22.4	79	101	31	4
185	Vasantha	51	F	NACP	Yes	No	No	No	65	1.6	25.4	75	121	37	5.44
186	Devipriya	38	F	NACP	No	No	No	No	54	1.5	24.0	70.5	99	37	4.32
187	Muniyandi	53	M	NACP	Yes	No	Yes	No	78	1.68	27.6	86	117	32	4.36
188	Murugammal	66	F	NACP	Yes	Yes	No	No	68	1.59	26.9	80.5	101	38	4.59
189	Raajam	48	F	NACP	No	No	No	Yes	67	1.64	24.9	78.5	99	36	4.84
190	Kayalvizhi	55	F	NACP	No	Yes	No	No	63	1.57	25.6	76.5	121	41	4.99
191	Meenakshi	63	F	NACP	Yes	Yes	No	No	63	1.61	24.3	79.5	119	41	5.24
192	Visalatchi	71	F	NACP	No	Yes	No	No	58	1.55	24.1	71	110	37	4.81
193	Saroja	65	F	NACP	Yes	Yes	No	No	61	1.64	22.7	74	116	41	4.94
194	Arockiyasamy	61	M	DCMP	Yes	Yes	No	No	90	1.65	33.1	114.5	198	36	7.86
195	Muniappan	67	M	CAD	Yes	Yes	No	No	85	1.71	29.1	98.5	202	39	6.87
196	Pachamuthu	55	M	NACP	No	Yes	No	Yes	65	1.69	22.8	80	109	39	3.44
197	Rajkumar	56	M	NACP	Yes	No	No	No	71	1.76	22.9	83.5	129	36	4.59
198	Saravanavel	49	M	NACP	No	Yes	Yes	Yes	62	1.79	19.4	78	151	39	5.05
199	Velayudham	53	M	NACP	No	No	Yes	Yes	60	1.68	21.3	80.5	97	35	3.56
200	Ganapathy	60	M	CAD	Yes	No	No	Yes	77	1.72	26.0	89	106	39	3.47