DISSERTATION ON

A STUDY ON BODY FAT DISRIBUTION AND CARDIOVASCULAR RISK FACTORS

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON BODY FAT DISTRIBUTION AND CARDIOVASCULAR RISK FACTORS"** submitted by **Dr. G.GOPINATH** appearing for M.D. Branch I General Medicine Degree examination in March 2009 is a bonafide record of work done by him under my direct audience and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, and India.

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DECLARATION

I, **Dr.G.Gopinath** solemnly declare that I carried out this work on "**A STUDY ON BODY FAT DISTRIBUTION AND CARDIOVASCULAR RISK FACTORS"** at Institute of Internal Medicine, Government General Hospital and Madras Medical College during the period of November, 2007 - November, 2008. I also declare this bonafide work or a part of this work was not submitted by me or any other Person for any award, degree and diploma to any university, board either in India or abroad.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M.D. in General Medicine Degree examination.

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CONTENTS

INTRODUCTION

Cardiovascular disease is the leading cause of death in global deaths and accounts for 17 million people worldwide. Despite the favorable changes in the risk factors, new risk factors have emerged. These include abdominal obesity and physical inactivity, both of which are considered independent and mediating factors in the development of cardiovascular disease. They are also associated with type 2 Diabetes and Metabolic syndrome, growing health hazards all over the world and the major risk factors for cardiovascular disease.

Visceral fat accumulation may underlie the adverse metabolic profile associated with obesity. Indeed Waist circumference and Waist Hip ratio as indicators of Abdominal obesity have been shown to be better than body mass index as an indicator of total adiposity for identifying individuals at higher risk of developing atherosclerotic diseases.

It is plausible that Body mass index may be less sensitive than Waist circumference or Waist Hip ratio at capturing the underlying and disparate metabolic effects of fat depots.

A case control study involving the populations worldwide recently reported that Waist Hip ratio was associated with Acute Myocardial Infarction independently of, and more strongly than Body Mass index.

Furthermore Waist and Hip circumferences have been shown to have separate and opposite cross sectional associations with metabolic factors.

The Obesity associated increased risk for developing cardiovascular disease could be due to the adverse metabolic profile associated with increased visceral fat accumulation rather than to subcutaneous fat, which comprises >85% of the total body fat.

However using more sophisticated instruments, such as MRI, to accurately quantify fat in specific depots is impractical for in clinical setting. Simple anthropometric measures, which are known to correlate with fat distribution, would therefore be preferred.

Body mass Index, which is weight/ height², is a measure used to define overweight and obesity, but this measure does not provide enough information on fat distribution. Alternatively, Waist circumference could be measured and is simple enough to use in assessing abdominal obesity over time.

However Waist Girth when correlated with Hip circumference, a measure that showed an independent and seemingly and protective effect on Cardiovascular Disease. Without Hip girth taken into account waist circumference may underestimate the true cardiovascular risk.

Waist Hip ratio may be an alternative measure to use because it is strongly predictive of cardiovascular risk in both men and women.

Even lean individuals (BMI<25 kg/m²), an increased Waist Hip ratio was associated with higher cardiovascular risk, suggesting that the impact of excess visceral fat can be observed even without gaining so much weight as to be considered overweight or obese.

However, despite the need to reduce excess weight in healthy individuals, the role of excess weight reduction in patients with known history of cardiovascular disease or diabetes needs further investigation.

Skinfold thickness is also used as a measure of adiposity, but studies comparing its predictive ability to other measures of adiposity have produced inconsistent findings

Indians have a low average body-mass index (20–30 kg/m^2) and low rates of obesity (10–15%) in association with higher prevalence of cardiovascular disease and diabetes .Mean waist-hip girth ratios were higher and trunk skin folds thicker in south-Asian than in European men and women in the absence of corresponding ethnic differences. In Indians, wherever they are living, glucose intolerance and insulin resistance are associated with obesity and especially with a pattern of obesity in which a high proportion of body fat is deposited on the trunk and in the abdomen.

AIM OF THE STUDY

1) To investigate the associations of abdominal obesity and overall obesity with the risk of acute coronary events.

2) To determine the associations of Waist circumference and Waist Hip ratio with the risk of incident cardiovascular events and to determine the strength of association of waist and waist hip ratio with cardiovascular risk is different.

3) To determine which of the five risk factors maintains the strongest association with cardiovascular risk.

REVIEW OF LITERATURE

The leading cause of death in the world is ischemic heart disease, a condition characterized by reduced blood supply to the heart that is usually due to coronary artery disease. In 2001 alone, some 7.1 million deaths were attributed to ischemic heart disease, 80% of which were in relatively poor countries[6]. Medical and public health professionals expect that in developing countries, there will be a 137% and 120% increase in the disease for males and females, respectively, whereas these predictions lie in the 30% to 60% range for developed countries[6].

Table 1. Estimates of 10 leading causes of global death in GBD 2000 study

Developed countries			Developing countries		
Rank	Cause	% of total deaths	Rank	Cause	% of total deaths
1	Ischemic heart disease	22.6	1	Ischemic heart disease	9.1
2	Cerebrovascular disease	13.7	2	Cerebrovascular disease	8.0
3	Trachea, bronchus, lung cancers	4.5	3	Lower respiratory infections	7.7
4	Lower respiratory infections	3.7	4	HIV/AIDS	6.9
5	COPD	3.1	5	Perinatal conditions	5.6
6	Colon and rectum cancers	2.6	6	COPD	5.0
7	Stomach cancer	1.9	7	Diarrheal diseases	4.9
8	Self-inflicted injuries	1.9	8	Tuberculosis	3.7
9	Diabetes	1.7	9	Malaria	2.6
10	Breast cancer	1.6	10	Road traffic accidents	2.5

COPD: chronic obstructive pulmonary disease

RISK FACTORS FOR CORONARY ARTERY DISEASE

CONVENTIONAL RISK FACTORS

1.	NON MODIFIABLE	a)	Age
		b)	Sex
		c)	Family H/o
		d)	Genetic
2.	MODIFIABLE	a)	Smoking
		b)	Alcohol intake
		c)	Atherogenic Diet
		d)	Physical Inactivity
		e)	Obesity
		f)	Diabetes
		g)	Metabolic Syndrome
		h)	Hypertension
		i)	Dyslipidemias
		j)	Mental Stress

3. NOVEL RISK FACTORS

a) C-Reactive Protein

b) Homocysteine

c) Fibrinogen and D-Dimer

d) Lipoprotein a

CAD IN INDIA

Specific mortality data ideal for making comparisons with other countries are not available in India. This is due to inadequate and inappropriate death certification, and multiple concurrent causes of death. Cardiovascular disease (CVD) is the leading cause of death in India, and its contribution to mortality is rising; deaths due to CVD are expected to double between 1985 and 2015.

Multiple studies have clearly shown that CHD is a significant problem in India and coronary risk factors: hypertension, smoking, physical inactivity, obesity and truncal obesity, and improper diet leading to hypercholesterolemia and hypertriglyceridemia are widespread.

CHD prevalence in urban populations increased from 3.5% in 1960s to 9.5% in 1990s. In rural areas it increased from 2% in 1970s to

4% in 1990s.3-4. The rates appear to be higher in South India with highest in Kerala[7].

The high rates of CAD in urban India compared to rural, despite lower rates of smoking, suggest important roles for nutritional and environmental factors. There is a significant increase in BMI in urban compared to rural (BMI 24 versus 20 in men, 25 versus 20 in women)[7].

There is also a higher rate of abdominal obesity among the urban population, with urban men having a waist to hip ratio (WHR) of 0.99 compared to 0.95 among rural men, these increases in BMI and WHR result in significant insulin resistance and dyslipidaemia[7]. Urban-rural differences in prevalence of coronary risk factors also provide important information regarding risk factors that need prevention. These include sedentary life style, hypertension, BMI, WHR / truncal obesity, total and LDL Cholesterol/ hypercholesterolemia, triglyceride levels, fasting insulin levels/ insulin resistance etc.

Based solely on projected demographic trends, it has been estimated that deaths attributable to CAD would nearly double, in both sexes, in the period 1985-2015 and CAD would emerge, over this period, as the single largest contributor to mortality, accounting for nearly a third of all deaths, globally[7].

FACTORS CONTRIBUTING TO THE ACCELERATION OF CAD EPIDEMIC IN INDIA

(i) Demographic transition to an older population, as a result of increasing life expectancy

(ii) Confluence of both conventional risk factors and non-conventional risk factors in Indians. Conventional factors like hypertension, diabetes, hypercholesterolemia, smoking etc. owe their origin to growing urbanization and western 'acculturation' amongst Indians. Non conventional risk factors like hyperinsulinaemia, insulin resistance, lipoprotein A etc are determined by genes or other 'programming' factors and their high prevalence amongst Indians probably explain the malignant, precocious nature of CAD that typically affects Indians.

(iii) Recently indicated relationship between low birth-weight which is widely prevalent amongst Indian newborns and enhanced susceptibility to CAD in adult life ('Barker hypothesis'). These multiplicative effects of conventional and emerging risk factors appear to provide a plausible explanation for the excess burden of CAD among Indians, many of whom are lean, non-smoking, vegetarian, yoga guru and marathon athletes.

The excess risk of CAD in Indians appears to be greater at younger ages. When people move from a rural to an urban environment, they become sedentary and/or may adopt western lifestyles. Decreased physical activity and increased consumption of calories and saturated fat result in abdominal obesity, insulin resistance and atherogenic dyslipidaemia. These acquired metabolic abnormalities appear to have a synergistic effect on the development of CAD in genetically predisposed individuals.

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, obesity, cigarette smoking
  - Cholesterol levels: similar to Whites but higher than other Asians
- Higher prevalence of emerging (thrombogenic) risk factors
  - High levels of lipoprotein a, homocysteine, Apoprotein 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 may be defined as an abnormal growth of adipose tissue due to an enlargement of fat cell size or increase in the fat cell number or both. Although viewed as increased body weight, this need not be the case – lean but very muscular individuals may be overweight without increased obesity.

Obesity is becoming a global epidemic in both children and adults. Obesity is an independent risk factor for CVD, and CVD risks have also been documented in obese children. Obesity is associated with an increased risk of morbidity and mortality as well as reduced life expectancy. Health service use and medical costs associated with obesity and related diseases have risen dramatically and are expected to continue to rise.

Besides an altered metabolic profile, a variety of adaptations/alterations in cardiac structure and function occur in the individual as adipose tissue accumulates in excess amounts, even in the absence of co morbidities.

Hence, obesity may affect the heart through its influence on known risk factors such as dyslipidaemia, hypertension, glucose intolerance, inflammatory markers, obstructive sleep apnea/hypoventilation, and the prothrombotic state, in addition to as-yet-unrecognized mechanisms. On the whole, overweight and obesity predispose to or are associated with

numerous cardiac complications such as coronary heart disease, heart failure, and sudden death because of their impact on the cardiovascular system.

However obese individuals differ not only in the amount of excess fat that they store but also in the regional distribution of the fat within the body. The distribution of fat induced by weight gain affects the risk associated with obesity and the kind of disease it results. It is useful therefore, to be able to distinguish between those at increased risk as a result of abdominal fat distribution or "Android obesity" from those with the less serious "Gynoid fat" distribution, in which fat is more evenly and peripherally distributed around the body.

BODY FAT AND METABOLIC SYNDROME

The metabolic syndrome is a constellation of metabolic risk factors that consist of the following[3]:

1. Atherogenic dyslipidaemia [serum elevations of triglycerides Apolipoprotein B (apo B), and small low-density lipoprotein (LDL) particles plus low high-density lipoprotein (HDL) cholesterol]

2. Elevated blood pressure

3. Elevated glucose associated with insulin resistance

4. Prothrombotic state

5. Proinflammatory state

National Cholesterol Education Program Adult Treatment Panel III report proposed a simple scheme for the routine diagnosis of metabolic syndrome. According to this scheme, a diagnosis of metabolic syndrome can be made if a person has three of the following five features[3]:

1. Increased waist circumference (≥102 cm in men and ≥88 cm in women)

2. Elevated triglycerides (≥150 mg/dl)

3. Reduced HDL cholesterol (<40 mg/dl in men and < 50 mg/dl in women)

4. Elevated blood pressure (≥130/85 mm Hg or on treatment for Hypertension)

5. Elevated glucose (≥100 mg/dl)

When the waist circumference is 102 cm or more in men or 88 cm or more in women, the term abdominal obesity can be applied[3]. The advantage of measuring waist circumference is that an excess abdominal fat is correlated more closely with the presence of metabolic risk factors than total body fat.

The cut points for defining abdominal obesity are arbitrary. For susceptible individuals, lesser accumulations of abdominal fat can

precipitate or aggravate metabolic risk factors. This is particularly so in certain populations; for example, in Asian populations lower waist circumference cut points have been identified to define abdominal obesity[3].

A disputed area in the relation of obesity and metabolic syndrome concerns the role of insulin resistance. The interaction between obesity and defects in insulin signaling is so complex that it is so far not possible to disentangle the two. Obesity causes insulin resistance, whereas insulin resistance seemingly exacerbates the adverse effects of obesity. there is little doubt that increasing prevalence of overweight/obesity is mainly responsible to the rising prevalence of the metabolic syndrome.

ETIOLOGY

The cause of obesity remains elusive. This reflects the fact that obesity is 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 Neuro Endocrine and metabolic systems that regulate the energy intake, storage and expenditure, it has been difficult to quantitate all the relevant parameters over time in human subjects.

ROLE OF GENES VERSUS ENVIRONMENT : Obesity is commonly seen in families and the heritability of body weight is similar to that of height. Inheritance is usually not Mendelian, however, and it is difficult to distinguish the roles of genes and environmental factors. Identical twins have similar BMI's even when reared separately.

Whatever the role of genes, it is clear that environment plays a key role in Obesity, as evidenced by the fact that famine prevents obesity in even the most obese prone individual. Cultural factors are also important .These relate to both the availability and the composition of the diet and to changes in the level of physical activity. In the industrialized world obesity is more common among the poor women, whereas in the underdeveloped countries obesity is more common among the wealthier women. Although the role of diet composition in obesity continues to generate controversy, it appears that high fat diets may promote obesity, especially when combined with diets rich in simple carbohydrates.

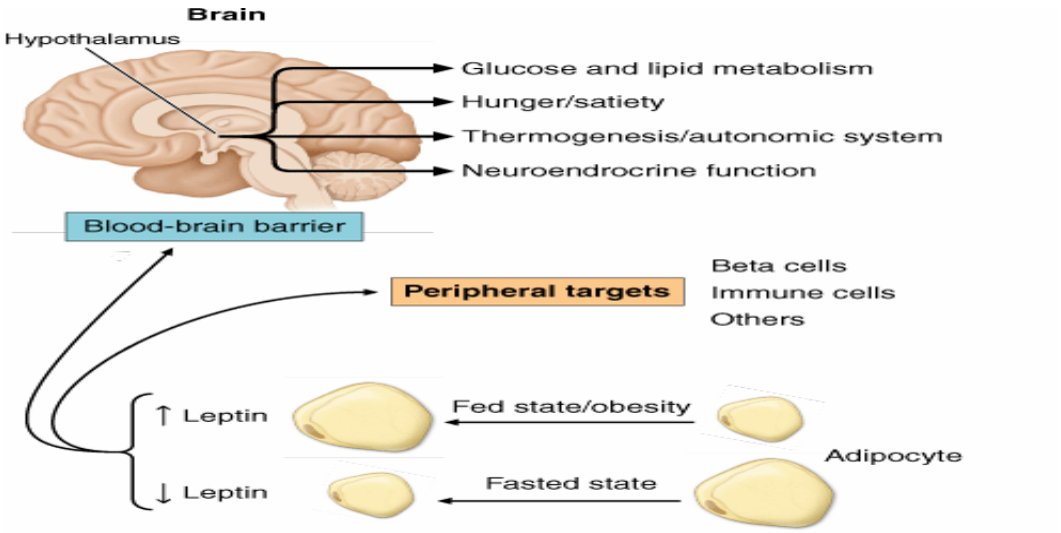
SPECIFIC GENES : A major breakthrough in this field came through the identification of the ob gene. The genetically obese mouse (ob/ ob) developed severe obesity, insulin resistance and hyperphagia as well as efficient metabolism. The product of the ob gene is the peptide LEPTIN[1].

It Acts primarily through the hypothalamus and decreases the food intake and increases the energy expenditure.

PATHOGENESIS

The adipose tissue is not simply a passive storehouse for fat but an endocrine organ that is capable of synthesizing and releasing into the bloodstream an important variety of peptides and nonpeptide compounds that may play a role in cardiovascular homeostasis. They are[2]:-

1. Leptin
2. Tumor necrosis factor-α (TNF-α)
3. Interleukin-6 (IL-6)
4. Plasminogen activator inhibitor-1
5. Resistin
6. Lipoprotein lipase
7. Acylation stimulating protein
8. Cholesteryl-ester transfer protein
9. Retinal binding protein
10. Angiotensinogen
11. Estrogens (through P450 Aromatase activity)
12. Adiponectin
13. Insulin-like growth factor-I (IGF-1)
14. Insulin-binding protein 3 (IGFBP3)
15. Monobutyrin.
16. Non Esterified Fatty Acids (NEFA)



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 17th Edition: http://www.accessmedicine.com

ROLE OF NEFA'S

Obese persons release increased amounts of NEFAs into the circulation. NEFAs are derived by lipolysis of adipose tissue triglycerides. The greater the amount of fat in adipose tissue, the more the amounts of NEFAs released will be. This greater release of NEFAs proceeds despite the higher insulin levels that are present in obese persons. Even though high insulin levels suppress adipose tissue lipolysis, they cannot reduce NEFA release to normal in obesity. NEFAs are the primary source of nutrient energy in the fasting state[3]. Excessive influx of NEFAs into muscle leads to insulin resistance[3]. The mechanisms wherein increased fatty acids in muscle cause insulin resistance have not been fully elucidated. Recent research suggests that muscle levels of diacylglycerol are raised, which stimulates the serine phosphorylation of the insulin receptors and thereby inhibits normal insulin signaling. The

resulting insulin resistance in muscle predisposes to hyperglycemia; the latter becomes clinically manifest in those persons to acquire a defect in insulin secretory capacity.

Influx of excess NEFAs into the liver increases the triglyceride content of the liver (fatty liver)[3]. Fat accumulation in the liver seemingly produces insulin resistance as it does in muscle. Reduction in insulin action in liver allows for enhanced glyconeogenesis and increased hepatic glucose output; this will accentuate hyperglycemia in those patients who have reduced insulin secretory capacity[3].

OTHER FACTORS : Adiponectin increases insulin sensitivity and lipid oxidation and has vascular protective effects. Resistin and RBP-4 increases insulin resistance. IL-6, TNF-α, other cytokines is increased in obese individuals and causes insulin resistance in adipocytes. Abdominal adipose tissue is more active in PAI-1 synthesis than lower-body adipose tissue[3]. The resulting high PAI-1 level in obese persons together with the high plasma fibrinogen observed in such persons contributes to a prothrombotic state.

ATHEROGENIC DYSLIPIDEMIA : Increased fat in the liver also promotes development of atherogenic dyslipidaemia. It provides a stimulus for increased formation and secretion of very LDL (VLDL) particles. The result is higher serum levels of triglyceride, apo B, and small LDL particles. High serum triglycerides reduce HDL-cholesterol

concentrations through exchange of VLDL triglycerides with HDL cholesterol esters. HDL-cholesterol lowering is accentuated by an increase in synthesis of hepatic lipase that occurs in people with obesity-induced fatty liver; lipase degrades HDL particles, converting large HDL into small HDL.

A theory widely held is that smaller LDL particles are more atherogenic than larger LDLs[3]. Smaller LDLs may filter more readily into the arterial wall. They further may be more prone to atherogenic modification. Even so, not all investigations are convinced that small LDL particles are unusually atherogenic, compared with other apo B-containing lipoproteins.

Nonetheless, when small LDLs are present, the total numbers of lipoprotein particles in the LDL fraction usually are increased.

CARDIOVASCULAR IMPACT OF INCREASED ADIPOSE TISSUE MASS

Obesity produces an increment in total blood volume and cardiac output that is caused in part by the increased metabolic demand induced by excess body weight. Thus, at any given level of activity, the cardiac workload is greater for obese subjects. Obese subjects have higher cardiac output and a lower total peripheral resistance than do lean individuals. The increased cardiac output is attributable mostly to increased stroke volume because heart rate increases little if at all. Also, in obesity, the

Frank-Starling curve is shifted to the left because of incremental increases in left ventricular filling pressure and volume, which over time may produce chamber dilation. Ventricular chamber dilation may then lead to increased wall stress, which predisposes to an increase in myocardial mass and ultimately to left ventricular hypertrophy, characteristically of the eccentric type.

Eccentric LVH which is commonly present in morbidly obese patients (BMI≥40 kg/m^2), is often associated with left ventricular diastolic dysfunction[2]. Moreover, as with left ventricular mass, longer durations of obesity are associated with poorer left ventricular systolic function and greater impairment of left ventricular diastolic function.

Because of the presence of nonspecific symptoms, the evaluation of the presence of left ventricular diastolic dysfunction is clinically important in obese subjects. Age and cardiac hypertrophy of the concentric or, more commonly, the eccentric type predispose to left ventricular systolic dysfunction.

In humans and most animal models, the development of obesity leads not only to increased fat depots in classic adipose tissue locations but also to significant lipid deposits in other organs. With fat gain, lipid deposition can impair tissue and organ function in 2 possible ways[2]:

1. The size of fat pads around key organs may increase substantially, modifying organ function either by simple physical compression or

because periorgan fat cells may secrete various locally acting molecules, and

2. Lipid accumulation can occur in nonadipose cells and may lead to cell dysfunction or cell death, a phenomenon known as lipotoxicity.

CLINICAL AND LABORATORY ASSESSMENT OF OBESE INDIVIDUALS

The 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 the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults (1998) are based on an increased risk of morbidity and mortality in different populations[8].

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 kg/m2)

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[9].

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 cut-off point for waist circumference of 88 cm in women and 102 cm in men and 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).

SEX	IDEAL	INCREASED RISK	GREATER RISK
M	‹94 cm	94 – 101cm	›102 cm
F	‹80 cm	80 – 87 cm	›88 cm

(Ref: Han et al , BMJ 1995 311:1401 – 5)

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)[8]. 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 Cutoff values for Waist circumference were 85 and 80 cm for men and women[9].

Measure the waist circumference as recommended by the World Health Organization[11]:

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":

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

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[8].

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)[9].

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 intra-abdominal fat and probably also of total fat than BMI when validated against computer tomography or magnetic resonance imaging [8].

Although the current recommendation seems to favor the use of waist circumference in assessing abdominal obesity, WHR remains a

suitable method for research purposes (World Health Organization 2000, p.10)[8].

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)[8]. 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)[8].

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)[8].

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)[8], and morbidity risks at the same level of overweight vary across different ethnic populations (Seidell et al. 2001a)[8]. Therefore, instead of globally accepted cut-off points for obesity, there need to be age, sex, and race-specific categories for overweight and obesity.

Nevertheless, 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.

A Skinfold Caliper is designed specifically for simple accurate measurement of subcutaneous tissue. Either a 7 or 3 site skinfold may be assessed.

7 Site skinfold

- chest

- triceps
- subscapular
- axilla
- suprailiac
- abdomen
- thigh

3 Site skinfold (Men)

- chest
- abdomen
- thigh

3 Site Skinfold (Women)

- tricep
- suprailiac
- thigh

BICEPS : The anterior surface of the biceps midway between the anterior auxiliary fold and the antecubital fossa[11].

TRICEPS : A Vertical fold on the posterior midline of the upper arm, over the triceps muscle, halfway between the acromion process (bony process on top of the shoulder) and olecranon process (bony process on elbow). The elbow should be extended and the arm relaxed[11].

SUBSCAPULAR : The fold is taken on the diagonal line coming from the vertebral border to between 1 and 2 cm from the inferior angle of the scapulae. (A diagonal fold about 1 to 2 cm below the point of the shoulder blade and 1 - 2 cm toward the arm)[11].

SUPRAILIAC : A diagonal fold above the crest of the ilium at the spot where an imaginary line would come down from the anterior auxiliary line just above the hip bone and 2 - 3 cm forward[11].

CHEST (JUXTA-NIPPLES) : A diagonal fold taken one half of the distance between the anterior auxiliary line and the nipple. (The anterior auxiliary line is the crease where the top of the arm, when hanging down, meets the chest.)[11]

ABDOMINAL : The vertical fold taken at the lateral distance of approximately 2 cm from the umbilicus (2cm to the side of the umbilicus)[11].

THIGH : A vertical fold on the anterior aspect of the thigh, midway between the hip and knee joints (on the front of the thigh halfway between the hip joint, where the leg bends when the knee is lifted, and the middle of the knee cap). The leg should be straight and relaxed)[11].

If each test is performed correctly according to the recommended guidelines, there is a +/- 3% error. Validity 7 site skinfold (r = .90), 3 site skinfold (r = .89)[15].

Advantages

- Easy to use once skill has been mastered
- Does not require much time
- Noninvasive 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

A major source of error in measuring skinfold fat is variation in the placement of the skinfold caliper at the selected anatomical site. The subcutaneous fat variability can be a problem when measurements at one or several sites are used to represent overall body fat composition. These measurements overall are substantially less reproducible than most other anthropometric measurements. Several investigations have suggested that truncal obesity may have a greater correlation to carbohydrate and lipid metabolism disorders and hypertension compared to peripheral obesity.

Because of the increased errors involved, it is usually not appropriate to convert skinfold measure to percentage body fat (%BF). It

is best to use the sum of several sites to monitor and compare body fat measures[15].

Although measurement errors in skinfold thickness tended to increase with increasing obesity levels, the influence was smaller for the abdominal and suprailiac skinfolds compared with other sites.

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[12]:

- 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 produce 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[12].

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

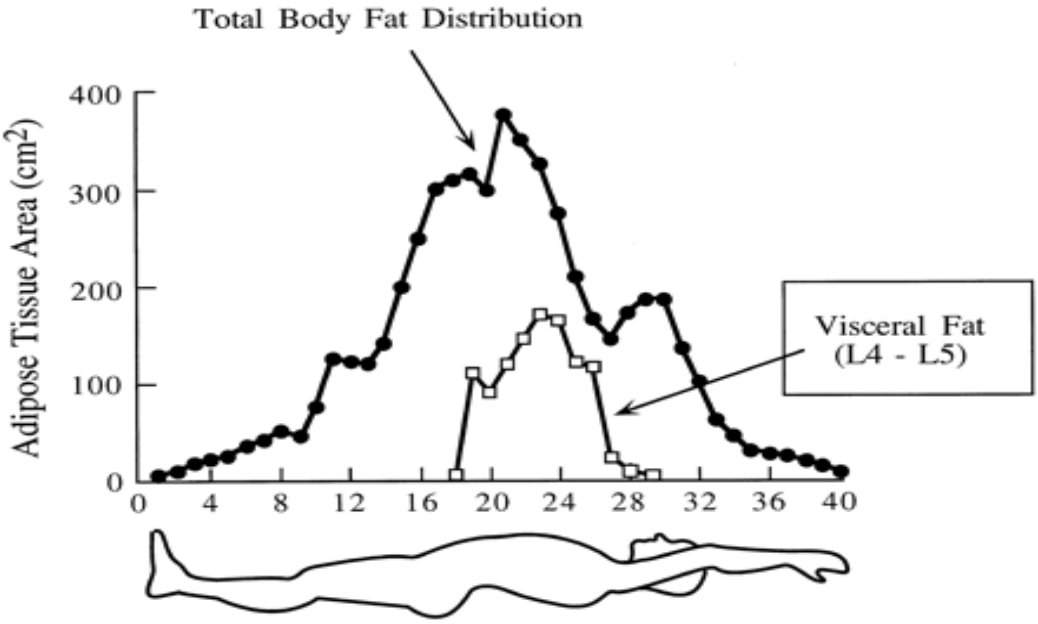
DEXA is costly

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[12].

MAGNETIC RESONANCE IMAGING:-

Cross-sectional abdominal images obtained with MRI, as shown in Figure, 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[12].

Fig : Distribution of total body fat and visceral fat in an adult, nonobese male obtained using magnetic resonance imaging.



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[12].

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[11].

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

TREATMENT:-

DIAGNOSTIC APPROACH:-

- Calculate body mass index and measure waist circumference.
- Determine degree and rate of acquisition of obesity.
- Exclude identifiable causes of obesity.
- Assess co morbid conditions, presence of cardiovascular risk factors, and absolute risk status.

- Conditions that indicate high absolute risk for obesity-related disorders
- Established coronary artery disease
- Other atherosclerotic disease
- Type 2 diabetes mellitus
- Sleep apnea
- ≥3 of the following indicate high absolute risk for obesity related disorders.
- Hypertension
- Cigarette smoking
- High low-density lipoprotein cholesterol level (>160 mg/dL)
- Low high-density lipoprotein cholesterol level (<35 mg/dL)
- Impaired fasting glucose
- Family history of early coronary artery disease
- Age > 45 in men and age > 55 in women.

TREATMENT APPROACH

Patients who meet the following criteria should be considered for treatment.

- BMI >30 mg/kg^2
- BMI 25–29.9 mg/kg^2 and presence of ≥2 risk factors
- BMI 25–29.9 mg/kg^2 and waist circumference >102 cm (40 in) in men or >88 cm (35 in) in women
- Combined therapy with a low-calorie diet, increased physical activity, and behavior therapy provide the most successful intervention for weight loss and weight maintenance.
- All patients should be counseled on lifestyle and behavioral modifications (appropriate diet and physical activity), and weight loss goals should be individualized.
- Treatment goals should be guided by the health risks of obesity in any given person.

Specific Treatments

Behavior modification

- The principles of behavior modification provide the underpinnings for many current programs of weight reduction.
- Goal of behavior modification is to modify maladaptive behaviors, including eating habits and physical activity.
- Patient is asked to monitor and record the circumstances related to eating and physical activity.

Dietary therapy

- Reduced caloric intake is the cornerstone of obesity treatment.
- There is no scientific evidence to validate the utility of specific "fad diets."
- General facts relevant to food intake and weight loss
- Deficit of 7,500 kcal will produce a weight loss of ~1 kg.
- Consuming 100 kcal/d less for 1 year should cause a 5-kg weight loss.
- Consuming 1,000 kcal/d less should cause a loss of ~1 kg per week.

- Rate of weight loss on a given caloric intake is related to rate of energy expenditure.

- Obese persons have a higher metabolic rate than lean persons.

- Men have a higher metabolic rate than women (due to their greater lean body mass); thus, the rate of weight loss is greater among more obese and among men.

- With chronic caloric restriction, the metabolic rate decreases.

- With total starvation or diets restricted to < 600 kcal/d, initial weight loss over the first week results predominantly from natriuresis and the loss of fluids.

Very-low-calorie diets (400–600 kcal/d)

It may be appropriate for short-term treatment of obesity in selected patients.

- Use should be restricted to patients >130% of ideal body weight.

- Most commonly used for 1–2 months to initiate more rapid weight loss, improve co morbid conditions, and provide patients with positive feedback

- Safe in selected patients under medical supervision

Daily diet composition

- 45–70 g high-quality protein
- 30–50 g carbohydrate
- 2 g fat
- Supplements of vitamins, minerals, and trace elements

Advantages

- Greater rate of weight loss compared with less restrictive diets
- Possible beneficial effect of hunger suppression from production of ketones
- Blood pressure and blood glucose, cholesterol, and triglyceride levels decrease.
- Pulmonary function and exercise tolerance improve.
- Sleep apnea may improve within a few weeks.

Complications

Minor

- Fatigue, constipation or diarrhea

- Dry skin
- Hair loss
- Menstrual irregularities
- Orthostatic dizziness
- Difficulty concentrating
- Cholelithiasis and pancreatitis may occur when a very-low-calorie diet is interrupted by binge eating.
- Gallstones develop in up to 25% of patients on a very-low-calorie diet.
- Close supervision is required in patients with diabetes who are receiving insulin or oral agents.

Contraindications

- Pregnancy
- Cancer
- Recent myocardial infarction
- Cerebrovascular disease
- Hepatic disease

- Untreated psychiatric disease
- Patients may benefit from counseling offered in a stable group setting for extended periods of time, including after weight loss.

Low-calorie diets (>800 kcal/d)

- Are applicable to most patients and have fewer restrictions than very-low-calorie diets.
- Considerable controversy surrounds the question of what constitutes the best diet composition for promoting weight loss.

Low-fat diets

- Commonly recommended, but benefits to obesity reduction from very-low-fat diets are modest at best
- Health effects aside from weight reduction may be important.
- Large amounts of simple carbohydrates are substituted for fats but may actually promote obesity.
- Diets rich in fruits, vegetables, whole grains, and other low–glycemic index carbohydrates may promote weight loss and are preferable to low-fat diets.

- Diets with protein replacement of simple carbohydrates aim to minimize insulin production.

- Efficacy of this strategy, aside from overall calorie reduction, is unknown.

Very-low-carbohydrate "Atkins" style diets

- More effective for short-term weight loss when compared to standard caloric restriction, but have not been shown to be more effective in maintaining weight loss

- Consequences of maintaining a lower body weight at the expense of consuming more saturated fat are unknown.

- Diet therapy education is important to prevent weight regain.

- Patients should be counseled on the caloric content of specific portions to promote weight loss and maintenance.

Exercise

- Physical activity is an important component of the overall approach to weight reduction and maintenance.

- The effect of an exercise regimen as a sole therapy for obesity is not established, but exercise is a valuable means to sustain diet therapy.

- **Additional benefits**

  - Improves cardiovascular tone and reduce blood pressure, independent of weight loss

  - Helps reduce appetite

  - Increases the likelihood of weight maintenance once targets are achieved

  - Reduces intra-abdominal fat

  - Reduces risk of glucose intolerance

- Many obese persons have not exercised on a regular basis and may have cardiovascular risk factors.

  - Exercise should be introduced gradually under medical supervision, especially in the most obese patients.

- Minimal physical activity recommendations

  - Adults should engage in moderate-intensity physical activities for ≥ 30 minutes on ≥ 5 days of the week **or**

  - Adults should engage in vigorous-intensity physical activity ≥ 3 days per week for ≥ 20 minutes per occasion.

Pharmacotherapy

- May be considered as adjunctive therapy in patients with a BMI ≥30 kg/m^2 or ≥27 kg/m^2 with other risk factors or diseases who fail to achieve weight loss goals through non pharmacologic approaches

- **Limitations**

- Medication-induced weight loss is not a cure despite modest short-term benefits from several agents.

- Safety and efficacy of weight loss agents beyond 2 years has not been established.

- Rebound weight gain after the cessation of drug use is common.

- Most agents are associated with substantial side effects, and some have potential for abuse.

- **PHENTERMINE :** approved for short-term use (<12 weeks).

o Mechanism of action: increases the release of nor epinephrine and dopamine from nerve terminals and inhibits their reuptake

o Dosing: 15 mg–30 mg/d

- Efficacy: modest (10 versus 4.4 kg of weight loss over 24 weeks in a well-controlled study)

- Side effects (numerous): insomnia, dry mouth, constipation, palpitations, hypertension

- **SIBUTRAMINE:** approved for long-term use.

- Mechanism of action: central reuptake inhibitor of both nor epinephrine and serotonin

- Dosing: 10 mg/d

- Efficacy: Once-daily dose over 24 weeks produced a 7% weight loss and decreased cholesterol and triglyceride levels in a double-blind, placebo-controlled trial.

- Adverse effects: increases pulse by an average of 4–5 beats/min and blood pressure by 1–3 mmHg

- **ORLISTAT :** approved for long-term use.

- Mechanism of action: inhibitor of pancreatic lipase with no systemic availability; causes modest weight loss due to drug-induced fat malabsorption

- Dosing: 120 mg 3 times daily, before meals, taken with a multivitamin

- Efficacy: 2-year randomized, double-blind trial revealed modest weight loss (8.7 kg for 120 mg of orlistat versus 5.8 kg from diet alone) during first year and better maintenance of weight loss in second year compared with the placebo group (3.2 kg regained vs. 5.6 kg regained for placebo)

- **Adverse effects**

- GI side effects include oily stools, flatulence, and fecal urgency; these usually diminish as patients limit fat intake to avoid symptoms.

- Absorption of fat-soluble vitamins is decreased.

- **METFORMIN** tends to decrease body weight in patients with obesity and type 2 diabetes mellitus.

- **RECOMBINANT LEPTIN**

- Is highly effective in rare cases of Leptin deficiency caused by mutations of the Leptin gene.

- Regulates hunger and induces loss of fat mass while preserving lean body mass.

- Response to Leptin is limited or absent in common obesity, which is associated with hyperleptinemia and Leptin resistance.

Surgery

- Bariatric surgery should be considered for patients meeting the following criteria.
- BMI >35 kg/m^2 with an associated co morbid condition or BMI >40 kg/m^2
- Repeated failure of other therapeutic approaches
- At eligible weight for 3–5 years
- Capable of tolerating surgery
- Absence of alcoholism, other addictions, or major psychopathology
- Prior clearance by a psychiatrist

- **Vertical-banded gastroplasty**

- Purely restrictive procedure

- **Roux-en-Y gastric bypass**

- Combines restriction with slight malabsorption

- May also reduce appetite via suppression of gastric hormone ghrelin
- Most often performed via laparotomy, but may be performed laparoscopically
- May be associated with risk of islet hyperplasia and hypoglycemia

- Laparoscopic adjustable gastric banding

Potential benefits

- Significant, sustained weight loss (10–159 kg over 1–5 years)
- Improvements in hypertension, diabetes, sleep apnea, congestive heart failure, angina, hyperlipidemia, and venous disease

Short-term complications

- Pulmonary embolus
- Anastomotic leak
- Bleeding
- Wound infection

Long-term complications depend on the specific surgical procedure but include:

- Dumping syndrome
- Stomal stenosis
- Marginal ulcers
- Hernias
- Lifelong medical monitoring after surgery is necessary.
- Lifelong supplementation with vitamin B_{12}, iron, folate, and calcium is recommended.

MATERIALS AND METHODS

PLACE OF STUDY : This is a standardized case control study conducted in the Government General Hospital and Madras Medical College, Chennai from November 2007 to November 2008.

INCLUSION CRITERIA

1) Consecutive cases of newly diagnosed Myocardial Infarction.

2) With/ without risk factors for IHD.

EXCLUSION CRITERIA

1) Old cases of Myocardial infarction/ unstable angina/ chronic stable angina.

2) Cardiogenic shock

3) Major Chronic diseases

4) Age > 60 yrs due to non reliability of skin fold thickness.

STUDY POPULATION

A total of 98 cases (49 Cases and 49 Controls) were selected. Newly diagnosed consecutive cases of myocardial infarction admitted to the department to the department of cardiology were enrolled into the study after obtaining consent.

These patients were subjected to anthropometric measurements namely waist hip ratio, weight height, skin fold thickness and S.Lipid profile was done. Controls were selected from the outpatient clinic of Institute of Internal Medicine. Controls were matched for age, sex, risk factors. They were also subjected to anthropometric measurements and S.Lipid profile.

STUDY DESIGN

Standardised Case Control Study, to assess whether markers of obesity especially waist hip ratio would be stronger indicators of myocardial infarction than body mass index, the conventional measure.

METHODOLOGY

1. BODY MASS INDEX

Weight was measured in KG's. The weighing apparatus was calibrated at the beginning of each and at the end of each stand. Weight was determined on a balance scale in light clothing to an accuracy of 100g.

Height was measured using Stadiometer in cm. Height was measured without shoes by a measuring tape against a wall to an accuracy of 0.1 cm with patient looking straight ahead, shoulders relaxed, arms by sides legs straight and knees together. Shoulder blades, buttocks and heels touching the measurement surface.

BMI was calculated using the following formula

BMI = Weight (in Kg) / Height in metre^2

The study population was divided into two groups < 25 and ≥25 (World Health Organization 2000, p. 9-11).

2. WAIST HIP RATIO

Waist circumference was measured over unclothed abdomen at the midpoint between the iliac crest and the lowest rib. It was calculated as an average of one measurement taken after inspiration and one taken after expiration.

Hip circumference was measured over light clothing at the level of the Tronchanteric major or widest diameter around buttocks.

All anthropometric measurements were carried out once.

WHR was calculated as waist divided by hip, and was used in the analyses either as a continuous variable or divided into sex-specific thirds.

3. SKIN FOLD THICKNESS

All the skin folds were taken with Vernier calipers. The measurements are taken on the right side of the body. The fold of skin

and underlying subcutaneous adipose tissue were gently grasped between the left thumb and forefingers.

Enough skin and adipose tissue was grasped to form a distinct fold that separates from the underlying muscle. The skinfold was grasped 2.0 cm above the place, the measurement taken .The jaws of the calipers were placed at the marked level, perpendicular to the length of the fold, and the skinfold thickness was measured to the nearest 0.1 mm while the fingers continue to hold the skinfold. Skin fold measurements were taken at 6 sites namely Triceps, Biceps, Thigh, Subscapular, Suprailiac and Abdomen. The measurements were divided into two groups Central fat Mass (CFM) and Peripheral fat mass (PFM). For the assessment of CFM the sum of Abdominal, Suprailiac and Subscapular were used and for the assessment of PFM the sum of Triceps, Biceps and Thigh were used. The ratio of CFM to PFM was analysed in both control and study group, sex wise (European Journal of Endocrinology, Volume 156, Issue 6, 655-661).

4. SERUM LIPID PROFILE

For venous blood specimen, participants were advised overnight fasting and blood sample drawn the other day morning and sent for analysis. Total cholesterol, HDL, Triglycerides were analysed from the serum blood samples and LDL was calculated using the following formula:-

LDL = TC –TGL/5 – HDL.

Consent	:	Obtained
Financial support	:	Nil
Ethical committee clearance	:	Obtained
Conflict of interest	:	Nil

RESULTS

The results of clinical evaluation were shown below. In our study, the study population was divided into IHD and Non IHD groups. Statistical analysis was carried out in 98 patients after categorizing each variable. Age, BMI, Waist hip ratio, sum of central skin fold thickness, sum of peripheral skin fold thickness, LDL in analysed in IHD and non IHD patients.

Statistical analysis was carried out using standard formulae. Microsoft excel 2003 and SPSS (statistical package for social sciences) version 13.0 softwares were used for data entry and analysis.

BMI, Waist hip ratio, LDL were analysed using Chi – Square test. The significance of difference in mean in means for sum of central and peripheral skin folds thickness were calculated using t tests. Statistical significance is taken when $P < 0.05$.

Both the groups were matched for age, sex and risk factors other than the one studied, namely smoking, alcoholism, diabetes, hypertension, family H/o. There is no statistical difference is noted between the two groups in age, sex, smoking, alcoholism, Hypertension, Diabetes, Family H/o. In all these parameters compared between these groups the P value is more than 0.05 which is statistically insignificant.

TABLE 1

AGE DISTRIBUTION

Age Group (in years)	Cases and Controls	
	Number	Percentage
≤ 30	6	6.74
31-40	14	14.28
41-50	38	38.77
51-60	40	40.81
Total	98	100
Mean Standard deviation	47.54 yrs 8.4yrs	

The Mean age of patients in our study was 47.54±8.4yrs. Majority of patients were in the 4^{th} to 5^{th}decade at the time of presentation.

TABLE 2

SEX DISTRIBUTION

Table 2 shows sex distribution of the study. Majority of the patients were males. Male to female ratio was 5:1

SEX	CASES	CONTROLS	TOTAL	PERCENTAGE
MALE	41	41	82	83.7%
FEMALE	8	8	16	16.3%
TOTAL	49	49	98	100%

TABLE 3
RISK FACTORS

RISK FACTOR	CASES	PERCENTAGE	CONTROLS	PERCENTAGE
SMOKING	32	65.3%	32	65.3%
ALCOHOL INTAKE	27	55.1%	27	55.1%
DIABETES	10	20.4%	10	20.4%
HYPERTENSION	11	22.4%	11	22.4%
FAMILY H/O IHD	11	22.4%	11	22.4%
NO RISK FACTORS	8	16.32%	8	16.32%

Table 3 shows the risk factors. Majority of the patients had atleast one risk factor. 8 of them had no risk factors.

TABLE 4

BODY MASS INDEX (BOTH MALE& FEMALE)

GROUPS	BMI	NUMBER	PERCENTAGE	P VALUE
CASES	< 25	40	81.6%	0.13648
	≥ 25	9	18.4%	
CONTROLS	< 25	45	91.8%	
	≥ 25	4	8.2%	

Table 4 shows BMI. It is statistically insignificant and shows a weaker association between IHD and BMI. The mean of the BMI in Females were comparable (22.12 in Cases, 21.82 in Controls) with that of males (22.17 in Cases, 21.48 in Controls).

TABLE 5

WAIST HIP RATIO FOR MALES

WAIST HIP RATIO	CASES	%	CONTROLS	%	P VALUE
NORMAL (≤ 0.88)	3	10.7%	25	89.3%	0.001
ABNORMAL (> 0.88)	38	70.4%	16	29.6%	

TABLE 6

WAIST HIP RATIO FOR FEMALES

WAIST HIP RATIO	CASES	%	CONTROLS	%	P VALUE
NORMAL (≤ 0.81)	0	0%	2	100%	0.13
ABNORMAL (> 0.81)	8	57.1%	6	42.9%	

Table 5 & 6 shows waist hip ratio. For Males it was statistically significant, females it was statistically insignificant. 70.4% had abnormal ratio in cases than in controls (30%) in Males. In females 57.1% had abnormal ratio in cases than in controls (42.9%).Mean of WHR among males between the two groups were 0.97 (cases) and 0.87 (controls). Among the females the mean between the two groups were 0.96 (cases) and 0.87 (controls).

TABLE 7

WAIST CIRCUMFERENCE FOR MALES

WAIST	CASES	%	CONTROL	%	P VALUE
NORMAL ≤ 85 cm	19	38%	31	62%	0.006
ABNORMAL > 85 cm	22	68.8%	10	31.3%	

TABLE 8

WAIST CIRCUMFERENCE FOR FEMALES

WAIST	CASES	%	CONTROL	%	P VALUE
NORMAL ≤ 80 cm	3	37.5%	5	62.5%	0.31
ABNORMAL > 80 cm	5	62.5%	3	37.5%	

Table 7 & 8 shows the waist circumference. It was statistically significant in males, but statistically insignificant in females. Cases had higher waist circumference. The mean of WC among males between the two groups were 85.24 (cases) and 78.41 (controls). Among the females, between the two groups it was 85.52 (cases) and 78.39 (controls).

TABLE 9

RATIO OF CENTRAL TO PERIPHERAL

SKIN FOLD THICKNESS FOR MALES

RATIO	NUMBER	MEAN	SD	P VALUE
CASES	41	1.3098	0.223	0.872
CONTROLS	41	1.3098	0.234	

TABLE 10

RATIO OF CENTRAL TO PERIPHERAL

SKIN FOLD THICKNESS FOR FEMALES

RATIO	NUMBER	MEAN	SD	P VALUE
CASES	8	1.1955	0.230	0.825
CONTROLS	8	1.1749	0.121	

TABLE 11

CENTRAL AND PERIPHERAL SKIN FOLD THICKNESS IN MALES

GROUP	SUM OF CENTRAL SKIN FOLDS		SUM OF PERIPHERAL SKIN FOLDS	
	MEAN	SD	MEAN	SD
CASES	4.81	1.14	3.71	0.9
CONTROLS	3.47	1.06	2.68	0.71
P VALUE	0.001		0.001	

TABLE 12

CENTRAL AND PERIPHERAL SKIN FOLD THICKNESS IN FEMALES

GROUP	SUM OF CENTRAL SKIN FOLD THICKNESS		SUM OF THE PERIPHERAL SKIN FOLD THICKNESS	
	MEAN	SD	MEAN	SD
CASES	6.21	1.5	5.37	1.72
CONTROLS	4.65	1.52	4.05	5.37
P VALUE	0.058		0.135	

TABLE 13

SUM OF ALL SKIN FOLD THICKNESS IN BOTH SEXES

GROUP	MALES		FEMALES	
	MEAN	SD	MEAN	SD
CASES	8.52	1.91	11.58	3.11
CONTROLS	6.15	1.69	8.7	3.1
P VALUE	0.001		0.085	

Table 9 to 13 shows the skin fold thickness between the two groups for males and females respectively.

The skin fold thicknesses were more in females than in males with the mean of sum of the central skin fold thickness in females (6.21) more than males (4.81) and the mean of sum of the peripheral thickness in

females (5.37) more than males (3.71). The skin fold thicknesses were more in cases than controls. The mean of the sum of central skin fold thickness was more in cases (4.81 in Males, 6.21 in Females) than in controls (3.47 in males, 4.65 in females). The mean of the sum of peripheral skin fold thickness was more in cases (3.71 in Males, 5.37 in females) than in controls (2.68 in Males, 4.05 in Females).

Central skin fold thickness was more than the peripheral skin fold thickness. The mean of the sum of CENTRAL SKIN FOLD THICKNESS was more in CASES (4.81 in males, 6.21 in Females) and in CONTROLS (3.47 in Males, 4.65in Females) than the mean of the sum of the PERIPHERAL SKIN FOLD THICKNESS in CASES (3.71 in Males, 5.37 in females) and in CONTROLS (2.68 in Males, 4.05 in Females).

TABLE 14

LDL FOR BOTH SEXES

LDL	CASES	%	CONTROLS	%	P VALUE
< 130 mg/dl	16	28.1%	41	71.9%	0.00
≥ 130 mg/dl	33	80.5%	8	19.5%	

TABLE 16

CORRELATION BETWEEN LDL, WAIST CIRCUMFERENCE, WH RATIO, BMI, SKIN FOLD THICKNESS FOR MALES

	BMI	WH RATIO	WC	SUM OF ALL SKIN FOLDS	CFM	CFM TO PFM RATIO
LDL	0.187	0.001	0.065	0.001	0.001	0.936

Table 16 shows correlation between the Waist hip ratio correlated well with LDL in males which is statistically significant. Sum of skin fold thickness and Central skin fold thickness correlated with LDL with a statistical significance. But the ratio of central to peripheral skin folds did not correlate with LDL.

TABLE 17

CORRELATION BETWEEN LDL, WAIST CIRCUMFERENCE, WH RATIO, BMI, SKIN FOLD THICKNESS FOR FEMALES

	BMI	WH RATIO	WC	SUM OF ALL SKIN FOLDS	CFM	CFM TO PFM RATIO
LDL	0.739	0.268	0.080	0.019	0.014	0.958

Table 16 shows correlation between the Waist hip ratio did not correlate with LDL in females. Sum of skin fold thickness and Central skin fold thickness correlated with LDL with a statistical significance. But the ratio of central to peripheral skin folds did not correlate with LDL.

TABLE 18

CORRELATION BETWEEN WH RATIO AND SKIN FOLD THICKNESS

	CFM
WH RATIO	MALES - 0.001 FEMALES – 0.70

Table 18 WH Ratio correlated with CFM in males but with poor correlation in females.

DISCUSSION

In the western population numerous studies are being conducted on the association of obesity with cardiovascular risk.In India clinical regarding obesity and its role in Ischemic Heart disease are limited. This study is an attempt to emphasize the magnitude of the problem and to highlight the importance of early detection and treatment.

COMPARABLE STUDIES

1. INTERHEART STUDY, Lancet 2005; 366

A Standardized case control study was performed in 15152 cases of first myocardial infarction and 14820 age matched and sex matched controls. The main purpose of the study was to assess whether markers of obesity especially waist to hip ratio would be a stronger indicator of myocardial infarction than body mass index.

BMI was only slightly higher or there was no difference between the cases and controls. Cases had a higher waist to hip ratio than controls, an observation consistent in all the regions of the world. The risk of myocardial infarction rose progressively with increasing values for waist hip ratio.

Waist circumference was strongly related to myocardial infarction risk and this relation was continuous and persisted even after adjustment

for BMI and Height. Waist circumference was intermediate between BMI and waist hip ratio.

A trend towards lower risk of myocardial infarction was noted as hip circumference increased. This trend was highly significant after adjustment for BMI and height.

Of the two measures compared, BMI showed the weakest association with myocardial infarction risk in all ethnic groups with no significant relation in south Asians.

OUR STUDY : BMI showed weak association with Myocardial infarction. Waist hip ratio was strongly correlated with IHD overall (P value of 0.001). Cases had higher waist hip ratio than controls overall [70.4% of cases had abnormal WH ratio when compared to controls (29.6%)]. Waist circumference was strongly correlated with IHD overall (0.0040). 67.5% of the cases had abnormal waist circumferences than controls (32.5%). Waist circumference was in between waist hip ratio and BMI. All these go with our study also.

2. British journal of nutrition 2001 Vol 86: BMI does not accurately predict overweight in Asian Indians in northern India (AIIMS, NEW DELHI)

The main purpose of the study was to establish appropriate cut-off levels of the BMI for defining overweight, considering percentage BF in

healthy Asian Indians in northern India as the standard. A total of 123 healthy volunteers (eighty-six males aged 18±75 years and thirty-seven females aged 20±69 years) participated in the study.

Two definitions of overweight were used. BMI >25 kg/m2 and BF >25 % in males and >30 % in females were used as twin criteria. A high waist hip ratio was defined as >0.95 in males and >0.80 in females. The sum of skinfold thickness was defined as high when the value exceeded 50 mm. A high waist circumference was defined as 102 cm in males and 88 cm in females.

The mean BMI of females was higher than that of males (P <0.029).The waist circumference was similar in males and females (P <0.367). But the waist hip ratio was significantly higher in males (P <0.001): The sum of skinfold thickness was higher in females than in males (P <0.001). Similarly, peripheral and central skinfold thickness was higher in females (P <0.001 and P <0.048 respectively).

OUR STUDY : BMI of males and females were equal. Waist hip ratio was significant in males (P value 0.001) but not significant in females (P value 0.31). Waist hip ratio was higher in males than in females. Both Central and peripheral skin fold thickness were more in Females than in males. Sum of the skin fold thickness were more in females than males.

3. Relation of anthropometry to CVD risk factors in young obese women: Biomedical Research 2005; 16 (2): 137-141:-

The main objective of the study was to know the correlation of obesity as determined by anthropometry (height, weight, triceps skinfold thickness, hip and waist circumference) to altered blood lipid levels, glucose, fibrinogen and Blood Pressure (BP) in nulliparous women of 18-26 years. It involved 50 women in the age group of 18-26 years of which, twenty five women with BMI $\geq$ 25 kg/m^2 constituted the test group while twenty five women whose BMI was < 25kg/m^2 were taken as controls. All the women selected were nulliparous, non-diabetic and healthy subjects.

A significant increase in BMI, waist circumference, hip circumference and skinfold thickness was observed in the obese group. However WHR was not found to be statistically significant. Significant increase in the both the Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) was observed in the obese group. Most important finding was a significant decrease in HDL-C levels in the test group. Paradoxically TC was also found to be lesser in the test group. There was no statistically significant difference between the mean values of TG, LDC -C or Non HDL-C of the two groups.

A significant negative correlation of BMI with HDL–C and TG (r = -0.415; -0.402 respectively) was observed in the test group.

OUR STUDY : WH Ratio was statistically insignificant in Females which goes with this study.

4. Journal of Clinical and Diagnostic Research. 2008Aug (2) Anthropometric Profile in Normolipidaemic Myocardial Infarction Patients in South Asia

The objective of the study was to evaluate the changes in anthropometric variables in Normolipidaemic acute myocardial infarction (AMI) patients, and to determine the significance of waist-hip ratio and basal metabolic index in assessment of risk of myocardial infarction as compared to normal healthy controls. A ratio of ≥ 0.85 for women and ≥ 0.9 for men was considered for the prediction of risk.

Abdominal fat was found to be the strongest predictor of cardiovascular complications in subjects whose W/H ratio was in the top quartile (>0.98 for men and >0.091 for women). The estimated percentage rate of coronary heart disease (CHD, $p<0.01$) and death ($p<0.01$), myocardial infarction ($p<0.01$), stroke ($p<0.01$), and total CVD ($p<0.01$), increased with the increasing quartile of W/H ratio in both men and women. It was concluded that Asian Indians have a higher cardiovascular risk, even when BMI and WC values are within normal range, and suggested that the definitions of "normal" ranges of BMI and WC need to be revised for Asian Indians and Waist hip ratio is a useful

predictor of CVD than BMI. Waist-hip ratio is a useful predictor of CVD than BMI.

OUR STUDY : Abdominal fat was found to be the strongest predictor of cardiovascular events with increased waist hip ratio in cases than in controls. 70.4% of cases had high waist hip ratio when compared to controls (30%) which also concludes that Indians have a higher risk of cardiovascular risk even when BMI, WH ratio was normal.

5. 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:-

Optimal limit of body mass index (BMI) for Asian Indians remains to be defined. In this study, we describe the anthropometric and lipid profiles and determine the appropriate cut-offs of BMI for defining obesity in dyslipidaemic patients. Correlations were carried out between lipid profile and anthropometric variables in 217 dyslipidaemic Asian Indians and the data were compared to those of 123 healthy historical controls. Receiver operating characteristics (ROC) curve analysis was carried out to determine the appropriate cut-offs of BMI for defining obesity taking the percentage of body fat (% BF) as the standard.

Values of sigma 4 SF ($P<0.001$), and percentage of BF ($P<0.001$) were significantly higher in females. The central to peripheral skinfolds ratio were higher in men as compared to women ($P<0.001$).

Significantly higher mean values of other variables in dyslipidaemic subjects were; WC (P<0.05 in both genders), sigma 4SF (P<0.05 in both genders), central skinfolds (P<0.05 in both genders), central to peripheral skinfolds ratio (P<0.05 in males and P=NS in females).

OUR STUDY : Ratio of the skin fold thickness were more in males than females (Mean – 1.3058, Females 1.1852, P value 0.048 which was statistically significant at 5% level). The ratio of central to peripheral skin fold thickness was insignificant in both males and females and did not correlate with LDL which goes against the above study. LDL correlated with WH ratio in males but not in females. LDL also correlated with Central skin folds and the sum of all the skin folds in both the sexes. All these go with our study also.

CONCLUSIONS

1. The mean BMI of the south Indian males was 21.82 and that of south Indian females was 21.97.

2. BMI does not properly define obesity and the risk of cardiovascular events. BMI can be normal in a patient with cardiovascular disease.

3. Abdominal obesity is an independent risk factor for coronary heart disease. Waist hip ratio and waist circumference are better indicators of the cardiovascular risk in a given individual than BMI.

4. Waist hip ratio is a significant factor in males but not in females.

5. The central skin fold thickness was significant in males but not in females.

6. For the same BMI, females had more uniform distribution of fat than males who had predominantly more distribution of fat in abdominal region.

7. Central obesity leads to atherogenic lipid profile in both the sexes and places the individual at high risk of cardiovascular events.

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PROFORMA

Name:-

Age:-

Sex:-

Address:-

Occupation:-

Smoker/ Not:-

Alcoholic/ Not:-

H/o Diabetes:-

H/o HT:-

Family H/o of IHD, Diabetes:-

O/E: - Height:-

Weight:-

BMI:-

Waist Circumference:-

Hip Circumference:-

Waist Hip Ratio:-

Skin Fold Thickness:-

1) Triceps:-
2) Biceps:-
3) Subscapular:
4) Supra-iliac:-
5) Abdomen:-
6) Thigh:-