" A STUDY OF CORRELATION BETWEEN BODY MASS INDEX, WAIST CIRCUMFERENCE, BLOOD PRESSURE AND LIPID PROFILE IN TYPE 2 DIABETES MELLITUS "

By

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Under the guidance of

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LIST OF ABBREVIATIONS USED

(In alphabetical order)

Abbreviations	
DM	Diabetes Mellitus
Type2DM	Type 2 Diabetes Mellitus
CVD	Cardiovascular Diseases
TG	Triglycerides
LDL-C	Low density lipoproteins- Cholesterol
HDL	High density lipoproteins
VLDL	Very low density lipoproteins
CHD	Coronary heart disease
BMI	Body mass index
WC	Waist circumference
WHR	Waist hip ratio
HTN	Hypertension
WHO	World health organization
TVD	Triple vessel disease
FFA	Free fatty acids
MI	Myocardial infarction
CAD	Coronary artery disease
VAT	Visceral adipose tissue
IL-6	Interleukin-6
TNF-α	Tumour necrosis factor- α
CPR	C-reactive protein
DXA	Dual–energy x-ray absortiometry
CT	Computed tomography
MRI	Magnetic resonance imaging
NHLBI	National Heart, Lung and Blood Institute
IDF	International Diabetes federation
BP	Blood pressure
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
FBS	Fasting blood sugar
PPBS	Post prandial blood sugar

ABSTRACT

Background and Objective:

Type2DM is associated with a marked increase in the risk of cardiovascular disease. Patients with type2DM are frequently affected by atherosclerotic vascular disease Multiple factors contribute to the accelerated atherosclerosis in type2DM. These factors include dyslipidemia, obesity, hypertension, and insulin resistance. These risk factors have a great potential for prevention through modification of life style and dietary changes. The purpose of this study is to determine the association between some anthropometric measurements (BMI, WC), blood pressure, and lipid profile as important CVD risk factors in type2DM.

Method:

This study consisted of 138 type2DM and 138 normal healthy individuals in the age group of 40-65 years. The BMI, WC, BP and lipid profile were compared between the normal and type2DM individuals. Also, the correlation between BMI with lipid profile and WC with lipid profile was studied in both type2DM and normal individuals.

Results:

The present study showed a significant increase in BMI ($p<0.001$) and WC ($p<0.0010$) in diabetic group. SBP ($p<0.001$) and DBP ($p<0.001$) both showed a significant increase in diabetic group. Serum levels of total cholesterol ($p<0.001$), Triglycerides ($p<0.001$), VLDL ($p<0.001$), LDL-C ($p<0.001$) FBS ($p<0.001$) and PPBS ($p<0.001$) were significantly higher and serum HDL-C ($p<0.001$) was lower in diabetic group. Also there was a positive correlation of BMI and WC with

Triglycerides, Total cholesterol, LDL, and VLDL and negative correlation with HDL-C in diabetics. Both BMI and WC had a positive correlation with FBS and PPBS. WC positively correlated with SBP and DBP in diabetic group .

Conclusions:

This study clearly shows that all lipid fractions (TG, TC, LDL-C, and VLDL-C) were abnormally elevated in type2DM when compared to controls except HDL-C suggesting that type2DM has a real impact on lipid metabolism. Type2DM is often associated with obesity, in particularly abdominal obesity as evident in the present study. Also in the present study BMI and WC had a positive correlation with total cholesterol, triglycerides, LDL-C, VLDL, FBS, PPBS and BP and negative correlation with HDL-C. Thus both BMI and WC independently contribute to the prediction of total, and abdominal subcutaneous and visceral fat and thus recommends health care practitioners routinely use both anthropometric variables to identify those at increased health risk.

Key words: Lipid profile, Type2DM, BMI, WC, HTN

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

INTRODUCTION

Diabetes Mellitus is a systemic disease that produces changes in the structure and functions of several tissues particularly connective tissue with complications that affects the eyes, kidneys, cardiovascular system and nervous system.

Diabetes mellitus (DM) is a group of chronic metabolic diseases characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both (1). The incidence of type 2 diabetes mellitus (Type2DM) is rapidly increasing worldwide and it constitutes a major health problem in both developed and developing countries (2). There is a great deal of evidence that both genetic and environmental factors are of importance in the pathogenesis of Type2DM. Obesity in particular the central obesity, physical inactivity and a diet rich in saturated fatty acids increases the risk of type2DM (3).

It has been centuries since this syndrome was first recognized. The term **DIABETES** which is from the Greek meaning to pass through was first used by ARETAEUS of Caappadocia in the second century AD as a generic description for conditions causing increased urine output. The association of polyuria with a sweet tasting substance in the urine was first reported in Sanskrit literature dating from the 5^{th} and 6^{th} century AD at the time of two notable physicians, Susruta and Charaka.The urine of certain polyuria patients was described as tasting like honey ***madhumeha being sticky to touch and strongly attracting ant****(4).*

Epidemiology of Diabetes Mellitus:

Global prevalence

The prevalence of diabetes for all age groups world wide was 2.8% in 2000 and is estimated to reach 4.4% by 2030.The total number of diabetics is projected to rise from 171million in 2000 to 366 million in 2030 (5).

Prevalence of diabetes in India.

The prevalence of diabetes in India is 2.4% in rural and 4 to 11.6% in urban dwellers. 20% of the current global population resides in regions of South East Asia. India comprises 85% of the adult population of South East Asia. Therefore, the major contribution to diabetics in South East Asia is from India (6).

CLASSIFICATION OF DIABETES MELLITUS (7)

Etiological classification of diabetes mellitus

I. Type1 Diabetes Beta Cell destruction usually leading to absolute insulin deficiency.

a. Immune mediated

b. Idiopathic

II. Type2 Diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretary defect with insulin resistance) .

III. Other specific type.

A. Genetic defect of Beta cell function

Chr12 HNF –α (MODY3), Chr7 glucokinase (MODY2), Chr20 HNF -4α (MODY1), Chr13 insulin promoter factor1 (IPF1, MODY4), Chr17 HNF -1β (MODY5), Chr2 neuroD, (MODY6), Mitochondrial DNA, Others .

B. Genetic defect in insulin action

TypeA insulin resistance, Leprechaunism, Rabson Mendenhall syndrome, Lipoatropic diabetes, Others

C. Diseases of the exocrine pancreas

Pancreatitis, Trauma/ Pancreatectomy, Neoplasia, Cystic fibrosis, Hemochromatosis, Fibrocalculous pancreatopathy, Others

D. Endocrinopathies

Acromegaly, Cushing's syndrome, Glucagonoma, Pheochromocytoma, Hyperthyroidism, Somatostatinoma, Aldosteronism, Others .

E. Drugs or chemical induced

Vacor, Pentamidine, Nicotinic acid, Glucocorticoids, Thyroid hormone, Diazoxide, β Adrenergic agonists, Thiazides, Dilantin, α-Interferon, Others .

F. Infections

Congenital rubella, Cytomegalovirus, Others .

G. Uncommon forms of immune mediated diabetes

Stiff man syndrome, Anti insulin receptor antibodies, Others .

H. Other genetic syndromes associated with diabetes

Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Wolfram's syndrome, Friedreich's chorea, Huntington's chorea, Laurence moon Biedl syndrome, Myotonic dystrophy, Porphyria, Prader Willi syndrome, Others.

IV. Gestational diabetes mellitus

PATHOGENESIS OF TYPE2 DIABETES.

Type2 diabetes mellitus is characterized by three patho physiologic abnormalities:

1. Impaired insulin secretion

2. Peripheral insulin resistance

3 .Excessive hepatic glucose production

Obesity, particularly visceral or central as evidenced by the waist-hip ratio is very common in Type2 diabetes mellitus. Insulin resistance associated with obesity augments the genetically determined insulin resistance Type2 diabetes mellitus. Adipocytes secrete a number of biologic products (Leptin, tumour necrosis factor A, free fatty acids) that modulate processes such as insulin secretion, insulin action and body weight may contribute to the insulin resistance.

In the early stages of disorder, glucose tolerance remains normal despite insulin resistance, because pancreatic β cells compensate by increasing insulin output. As insulin resistance and compensatory hyperinsulinemia progress, the pancreatic islets become unable to sustain the hyperinsulinemic state. Impaired glucose tolerance marked by elevation in post prandial glucose then develops. A further decline in insulin secretion and increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia. Ultimately, beta cell failure may ensure (8).

Metabolic abnormalities

A. Insulin resistance

This is caused by the decreased ability of insulin to act effectively on peripheral target tissues (muscle, liver) and is a prominent feature of type2 diabetes mellitus. Resistance to action of insulin impairs glucose utilization by insulin sensitive tissues and increases hepatic glucose output. Both these effects are contributing to the hyperglycaemia of diabetes mellitus.

Increased hepatic glucose output predominantly accounts for increased FPG levels, where as decreased peripheral glucose usage results in post prandial hyperglycaemia.

The precise molecular mechanism of insulin resistance in type2 diabetes mellitus has yet to be elucidated. Insulin receptor levels and tyrosine kinase activity in skeletal muscle are reduced, but these alterations are most likely secondary to hyperinsulinemia and are not a primary defect. Therefore, post receptor defects are believed to play a predominant role in insulin resistance. A current focus for the

pathogenesis of insulin resistance is on a PI-3 kinase signaling defect, which causes reduced traslocation of GLUT4 to the plasma membrane, among other abnormalities.

Another emerging theory proposes that elevated level of free fatty acid, a common feature of obesity may contribute to the pathogenesis of type2 diabetes mellitus. In several different ways, free fatty acid can impair glucose utilization in skeletal muscle, promote glucose production by the liver, and impair beta cell function(8).

B. Impaired insulin secretion

Insulin secretion and sensitivity are interrelated in type2 diabetes mellitus. Insulin secretion initially increases in response to insulin resistance in order to maintain normal glucose tolerance. Initially, insulin secretary defect is mild and selectively involves glucose stimulated insulin secretion.

The reason for the decline in insulin secretary capacity in type 2 diabetes mellitus is unclear. Despite, the assumption that a second genetic defect superimposed upon insulin resistance leads to beta cells failure, intense genetic investigation has so far excluded mutation in islet candidate genes. Islet amyloid polypeptide or amylin is co-secreted by beta cells and likely forms the amyloid fibrillar deposit which used to be found in the islets of individuals with longstanding type 2 diabetes mellitus. Whether such islet amyloid deposits are a primary or secondary event is not known. The metabolic environment may also impact islet function negatively for example chronic hyperglycemia paradoxically impairs islet function (glucose toxicity) and leads to worsening of hyperglycemia. Improvement in glycemic control is often associated with improved islet function. In addition, elevation of free fatty acid level (lipotoxicity) also worsens islet function (8).

C. Increased hepatic glucose production

In type2 diabetes mellitus, insulin resistance in the liver arises from the failure of hyperinsulinemia to suppress gluconeogenesis, which results in fasting hyperglycemia and decreased glucose storage by the liver in the post prandial state. How changes in hepatic glucose flux lead to insulin resistance is not clearly defined.

The mechanisms responsible for the increasing hepatic gluconeogenesis include Hyperglucagonemia, increased circulating levels of glucogenic precursors (lactate, alanine and glycerol), increased FFA oxidation enhanced sensitivity to glucagon and sensitivity to insulin. Although majority of evidence indicates that increased gluconeogenesis is the major cause of hepatic glucose production in type2 diabetes mellitus, it is likely that accelerated glycogenolysis also contribute (8).

Table 2: Major risk factors for type2 diabetes mellitus

1. Family history of diabetes mellitus,(parents or siblings with diabetes mellitus)
2. Overweight (BMI 25kg/m^2)
3. Habitual physical inactivity
4. Race/ ethinicity
5. Previously identified IFG or IGT
6. Hypertension (140/90mmHg in adults)
7. HDL cholesterol <35mg/dl(0.90mmol/L) and are a triglyceride level >250mg/dl (2.83mmol/L)
8. History of GDM or delivery of a baby weight >9lb
9. Polycystic ovary syndrome.

Cardiovascular disease (CVD) is a major cause of morbidity and mortality in patients with type2DM having 2 - 4 times higher risk of developing CVD when compared to non diabetics. In diabetics, cardiovascular complication occurs at an earlier age and often results in premature deaths (9). Patients with type2DM are frequently affected by atherosclerotic vascular disease. Multiple factors contribute to this accelerated atherosclerosis in type2DM. These factors include dyslipidemia, obesity, hypertension, and insulin resistance (10-12).

Lipid abnormalities are more common in type2DM and are aggravated with poor glycaemic control. The classical dyslipidemia in type2DM is so called atherogenic dyslipidemia. This is a constellation of lipid abnormalities which includes increased serum triglycerides (TG), increased low-density lipoprotein cholesterol (LDL-C) and decreased high-density lipoprotein cholesterol (HDL-C) also known as "lipid triad"(13). Lipid abnormalities play an important role in the causation of diabetic atherosclerosis. Elevated levels of TG, cholesterol and LDL-C increase the risk of atherogenesis and high levels of HDL-C in contrast bear an inverse relationship to the risk of atherosclerosis and coronary heart disease (CHD) (14).

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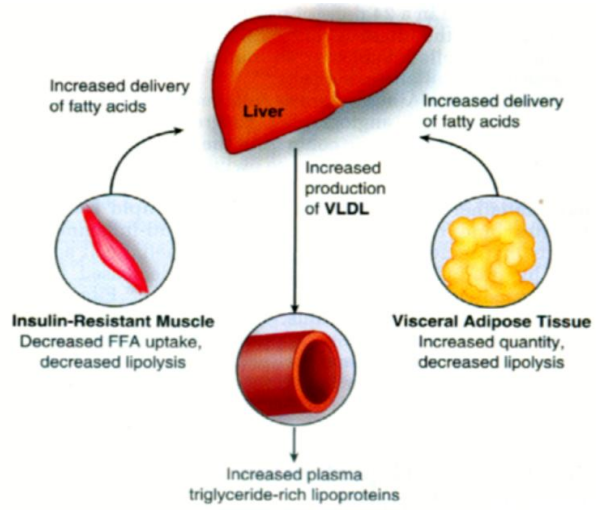
Type2DM is associated with centralized and disharmonious distribution of fat. There is a significant association between regional fat distribution and CVD risk factors. Abdominal or central adiposity is considered the most important determinant of CVD and Type2DM (15). Although, imaging techniques can accurately determine total body fat and its distribution in human but are not suitable for use in large population studies because of cost, irradiation exposure and limited availability (16). The use of simple anthropometric measurements seems to diagnose obesity in early stages due to its benefits in routine monitoring and assessment in patients. Some of the simple anthropometric measures used routinely include Body Mass Index (BMI), Waist Circumference (WC) and Waist to Hip Ratio (WHR) (17). BMI is widely used for classification of obesity, but it does not account for the variations of fat distributions. Waist circumference is the best simple anthropometric index of abdominal visceral adipose tissue and also the best index for predicting CVD risks (18).

In India, 50% of diabetics have hypertension (HTN). The frequency of hypertension in diabetic population is almost twice as compared to non-diabetic general population (10). In hypertensive patients with DM, atherosclerosis gets accelerated and its consequences get manifested earlier (19). Both HTN and type2DM are recognised as independent CVD risk factors (20).

These risk factors have a great potential for prevention through modification of life style and dietary changes. The purpose of this study is to determine the association between anthropometric measurements (BMI, WC), blood pressure and lipid profile as important CVD risk factors in type2DM.

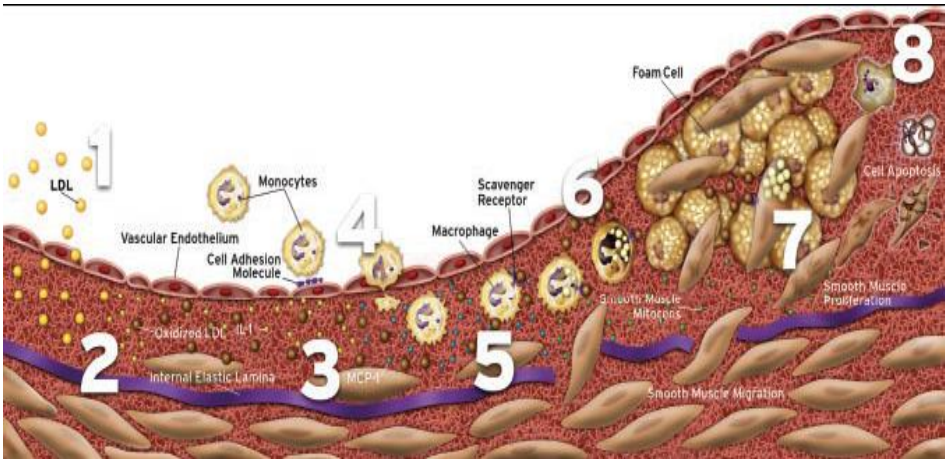
Cholesterol and triglyceride transfer between VLDL and LDL and depends on increased levels of VLDL, particularly when triglyceride concentration are higher than 130mg/dl (32).

Figure 1: Pathogenesis of dyslipidemia (32)



Small dense LDL-C are proatherogenic, first LDL-C moves into sub endothelium and is oxidized by macrophages and smooth muscle cells (stage 1 and 2). Release of growth factors cytokines attracts additional monocytes (stage 3 and 4). Foam cell accumulation and proliferation results in growth of the plague (stage 6, 7, and 8) (33).

Figure 2: The 7 stages of development of an atherosclerotic plague (34)



Atherosclerosis is the process underlying CVD, which includes coronary heart disease (CHD), myocardial infarction (MI), ischemic stroke, and peripheral vascular disease (PVD) (35). Atherosclerosis is the primary cause of death in patients with type2DM and it seems to be closely related to a specific cluster of lipid abnormalities, including low levels of HDL-C, increased numbers of small dense LDL-C, and elevated triglyceride levels (14). The classical dyslipidemia in type2DM is so called **atherogenic dyslipidemia**. This is a constellation of lipid abnormalities also known as "lipid triad"(13). This risk is even greater when the lipid triad is accompanied by insulin resistance, a procoagulant state, and hypertension—a condition known as the cardiovascular dysmetabolic syndrome. Each of these abnormalities is associated with an increased risk for cardiovascular morbidity and mortality.

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS (35)

Three ways to diagnose diabetes are possible, & each in the absence of unequivocal hyperglycemia, must be confirmed on a subsequent day, by any one of the three methods given in the table 1.

Diagnostic criteria for diabetes mellitus

1.Symptoms of diabetes plus casual plasma glucose concentration ≥200mg/dl(11.1mmol/l).Casual is defined as any time of the day without regard to **t**ime since last meal .The classic symptoms of diabetes include polyuria, polydipsia, & unexplained weight loss.

OR

2. FPG ≥126mg/dl(7mmol/l).**Fasting** is defined as no caloric intake for at least 8hours.

OR

3.Two hours post load glucose ≥200mg/dl(11.1mmol/l) during an OGTT. The test should be performed as described by WHO, using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in **water**.

In the absence of unequivocal hyperglycaemia, these criteria should be confirmed by repeat testing on a different day.

The third measure (OGTT) is not recommended for routine clinical use .The ADA recognizes both tests FPG and OGTT as valid but continue to recommend the FPG as the preferred diagnostic tool because it is more convenient, acceptable to patients and less expensive.

IMPAIRED GLUCOSE TOLERENCE (IGT) AND IMPAIRED FASTING GLUCOSE

This group is defined as having FPG levels 100mg/dl (5.6mmol/l) but < 126mg/dl (7 mmol/l) or 2-h values in the oral glucose tolerance test of 140mg/dl(7.8mmol/l) but <200mg/dl (11.1mmol/l). Thus, the categories of FPG values are as follows:

FPG<100mg/dl (5.6mmol/l)=normal FG

FPG 100-125mg/dl (5.6mmol/l-6.9mmol/l)=IFG

FPG 126mg/dl (7mmol/l) = Provisional diagnosis of diabetes (the diagnosis must be confirmed as described above).

The corresponding categories when the OGTT is used are the following

2 hrs post load glucose <140mg/dl(7.8mmol/dl)=normal OGTT

2 hrs post load glucose 140-199mg/dl(7.8-11.1mmol/l)=IGT

2 hrs post load glucose 200mg/dl (11.1mmol/l) = Provisional diagnosis of diabetes. (the diagnosis must be confirmed as described above).

Majority of Indian type2DM are dyslipidemic at baseline. The most common pattern of dyslipidemia is high LDL-C and low HDL-C among both males and females contributing to 22.7% and 33% patients of diabetic dyslipidemia, respectively (31). The most prevalent problem among males is high LDL –C while among females low HDL –C emerged as a bigger threat. In Indian subjects with DM the lipid profile and pattern is greatly influenced by the ethnic origin, food habit, nutritional status and lifestyle influences. There has been a quantum increase in the incidence of

CAD amongst urbanites while the picture in rural India has changed very little, suggesting the major impact of lifestyle modifications on lipid profiles and the deleterious effect of the latter in causing accelerated and more extensive CAD as evident angiographically (38).

Hypercholesterolemia, hypertriglyceridemia, elevated LDL-C, and low high HDL-C are generally accepted as strong risk factors for cardiovascular disease (CVD) and mortality (39-41).

Obesity has become a major worldwide epidemic affecting more than 300 million people, with changing food habits and increasing sedentary lifestyles, the prevalence of obesity has increased markedly (2). Both absolute total fat and adipose tissue distribution are closely associated with the risk of diabetes, hypertension, hyperlipidemia and CVD (61). In developed countries 80% of type2DM are obese where as in developing countries less than 50% of type2DM are obese (2).

Although overweight and obesity are associated with an increased risk of type2DM and CVD, the pattern of fat distribution is also important in risk stratification.

Intra-abdominal (visceral) fat deposition is linked to an increased risk for developing DM and CVD compared with a more peripheral (subcutaneous) fat distribution (62). However, it remains uncertain which pattern of obesity is more significant predictor of metabolic syndrome. Many studies discovered that fat distribution, rather than absolute total fat, is more closely associated with these risk factors (61). Other studies however, found that total body fat, rather than its distribution, is strong predictor of metabolic risk (63).

Adipose tissue is now viewed as an active endocrine organ and not merely as energy deports. The visceral adipose tissue (VAT) accounts for approximately 15% of

total body fat in lean subject and includes the intraperitoneal (mesenteric and omental) fat, which drains into the portal circulation and retroperitoneal fat, which drains into the systemic circulation. The aetiology of increased cardio metabolic risk in patients with increased VAT is not known, but there are many theories. The VAT is more insulin resistant, fuelling the hyperlipolytic state and worsening of insulin resistance. The increase in VAT mass possibly reflects the lack of capacity of subcutaneous tissue to store the energy excess that accumulates in liver, muscle, and pancreas, worsening insulin resistance (64).

Numerous cytokines are secreted from adipose tissue, including proinflammatory molecules such as interleukin-6 (IL-6), tumour necrosis factor-α (TNF-α). Plasma levels of C-reactive protein (CPR), an inflammatory factor produced by liver and known to correlate with atherosclerosis, are increased in patients with visceral obesity (65). It has been recently shown that the adipose tissue is infiltrated by macrophages contributing to inflammatory processes. Adiponectin, a protein derived from the adipose tissue, is known to improve insulin sensitivity and may protect against atherosclerosis. Adiponectin levels are decreased in obesity and in viscerally obese patients (66). Another contributing factor may be local generation of cortisone due to increased activity of 11β-hydroxysteroid dehydrogenase in the VAT, which may further increase fat deposition, worsen insulin resistance, and increase cardio metabolic risks (67).

There are many ways to measure total body fat; traditionally, the gold standard for estimation has been hydro densitometry (underwater weighing), based on the fact that fat tissue is less dense than muscle and bone. Other methods used to assess total body fat Dual–energy x-ray absortiometry (DXA) and body-fat distribution using computed tomography (CT) and magnetic resonance imaging (MRI) in humans are

not suitable for use in large population studies because of cost, irradiation exposure, and limited availability, they are generally not appropriate outside specific research setting (16).

The use of simple anthropometric measurements seems to diagnose obesity in early stages. As a result, many attempts have been made to find out the most appropriate anthropometric index in different studies.

BMI or Quetelet index, defined as weight/height^2 is the most widely used and simple measure of body size, and frequently used to estimate the prevalence of obesity within a population. A BMI $\geq$ 25 Kg/m2 is associated with increased morbidity, primarily from DM and CVD, while a BMI >30 Kg/m2 is associated with increased risk for both morbidity and mortality, the latter mainly from diabetes, coronary heart disease (CHD), and stroke. Rationale use of BMI is that it is supposed to closely correlate with tissue density, which in turn closely correlates with percent fat in body tissue (adiposity) and also it approximately uncorrelated with height; in contrast, height is highly correlated with weight, as well as with most proposed obesity indices. An implicit assumption made in many epidemiologic analyses is that BMI alone is a sufficient measure of anthropometric effects. This is not necessarily true, however; whether BMI alone adequately captures the effect of anthropometric variables on health outcomes depends on many factors. In particular, although BMI may capture most of the information on the body composition contained in weight and height, it does not capture information on body size .Also a muscular patient may have high BMIs but low fat mass, and in elderly, BMI may underestimate fat mass due to decrease in lean body mass (68).

Body fat distribution is an important risk factor for obesity related disease. Increased visceral adipose tissue (also called central or abdominal fat) is associated

with increased risk for cardio metabolic disease. Clinical evidence suggests that the association of diabetes with central obesity is stronger than the association with general fat. Studies using computed tomography and magnetic resonance imaging have provided further evidence to support that central obesity, visceral adipose tissue, and upper-body non visceral fat are the major contributors to the metabolic complication (69).

Central obesity has been associated with decreased glucose tolerance, alteration in glucose insulin homeostasis, reduced metabolic clearance of insulin, and decreased insulin-stimulated glucose disposal (70).

WC is used as a surrogate marker of abdominal fat mass. WC correlates with subcutaneous and visceral fat mass and is related to increased cardio metabolic risks. The National Heart, Lung and Blood Institute (NHLBI) recommended measuring WC along with BMI to assess patients risk stratification in subjects with a BMI between 25 and 35 Kg/m2. Cut off points of WC that define higher risk for men and women based on ethnicity have been proposed by International Diabetes federation (IDF).

Some studies have proposed that WC is a superior indicator, because it requires only one measurement and is a better indicator of visceral fat and CVD risk (16). There is a growing opinion that WC should be considered as a 'vital sign' and recorded in the same manner as weight and height in the medical chart of every patient. Also WC could replace both BMI and WHR as a simple indicator of need for weight management as a health promotion activity (71).

Waist-to-hip ratio (WHR) is a common anthropometric index used to assess abdominal obesity. The WHO included a high WHR defined as a ratio > 0.9 in men and 0.85 in women as a criterion for diagnosing the metabolic risks. A higher WHR has been associated with increased cardiovascular and DM risk (72). Some studies

found that WHR is more accurate tool to diagnose patients with a higher CVD risk compared with WC and BMI, whereas other studies have found a better correlation of cardio metabolic risks with WC compared with WHR (16). WC provides a crude index of absolute amount of adipose tissue whereas WHR provides an index of relative accumulation of abdominal fat to generalized obesity (73).

More than three decades ago Harry Keen pinpointed two "bad companions" to diabetes: high blood glucose concentrations and high blood pressure, both associated with microalbuminuria (74). **HTN** is a very common co morbid condition in diabetes and accounts for up to 85% of excess CVD risk. Patients with hypertension are more prone to diabetes than are normotensive patients. When HTN coexist with diabetes, the risk of development of CVD is doubled (2). In type2DM, HTN usually clusters with the other components of cardio metabolic syndrome, such as central obesity, insulin resistance, dyslipidemia, hypercoagulation, increased inflammation and hyperuricemia. HTN in individuals with diabetes has characteristic features, including volume expansion, increased salt sensitivity, isolated systolic hypertension, loss of nocturnal dipping of blood pressure, increased propensity towards orthostatic hypotension and albuminuria. The association between HTN and insulin resistance, and the resultant hyperinsulinemia is well established.

In untreated patients with essential HTN, fasting and postprandial insulin levels were higher than in normotensive controls. Sensitivity to dietary salt intake is greatest in elderly and diabetics, this is particularly important to consider in the management of HTN in patients with diabetes. Patients with diabetes have loss of nocturnal dipping of blood pressure (BP), as demonstrated by 24 hour ambulatory monitoring of BP. This is particularly important since loss of nocturnal dipping conveys excessive risk for stroke and myocardial infarction. With the progression of

atherosclerosis in patients with diabetes, the larger arteries lose elasticity and become rigid. The systolic blood pressure (SBP) increases disproportionately because the arterial system is incapable of expansion for any given volume of blood ejected from the left ventricle, leading to isolated systolic HTN (2). Also there is a progressive increase in the prevalence of elevated blood pressure with adipose tissue (75,76).

Metabolic syndrome consists of a cluster of risk factors strongly associated with an increased risk for atherosclerotic cardiovascular disease and type2DM (77). The metabolic risk factors consist of a specific pattern of hyperlipidemia (elevated serum levels of triglycerides and apolipoprotein B, small LDL-C, and low levels of serum HDL-C), elevated BP, elevated plasma glucose concentration, a proinflammatory state, and a prothrombotic state (78). Central obesity is a key feature of the syndrome, reflecting the fact that the syndrome's prevalence is driven by the strong relationship between WC and increasing adiposity. The NCEP: ATPIII 2001 provided diagnostic criteria for metabolic syndrome that could be easily implemented in clinical practice. These criteria include WC, blood pressure measurements, and serum levels of triglycerides, HDL-C, and fasting glucose (3). Because obesity is the main culprit for metabolic syndrome, it is important to focus on life style changes that will improve all components of the syndrome instead of on each individual risk factor.

Weight loss is known to improve insulin resistance, DM control, HTN and lipid profile. Lifestyle changes, including weight loss and physical activity, have been shown to prevent or delay type2DM in high risk patients. In diabetes prevention program study, lifestyle intervention was associated with a marked reduction in visceral fat (measured by CT) and a smaller decrease in subcutaneous fat, body weight, BMI and WC in both men and women. Weight loss achieved through diet and

pharmacotherapy with sibutramine, orilistat, and rimonabant has shown a decrease in WC and improvement in cardio metabolic risk (79).

Efforts should be made to continuously educate the populace on diabetics, its management, feeding and lifestyles. Diabetes is spreading like an epidemic all over the world. As is wisely said “prevention is better than cure” we need cautious evaluation of various risk factors so that appropriate measures can be taken timely, in order to prevent grave sequelae later on. Lifestyle modifications, inclusive of dietary modification, regular physical activity and weight reduction are indicated for prevention of diabetes.

OBJECTIVES

To study and compare Body mass index, Waist circumference, Blood pressure and lipid profile between Type 2 diabetes mellitus and Non-diabetic healthy subjects.

REVIEW OF LITERATURE

As a result of increasing urbanisation and economic development, the prevalence of type2DM worldwide is rapidly increasing to epidemic proportions. The global burden of diabetes mellitus was estimated at 124 million people in 1997 with a projected increase to 221 million people by 2010. The projected increase is greatest in Asia, where there is a predicted rise of 57% from 2000 to 2010 (23). World Health Organization (WHO) has predicted that India would experience the largest increase (48% increase in total population and 168% increase in population with >65 years of age) in type2DM and would have the greatest number of diabetic individuals in the world by the year 2030 (31.7 million in 2000 to 79.4 million in 2030) (24,25).

Several evidences have contributed to our current understanding of the relationship between increase in plasma cholesterol and development of CHD. Premature atherosclerosis results from high cholesterol levels, even in the absence of other cardiovascular risk factors. Large population surveys have shown that plasma cholesterol level is predictive of CHD (41).

In the Framingham study individuals below 50 years, cholesterol level was directly related to cardiovascular mortality. The study highlights the profound effects of lipoprotein abnormalities on incidence of CAD in diabetics compared to nondiabetics (42). In a large prospective study, over 350,000 men aged 35 to 57 years were followed for 6 years. A curvilinear relationship between plasma cholesterol and coronary death rate was observed. If a risk ratio of 1 is assigned for a cholesterol level of 200 mg/dl, then at 250 mg/dl, the risk is doubled. This relation between cholesterol and CHD is not lost in the presence of other risk factors such as diabetes. The presence of diabetes further increased the risk of a given cholesterol level (43).

Because most cholesterol in plasma is transported in LDL and this is responsible for the correlation between plasma cholesterol and CHD.

On the contrary some studies showed no significant difference in the lipids and lipoprotein profiles of diabetics and that of control .

High-density lipoprotein cholesterol has been repeatedly shown to be an independent inverse predictor of CVD risk in epidemiological and observational studies, and patients with low HDL-C levels have been suggested to have a comparable CVD risks as those with high LDL-C levels (44). An inverse relationship between HDL-C and A1C levels has been described in type 2 diabetic patients (45). The distribution of HDL-C levels varies with age, sex, race, and education. Women have higher levels of HDL-C than men. Body mass index is negatively associated with HDL-C. Alcohol consumption is directly related to HDL-C levels and an inverse relation between smoking and HDL-C levels has been reported. Low HDL-C represents a highly prevalent and potentially modifiable risk factor for CVD prevention in type2DM (46,47).

A study (2009) conducted so as to compare the lipid profile of diabetic patients and healthy controls. The lipid profiles and lipoprotein levels of 50 known diabetic patients and 50 healthy subjects were studied. Total cholesterol (TC), Triacylglycerols (TG), Low density lipoprotein-cholesterol (LDL-C), and high density lipoprotein cholesterol (HDL-C) levels were assayed for each group using standard biochemical methods(9)**.**

The study concluded that the mean TC, TG, and low density lipoprotein cholesterol levels were lower in the diabetic than in the control subjects though these were not significant ($P > 0.05$). The frequency of high TC level was higher in the

diabetic group while the frequency of low HDL-C level was higher in the healthy controls. The prevalence of high TG and LDL-C were approximately equal in the two groups. The mean (±SD) HDL- C was significantly lower ($P < 0.05$) in males compared to the females for both diabetic and control groups. The better lipid profiles in the diabetic patients compared to the controls were apparently due to the regime of management of their condition.

A study (2002) was conducted to examine the effects of the degree of body mass index and weight gain as the risks for hypertension, hypercholesterolemia and diabetes in Japanese men, and to compare that to the corresponding effects in a Caucasian population (12). The study included a total of 4737 male employees followed until retirement. It was concluded that among Japanese, the degrees of body mass index associated with risks for hypertension, diabetes and hypercholesterolemia were lower than those in Caucasians. The risks for hypertension and hypercholesterolemia were strongly associated with weight gain in a Japanese male population who showed a low prevalence of severe obesity, and the risks were similar to or somewhat higher than those in a Caucasian population with a high prevalence of severe obesity.

The study (2006) conducted to evaluate and compare the associations between weight changes during 2 different periods of adult life and the risk of type 2 diabetes and age at diagnosis.

The study included 7720 men and 10 371 women from the European Prospective investigation into Cancer and Nutrition (EPIC)–Potsdam Study with information on weight history; 390 men and 303 women of these participants

received a clinical diagnosis of type 2 diabetes during 7 y of follow-up. Multivariate Cox regression models were used to estimate the relative risk (RR) of weight changes between ages 25 and 40 y and ages 40 and 55 y.

It was concluded that weight gain in early adulthood is related to a higher risk and earlier onset of type 2diabetes than is weight gain between 40 and 55 y of age.

To compare associations of diabetes incidence with general and central obesity indicators, a meta-analysis study (2007) was conducted based on published studies from 1966 to 2004 retrieved from a PubMed search(17) . The analysis was performed with 32 studies out of 432 publications initially identified.

Measures of association were transformed to log relative risks per standard deviation (pooled across all studies) increase in the obesity indicator and pooled using random effects models.

The pooled relative risks for incident diabetes were 1.87 (95% confidence interval (CI): 1.67, 2.10), 1.87 (95% CI: 1.58, 2.20), and 1.88 (95% CI: 1.61, 2.19) per standard deviation of body mass index, waist circumference, and waist/hip ratio, respectively, demonstrating that these three obesity indicators have similar associations with incident diabetes.

The study (2010) conducted in Khyber Medical College, Peshawar during 2008 to 2009. A total of 475 adult male and female volunteers were the subject of this research and were categorized in terms of their BMI. The BMI was determined from weight and height; the subjects were grouped as normal, overweight and obese. WC was determined by measuring the waist between the lower rib and iliac crest.

The results show a consistence relation between BMI and WC with diabetes mellitus. The Chi-square test for 95% confidence interval showed 2-sided

asymptomatic significance of diabetes mellitus with WC to be 0.016 and BMI 0.082. It was concluded that a higher trend of diabetes mellitus in males having WC greater then 40 inches (100 cm) and for female WC greater then 35 inches (87.5 cm) as compare to higher BMI.

Another study (2002) conducted to examine whether the prevalence of hypertension, type 2 diabetes mellitus, dyslipidemia, and the metabolic syndrome is greater in individuals with high compared with normal WC values within the same BMI category (18).

The subjects consisted of 14 924 adult participants of the Third National Health and Nutrition Examination Survey, which is a nationally representative cross-sectional survey. Subjects were grouped by BMI and WC in accordance with the National Institutes of Health cutoff points. Within the normal-weight (18.5-24.9), overweight (25.0-29.9), and class I obese (30.0-34.9) BMI categories, it was computed odds ratios for hypertension, diabetes, dyslipidemia, and the metabolic syndrome and compared subjects in the high-risk (men, >102 cm; women, >88 cm) and normal-risk (men, <or=102 cm; women, <or=88 cm) WC categories. With few exceptions, within the 3 BMI categories, those with high WC values were increasingly likely to have hypertension, diabetes, dyslipidemia, and the metabolic syndrome compared with those with normal WC values. Many of these associations remained significant after adjusting for the confounding variables (age, race, poverty-income ratio, physical activity, smoking, and alcohol intake) in normal-weight, overweight, and class I obese women and overweight men. It was concluded that The National Institutes of Health cutoff points for WC help to identify those at increased health risk within the normal-weight, overweight, and class I obese BMI categories.

A study (2005) conducted to compare body mass index (BMI), WC, and WHR in predicting type 2 diabetes . It was a prospective cohort study (Health Professionals Follow-Up Study) of 27 270 men . WC, WHR, and BMI were assessed at baseline. Covariates and potential confounders were assessed repeatedly during the follow-up. During 13 y of follow-up, it was documented 884 incident type 2 diabetes cases. Age-adjusted relative risks (RRs) across quintiles of WC were 1.0, 2.0, 2.7, 5.0, and 12.0; those of WHR were 1.0, 2.1, 2.7, 3.6, and 6.9; and those of BMI were 1.0, 1.1, 1.8, 2.9, and 7.9 (P for trend < 0.0001 for all). Multivariate adjustment for diabetes risk factors only slightly attenuated these RRs. Adjustment for BMI substantially attenuated RRs for both WC and WHR. The receiver operator characteristic curve analysis indicated that WC and BMI were similar and were better than WHR in predicting type 2 diabetes. The cumulative proportions of type 2 diabetes cases identified according to medians of BMI (>/=24.8), WC (>/=94 cm), and WHR (>/=0.94) were 82.5%, 83.6%, and 74.1%, respectively. The corresponding proportions were 78.9%, 50.5%, and 65.7% according to the recommended cutoffs. Both overall and abdominal adiposity strongly and independently predict risk of type 2 diabetes. WC is a better predictor than is WHR.

A study (2001) was conducted to examine the relationship between 24 h ambulatory blood pressure monitoring and three commonest anthropometric measurements for obesity: body mass index (BMI), waist-to-hip ratio (WHR) and waist circumference (W). Four-hundred and sixty-one overweight or obese subjects, non-diabetic, otherwise healthy, aged 20-70 y, of either sex, were consecutively recruited. All subjects underwent 24 h ambulatory blood pressure monitoring. The population study was separated in normotensive and hypertensive males and females and the possible risk factors for hypertension (W, WHR, BMI and age) were subdivided into different classes of values.Logistic regression shows that W is

the most important anthropometric factor associated with the hypertensive risk. Among males with W $\geq$ 102 cm the odds ratio (OR) for hypertension is three times that of males with W<94 cm using casual BP measure (OR 3.04), nearly four times higher using 24 h BP mean (OR 3.97), and even five times higher using day-time BP mean (OR 5.19). Females with W$\geq$88 cm have a risk for hypertension twice that of females with W<80 cm, whatever BP measurement was take (casual, 24 h or day-time). Males with WHR$\geq$0.96 and females with WHR$\geq$0.86 show significant OR for hypertension only by 24 h BP measurement and by day-time BP measurement. BMI seems to have no significant relationship to hypertensive risk. Age shows a significant relationship to hypertensive risk only considering males aged ≥55 y and females aged $\geq$ 50 y. It was concluded that the waist circumference seems to have a strong association with the risk of hypertension.

METHODOLOGY

Sampling size:

This study comprised of total 276 subjects: 138 Type2DM patients (males n=108, females n=30) and 138 age matched healthy normal subjects (males n=108, females n=30)

Ethical clearance:

Ethical clearance was obtained from the Sri B.L.D.E U'S Shri B.M.Patil Medical College ethical committee for human research to conduct study.

Source of data:

The present study was a comparative study in which 138 type2DM cases were recruited from those attending outpatient departments at Shri B. M. Patil Medical College and Research Hospital, Bijapur from Nov 2010 to March 2012. 138 healthy age and sex matched normal subjects were included in the study by history, questionnaire and by clinical examination from general population.

Method of collection of data:

Subjects were examined for their general physical health. Subject's clinical history and details were taken according to the standard proforma & questionnaire. Subjects were assigned into two different groups: Group1 – Controls (Non-diabetic healthy Subjects) and Group2- cases (Type2DM Patients). An informed written consent was obtained from all the subjects.

Inclusion criteria:

1. Healthy individuals of age group 40-65 years with no major illness. (Group 1)
2. Physician diagnosed and those on treatment for type-2 Diabetes mellitus of 40-65 years age group. (Group 2)

Exclusion criteria:

1. Subjects with history of cardiovascular diseases.
2. Subjects with history of Endocrine disorders.
3. Subjects with history of cerebro vascular accidents.
4. Subjects on drugs like diuretics, B-blockers, and glucocorticoids

Procedure: This study involved both non-invasive and invasive procedure as listed below with no financial burden on the subjects. The subject were informed about the procedure in brief and made comfortable for 5min in silent room.

Parameters used for comparison

- Body Mass Index (BMI)
- Waist Circumference (WC)
- Blood pressure (mmHg)
- FBS (mg/dl)
- PPBS (mg/dl)
- LIPID PROFILE: Total cholesterol, LDL-C, HDL-C, Triglycerides, VLDL

Assessment of Body mass index (69)

Weight (kg) and height(cm) were measured using standard calibrated balance scale with vertical measuring rods. Height of the subject was measured without shoes in meters and weight in kg. The BMI was calculated as

$$BMI = \frac{\text{Weight (in kgs)}}{(\text{Height in meters})^2}$$

Categorization of body mass index (8)

Category	BMI(kg/m^2)
Underweight	<18.5
Normal weight	18.5-24.9
Over weight	25-29.9
Obesity (class 1)	30-34.9
Obesity (class2)	35-39.9
Extreme obesity (class 3)	>40

National institute of health, National Heart, lung and blood institute; 2000

Assessment of waist circumference (16)

WC (cm) was measured with subject standing, bare midriff, after the subject exhales ,with both feet touching and arms hanging freely, the measuring tape is placed perpendicular to the long axis of the body and horizontal to the floor at the midpoint between the lowest rib and iliac crest in centimetres.

Categorization of waist circumference (60)

Category	Men	Women
Low risk	<94cm	<80cm
Moderate risk	94-101cm	80-87cm
High risk	>102cm	>88cm

Recording of blood pressure (80) (mmHg)

The subject rested for 5 min in supine position. Blood pressure was measured in the right arm using Diamond Regular Sphygmomanometer. At least 3 blood pressure measurements were taken at an interval of 5 mins. The first and fifth korotkoff phase used to define systolic and diastolic blood pressure. The mean of 3 measurements were considered.

Blood sampling

The blood samples were drawn after 12 hours of fasting. The venepuncture was done in the cubital fossa. Tourniquet was used but was released just before sampling to avoid artificial increase in the concentration of serum lipids. About 5 ml of blood was drawn using perfectly dry and sterile syringes and the blood was transferred to dried glass vials. Serum was separated within 2 hrs of collection to prevent artificial changes in concentration of HDL. The blood was centrifuged at 5000 rpm for 10 minutes. The supernatant clean serum was then pipette out using dry piston pipettes with disposable tips and stored in dry thin walled vials at 4..c The samples were analysed the same day. Care was taken to exclude the haemolysed

serum. Total cholesterol, triglycerides, HDL were measured by Technicon RA-XT Random access auto Analyzer KIT (ERBA)

FBS estimation by Trinders method, end point/ fixed (80)

Methodology- Trinders method

Glucose in the sample is oxidised to yield gluconic acid and hydrogen peroxide in the presence of glucose oxidase. The enzyme peroxidise catalyses the oxidative coupling of 4-aminoantipyridine with phenol to yield a coloured quinonamine complex, with absorbance proportional to the concentration of glucose in sample.

$$\text{Glucose} + O_2 + H_2O \xrightarrow{\text{Glucose oxidase}} \text{gluconic acid} + H_2O_2$$

$$H_2O_2 + \text{Phenol} + 4\text{AAP} \xrightarrow{\text{Peroxidase}} \text{Quinoeimine dye} + 2H_2O$$

Cholesterol estimation by enzymatic method (81)

Methodology - Modified Roeschlau's method using by ERBA diagnostics Mannheim GmbH kit.

$$\text{Cholesterol ester} + H20 \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol} + \text{Fatty acid.}$$

$$\text{Cholesterol ester} + O2 \xrightarrow{\text{Cholesterol oxidase}} \text{Cholesterone} + H2O2$$

$$2HO2 + \text{phenol} + \text{4amino-antypyrine} \xrightarrow{\text{Peroxidase}} \text{Redquinone} + 4H2O$$

The concentration of cholesterol in the sample is directly proportional to red complex (redquinone) which is measured at 500nm.

Estimation of triglycerides (82,83)

Methodology- Wako and modification of McGowan(1983) and Fossati et al (1969)

In the presence of lipoprotein lipase, triglycerides are split into glycerol and fatty acids. In presence of ATP and glycerol kinase, glycerol is converted into glycerol 3-phospate into dihydroxy- acetone phosphate and hydrogen peroxide. In presence of peroxide, hydrogen peroxide reacts with PAP and ESPAS to form a violet colored quinonamine as indicator. The intensity of color developed is proportional to TG concentration.

Estimation of HDL cholesterol (84)

Methodology- Burstein et al (1970) method

Chylomicrons, VLDL and LDL were precipitated by adding phosphotungstic acid and magnesium ions to the sample. Centrifugation of precipitant leaves only the HDL in the supernatant, which was separated out and its cholesterol content was determined enzymatically. In patients with high TG values the HDL estimation was done after dilution of serum with isotonic saline and resultant cholesterol value was multiplied by 2. This was done to prevent erroneous values of HDL due to impaired sedimentation of the precipitate in a serum with high concentration.

Estimation of LDL cholesterol

LDL was calculated by using standard formula based on total cholesterol, TG and HDL values.

Friedwald's formula (85,1,3)

LDL = Total cholesterol - HDL – TG/5

Estimation of VLDL cholesterol (85,1,3)

Since VLDL is the primary triglyceride carrying form in the fasting stage, its concentration can be approximated by

$$\text{VLDL} = \frac{\text{Plasma triglycerides}}{5}$$

Diagnosis of dyslipidemia (3)

Dyslipidemia is diagnosed when any one of the following or combination is found

Total cholesterol	>200mg/dl
Triglycerides	150mg/dl
HDL	<40mg/dl
LDL	130mg/dl
VLDL	>40mg/dl

Statistical Methods: (86-2)

The BMI, WC, BP and lipid profile were compared between the normal and type2DM individuals Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean $\pm$ SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups Inter group analysis) Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two groups. Student t test (two tailed; independent) has been used to test the homogeneity samples based on of age (or continuous parameters) and Chi-square test to test the homogeneity of samples based on parameters on categorical scale between two groups. Pearson correlation has been used to find the correlation of duration of disease in cases with lipid parameters.

1. **Chi-Square Test**

$\chi^2 = \frac{\sum (Oi - Ei)^2}{Ei}$, Where Oi is Observed frequency and Ei is Expected frequency

2. **Fisher Exact Test**

	Class1	Class2	Total
Sample1	A	B	a+b
Sample2	C	D	c+d
Total	a+c	b+d	N

2x2.Fisher Exact Test statistic= $\sum p = \frac{(a+b)!(c+d)!(a+c)!(b+d)!}{n!}\frac{1}{\sum a!b!c!d!}$

3. Student t test (Two tailed, independent)

$$t = \frac{(\bar{x}_1 - \bar{x}_2) - (\mu_1 - \mu_2)}{\sqrt{s^2(1/n1 + 1/n2)}}$$

Where $s^2 = \frac{(n1-1)\sum_{i=1}^{n1}(x1-\bar{x}1)^2 + (n2-1)\sum_{i-1}^{n2}(x2-\bar{x}2)^2}{n1+n2-2}$

4. Analysis of a correlation coefficient

Objective: To investigate whether the difference between the sample correlation co-efficient and zero is statistically significant.

Limitations: It is assumed that the x & y values originates from a bivariate normal distribution and that relationship is linear. To test an assumed value of population co-efficient other than zero, refer to the Z-test for a correlation co-efficient.

$r = \frac{\sum(x-\bar{x})(y-\bar{y})}{\sqrt{\sum(x-x)^2\sum\ (y-y)^2}}$ $\quad$ $t = \frac{r\sqrt{(n-2)}}{\sqrt{(1-r^2)}}$ is calculated and follows student t distribution with n-2 degrees of freedom.

6. Classification of Correlation Co-efficient (r)

Up to 0.1	Trivial Correlation
0.1-0.3	Small Correlation
0.3-0.5	Moderate Correlation
0.5-0.7	Large Correlation
0.7-0.9	V.Large Correlation
0.9- 1.0	Nearly Perfect correlation
1	Perfect correlation

7. Significant figures

+ Suggestive significance (P value: $0.05 < P < 0.10$)

* Significant (P value: $0.01 < P \leq 0.05$)

** Strongly significant (P value: $P \leq 0.01$)

Statistical software: The Statistical software Systat 11.0 was used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

Figure: 3 Recording of Blood Pressure

Figure 4: Recording of Height

Fig5 : Apparatus used

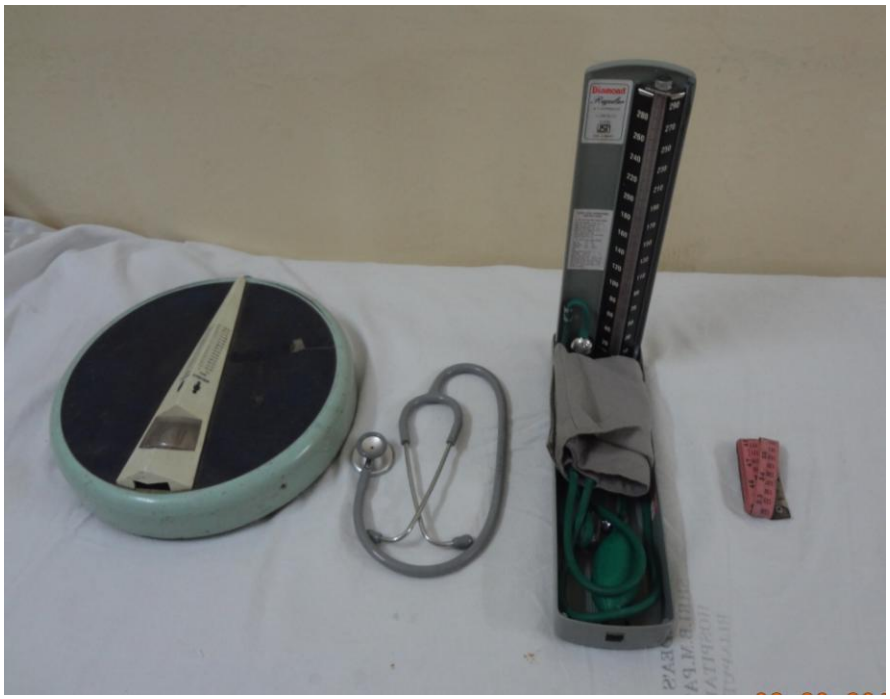


Fig:6:Recording of weight.

Fig 7: Technicon RA-XT Random access auto Analyzer

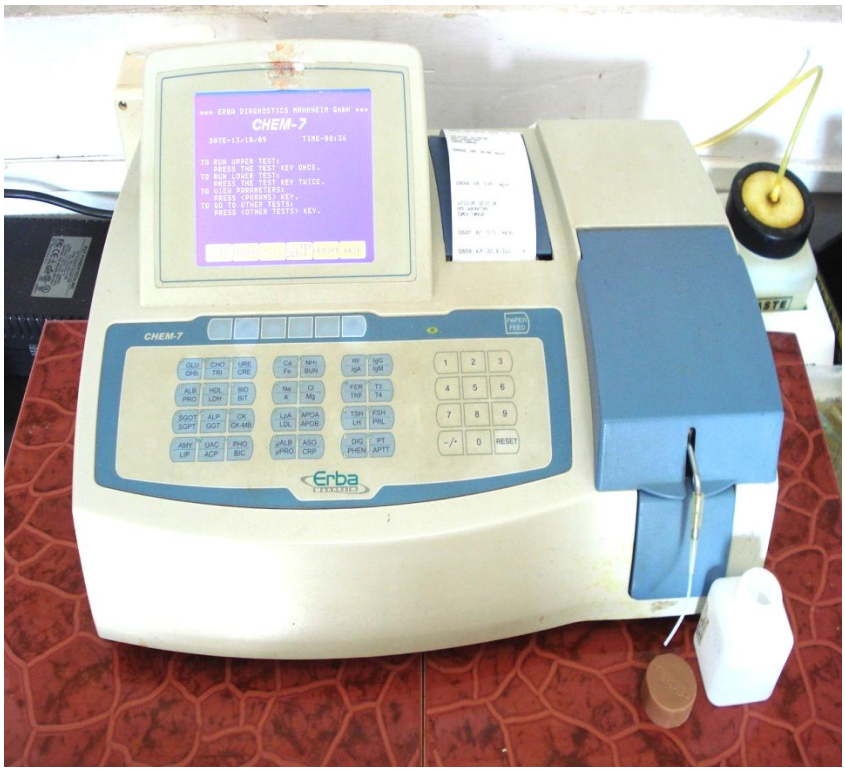


Fig 8: ERBA Diagnostic kit

RESULTS

This study consisted of 138 type2DM (males n=108, females n=30) and 138 normal healthy subjects (males n=108, females n=30) in the age group of 40-65 years. The BMI, WC, BP and lipid profile were compared between the normal and type2DM individuals. Also, the correlation between BMI and WC with BP, FBS and lipid profile was studied in both type2DM and normal individuals. The results obtained were expressed as mean ± standard deviation.

The mean age (yrs) of healthy group ranged between 52.60 ± 5.18 and that of diabetic group was between 53.09±6.52. There was no significant difference between the two groups with $p<0.468$ Hence the sample was age matched. (table 1, graph 1)

The mean BMI (Kg/m^2) in diabetic group was 26.15±3.22 and that of healthy group was 22.91±3.88. The diabetic group had significantly higher BMI when compared to healthy group with $p<0.001$ (table 2, graph 2)

The mean BMI (Kg/m^2) in diabetic male group was 26.12±3.35 and that of healthy male group was 23.08±3.83. The diabetic male group had significantly higher BMI when compared to healthy male group with $p<0.001$ (table 3, graph 3)

The mean BMI (Kg/m^2) in diabetic female group was 26.26±2.73 and that of healthy male group was 22.31±4.05. The diabetic female group had significantly higher BMI when compared to healthy female group with $p<0.001$ (table 4, graph 4)

The mean WC (cm) in diabetic group was 96.53±8.55 and that of healthy group was 85.03±9.72. The diabetic group had a significantly higher WC when compared to healthy group with $p<0.001$ (table 5, graph 5)

The mean WC (cm) in diabetic male group was 96.83±8.92 and that of healthy male group was 85.07±10.15. The diabetic male group had a significantly higher WC when compared to healthy male group with $p<0.001$ (table 6, graph 6)

The mean WC (cm) in diabetic female group was 95.43±7.12 and that of healthy female group was 84.90±8.20. The diabetic female group had a significantly higher WC when compared to healthy female group with $p<0.001$ (table 7, graph 7)

The mean systolic blood pressure (mm of Hg) in diabetic group was 134.76±11.54 and that of healthy group was 122.86±11.71. The diabetic group had significantly higher systolic blood pressure when compared to healthy group with $p<0.001$ (table 8, graph 8a)

The mean diastolic blood pressure (mm of Hg) in diabetic group was 86.72±6.61 and that of healthy group was 79.71±7.81. The DBP was significantly higher in diabetics when compared to healthy with $p<0.001$ (table 8, graph 8b).

The mean fasting blood sugar (mg/dl) in diabetic group was 148.75±36.61 and that of healthy group was 88.59±14.01. The FBS was significantly higher in diabetic group when compared to healthy group with $p<0.001$ (table 8, graph 8c)

The mean post prandial blood sugar (mg/dl) in diabetic group was 252.99±56.58 and that of healthy group was 104.20±16.98. The PPBS was significantly higher in diabetic group when compared to healthy group with $p<0.001$(table 8, graph 8d)

The mean cholesterol (mg/dl) in diabetics was 238.57±65.55 and that of healthy group was 174.05±21.55. The total cholesterol was significantly higher in diabetic group when compared to healthy group with $p<0.001$ (table 9, graph 9a)

The mean HDL-C (mg/dl) in diabetics was 34.98±6.68 and that of healthy group was 44.64±8.31 The HDL-C was significantly higher in healthy group when compared to diabetics with $p<0.001$ (table 9, graph 9b)

The mean triglycerides (mg/dl) in diabetics were 247.60±63.59 and that of healthy group was 134.07±28.29. The triglycerides was significantly higher in diabetics when compared to healthy group with $p<0.001$ (table9, graph9c)

The mean VLDL (mg/dl) in diabetics was 49.52±12.72 and that of healthy group was 26.86±5.66. The VLDL was significantly higher in diabetics when compared to healthy group with $P<0.001$ (table 9, graph 9d)

The mean LDL (mg/dl) in diabetics was 154.07±62.89 and that of healthy group was 102.58±21.76. The LDL was significantly higher in diabetics when compared to healthy group with $p<0.001$ (table 9, graph 9e)

Comparing abnormal levels of cholesterol (>200mg/dl), HDL-C (<40mg/dl), Triglycerides (>150mg/dl) and LDL-C (>130md/dl) in cases against controls, the percentage of lipid abnormalities was significantly higher in cases when compared to controls with $p<0.001$ (table 10, graph 10)

BMI positively correlated with total cholesterol, triglycerides, LDL and VLDL and it negatively correlated with HDL-C in diabetic group. Also BMI positively correlated with FBS and PPBS in diabetic group. (Table11)

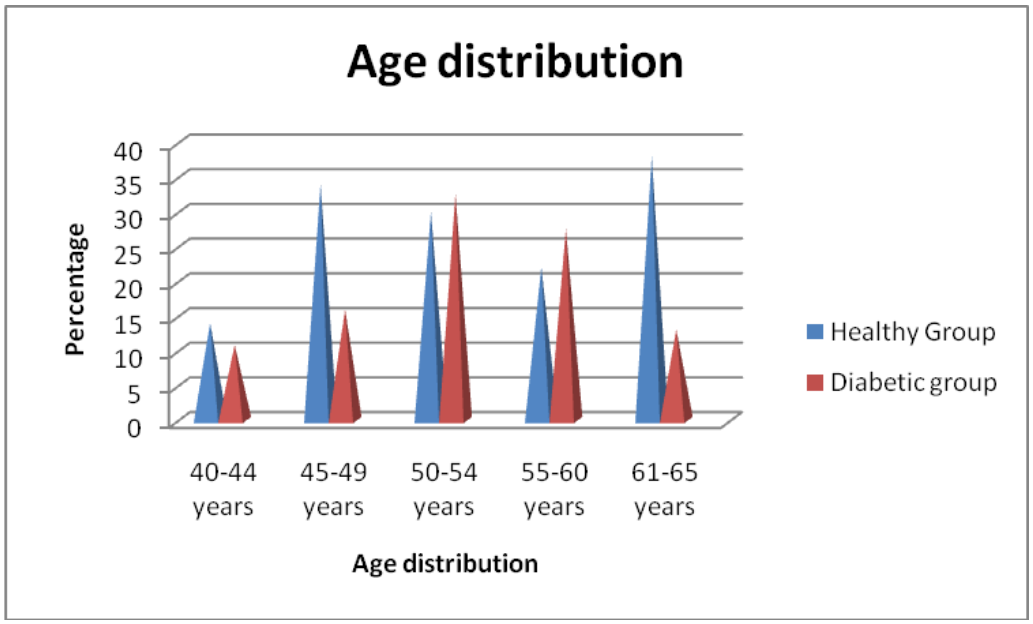
WC positively correlated with total cholesterol, triglycerides, LDL and VLDL and negatively correlated with HDL-C in Diabetic group. Also it had positive correlation with both SBP and DBP in diabetic group. WC had a small positive correlation with FBS and PPBS in diabetic group. (Table-12)

TABLES AND GRAPHS

Table 1: Comparison of age distribution

	Healthy group		Diabetic group	
	No	%	No	%
40-44	6	4.0	15	10.86
45-49	30	21.73	22	15.94
50-54	58	42.02	45	32.60
55-60	35	25.36	38	27.53
61-65	09	65.21	18	13.04
Mean ± SD	52.60±5.18		53.09±6.52	

Samples are age matched with $p<0.468$

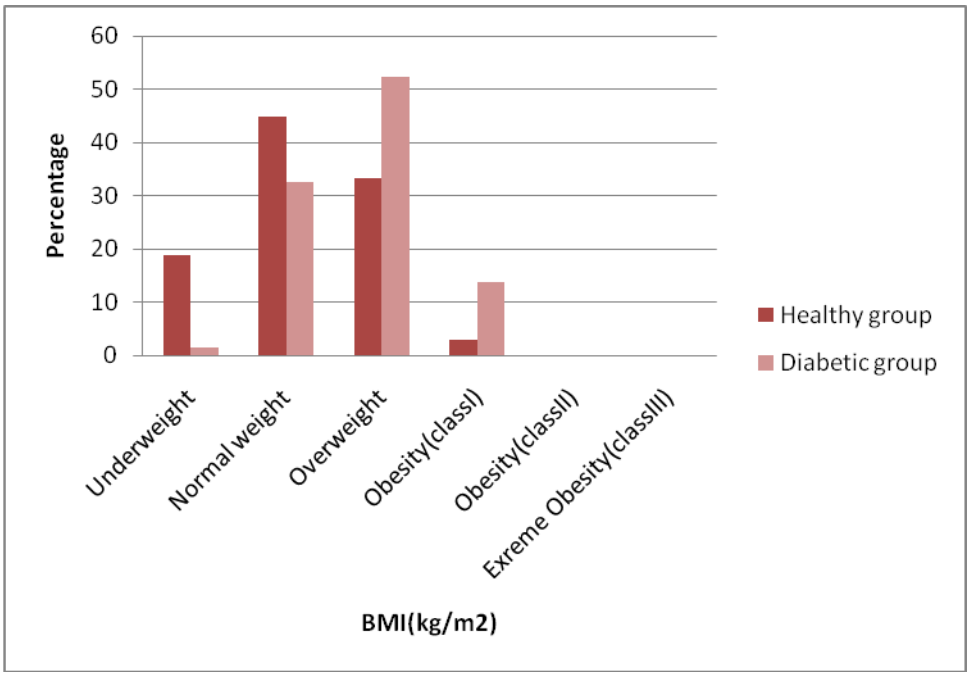


Graph-1 Comparison of age distribution

Table 2: Comparison of BMI .

BMI (kg/m^2)	Healthy group		Diabetic group	
	No	%	No	%
Underweight	26	18.84	02	1.44
Normal weight	62	44.92	45	32.60
Over weight	46	33.33	72	52.17
Obesity (class 1)	4	2.89	19	13.76
Obesity (class2)	0	0.0	0	0.0
Extreme obesity (class 3)	0	0.0	0	0.0
Total	138	100.0	138	100.0
Mean ± SD	22.91±3.88		26.15±3.22	

P<0.001*

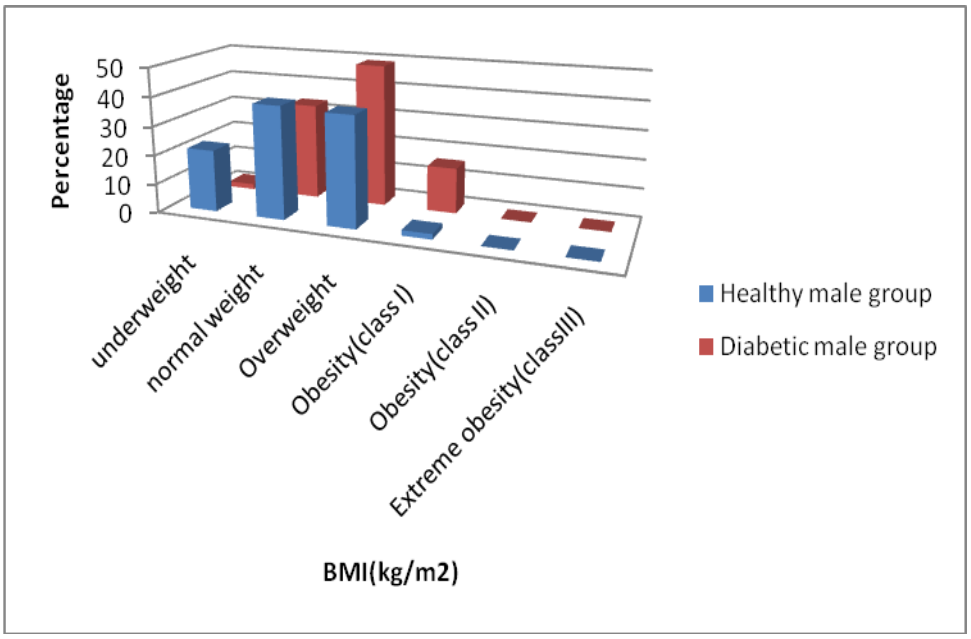


Graph-2 Comparison of BMI

Table 3.Comparison of BMI in study and control group (Male)

BMI (kg/m^2)	Healthy male group		Diabetic male group	
	No	%	No	%
Underweight	23	21.29	02	1.85
Normal weight	42	38.88	36	33.33
Over weight	41	37.96	53	49.07
Obesity (class 1)	02	1.85	17	15.74
Obesity (class2)	0	0.0	0	0.0
Extreme obesity (class 3)	0	0.0	0	0.0
Total	108	100.0	108	100.0
Mean ± SD	23.08±3.83		26.12±3.35	

P<0.001

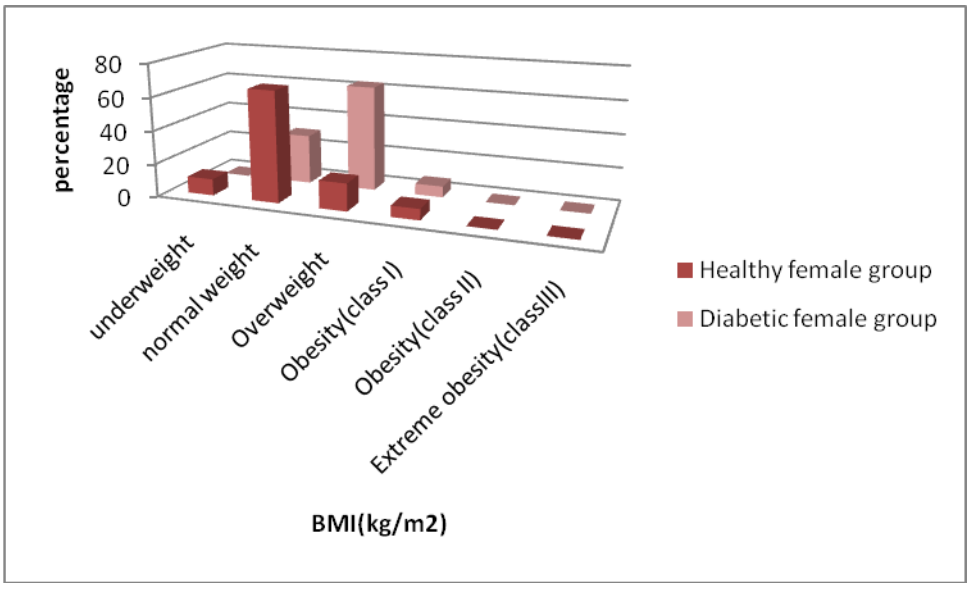


Graph-3 Comparison of BMI in study and control group (Male)

Table 4.Comparison of BMI in study and control group (Female)

BMI (kg/m^2)	Healthy female group		Diabetic female group	
	No	%	No	%
Underweight	03	10	00	00
Normal weight	20	66.66	09	30
Over weight	05	16.66	19	63.33
Obesity (class 1)	02	6.66	02	6.66
Obesity (class2)	0	0.0	0	0.0
Extreme obesity (class 3)	0	0.0	0	0.0
Total	30	100.0	30	100.0
Mean ± SD	22.31±4.05		26.26±2.73	

P<0.001

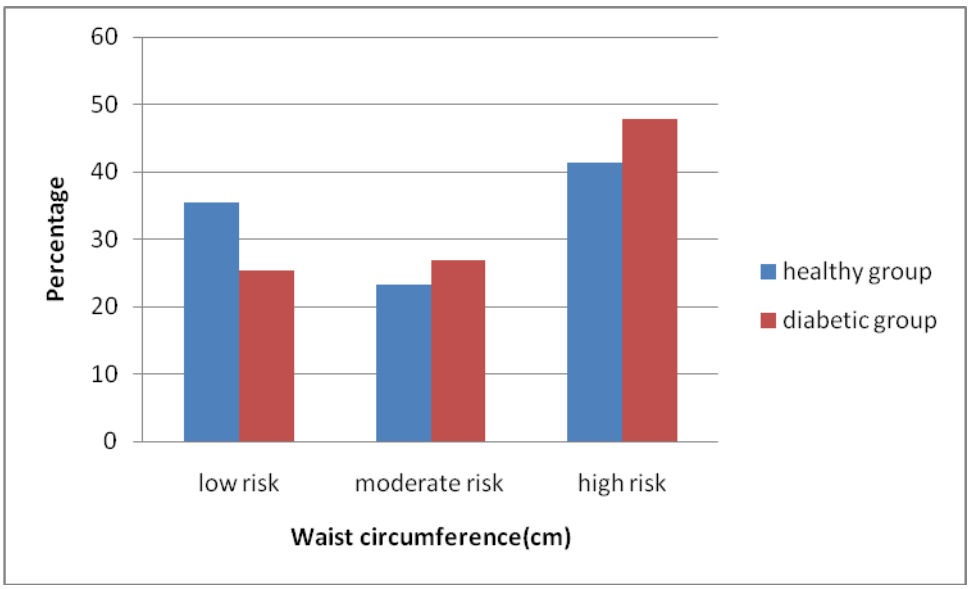


Graph-4 Comparison of BMI in study and control group (Female)

Table 5: Comparison of Waist circumference

Waist circumference (cm)	Healthy group		Diabetic group	
	No	%	No	%
Low risk	49	35.50	35	25.36
Moderate risk	32	23.18	37	26.81
High risk	57	41.30	66	47.82
Total	138	100.0	138	100.0
Mean ± SD	85.03±8.55		96.53±9.72	

p<0.001**

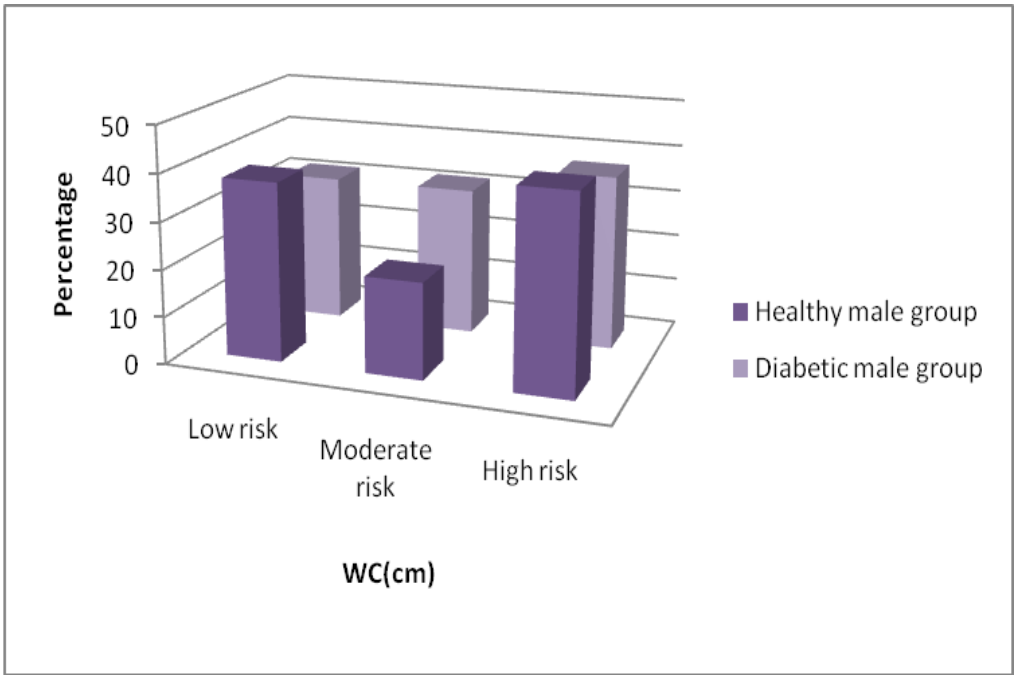


Graph-5 Comparison of Waist Circumference

Table 6.Comparison of Waist Circumference in study and control group (Male)

Waist circumference (cm)	Healthy male group		Diabetic male group	
	No	%	No	%
Low risk	41	37.96	34	31.48
Moderate risk	22	20.37	34	31.48
High risk	45	41.66	40	37.03
Total	108	100.0	108	100.0
Mean ± SD	85.07±10.15		96.83±8.92	

p<0.001**

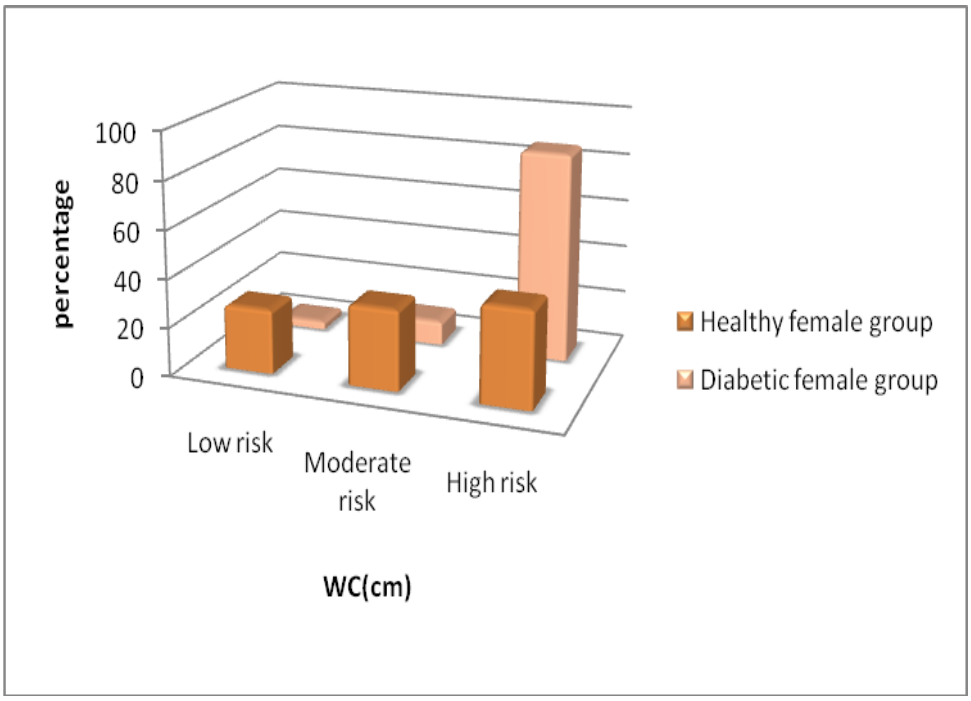


Graph-6 Comparison of Waist Circumference in study and control group (Male)

Table7 Comparison of Waist Circumference in study and control group

(Female)

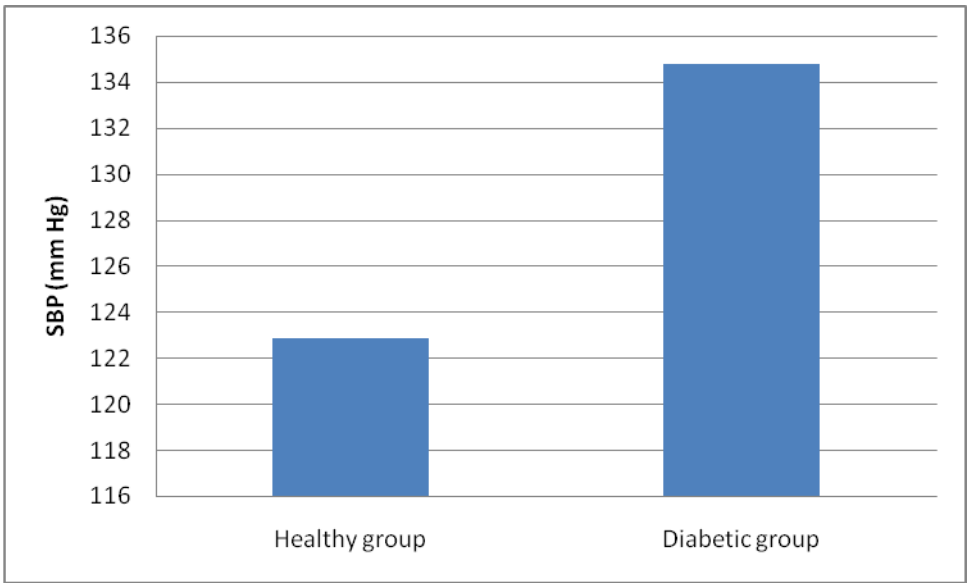
Waist circumference (cm)	Healthy female group		Diabetic female group	
	No	%	No	%
Low risk	08	26.66	01	3.33
Moderate risk	10	33.33	03	10
High risk	12	40	26	86.66
Total	30	100.0	30	100.0
Mean ± SD	84.90±8.20		95.43±7.12	



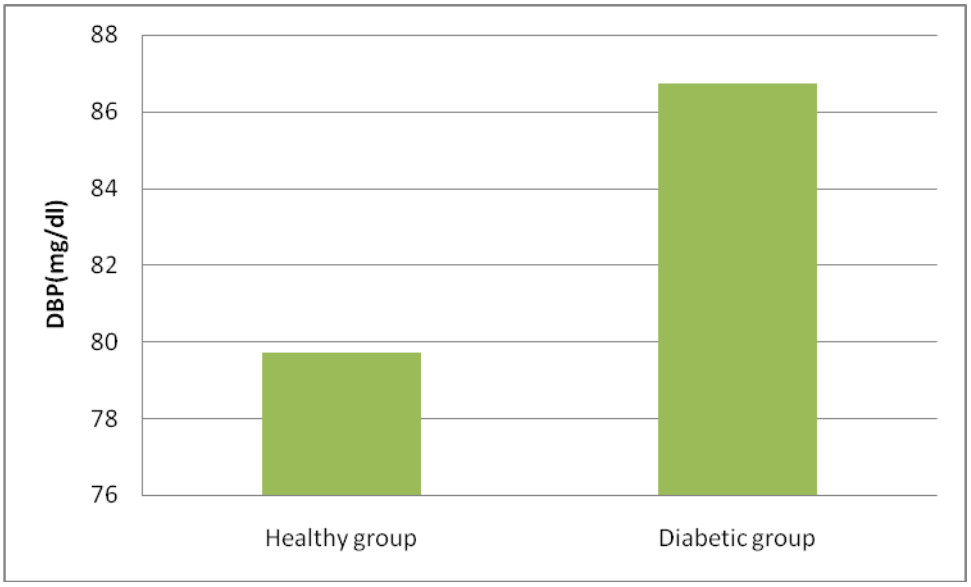
Graph-7 Comparison of Waist Circumference in study and control group (Female)

Table 8: Comparison of Blood Pressure and Fasting Blood Glucose

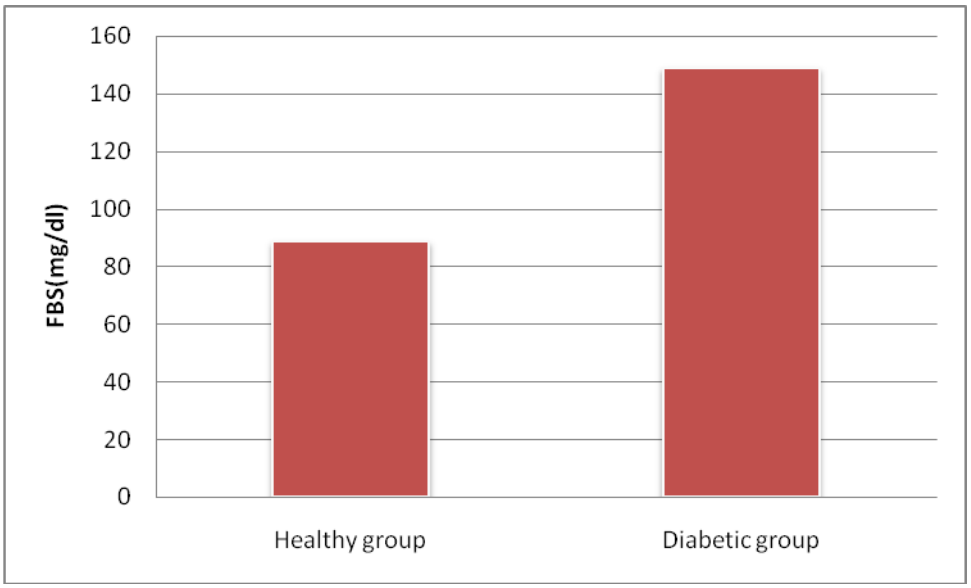
Variables	Healthy group	Diabetic group	P value
SBP (mm Hg)	122.86±11.71	134.76±11.54	<0.001**
DBP (mm Hg)	79.71±7.18	86.72±6.61	<0.001**
FBS (mg/dl)	88.59±14.01	148.75±36.61	<0.001**
PPBS(mg/dl)	104.20±16.98	252.99±56.58	<0.001**



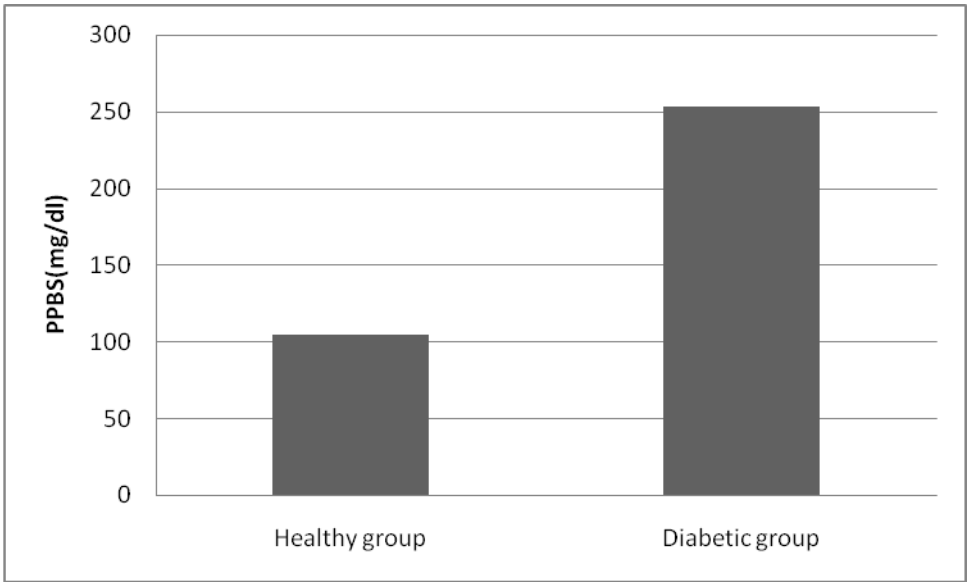
Graph -8 (a) Comparison of SBP between study and control groups.



Graph -8 (b) Comparison of DBP between study and control groups.



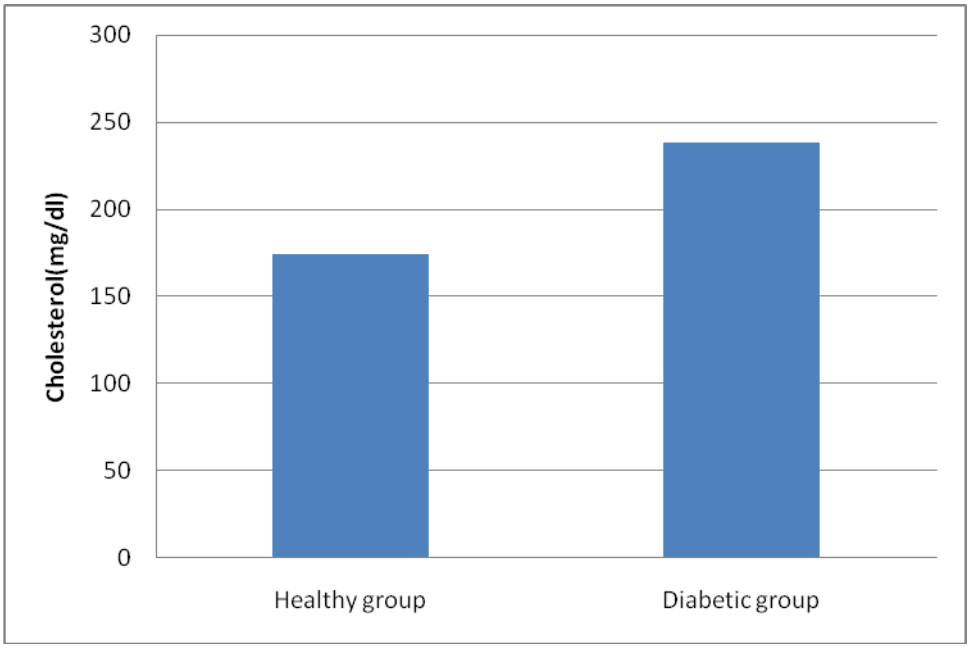
Graph -8(c) Comparison of FBS between study and control groups.



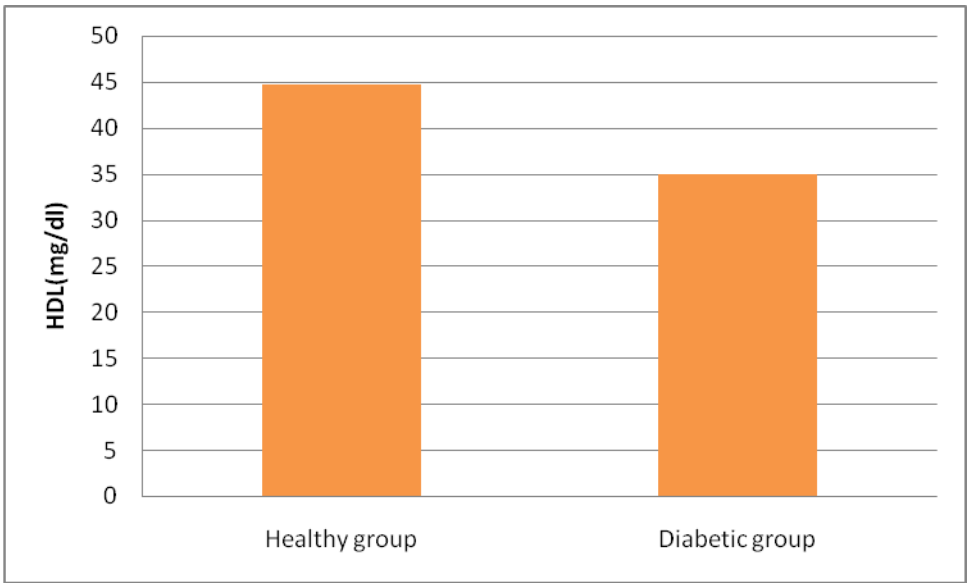
Graph- 8(d) Comparison of PPBS between study and control groups

Table-9 Comparison of Lipid Profile

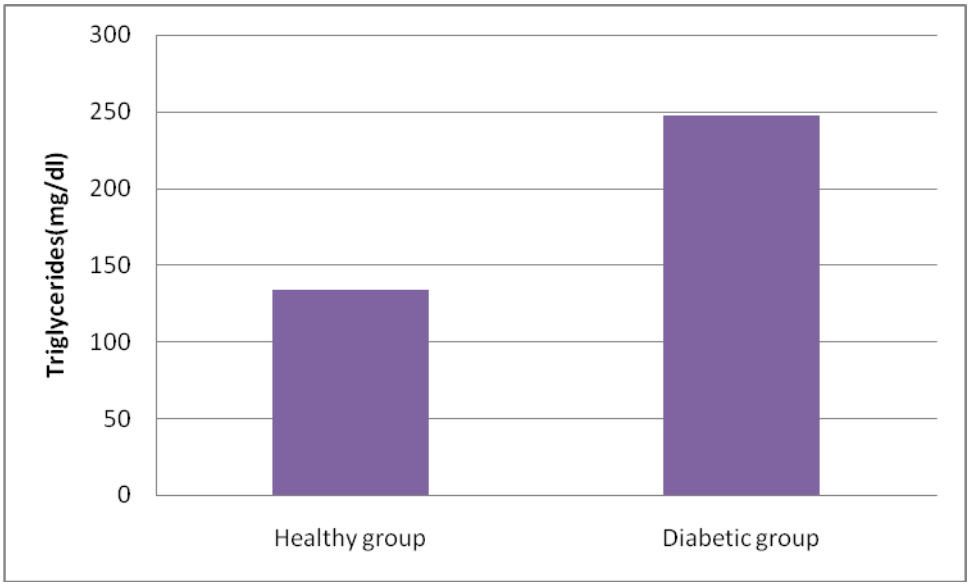
Lipid parameters	Healthy group	Diabetic group	Z values	P value
Cholesterol (mg/dl)	174.05±21.55	238.57±65.55	17.49	<0.001**
HDL (mg/dl)	44.64±8.31	34.98±6.68	-10.16	<0.001**
Triglycerides (mg/dl)	134.07±28.29	247.60±63.59	26.15	<0.001**
LDL (mg/dl)	102.58±21.76	154.07±62.89	14.09	<0.001*
VLDL (mg/dl)	26.86±5.66	49.52±12.72	26.09	<0.001**



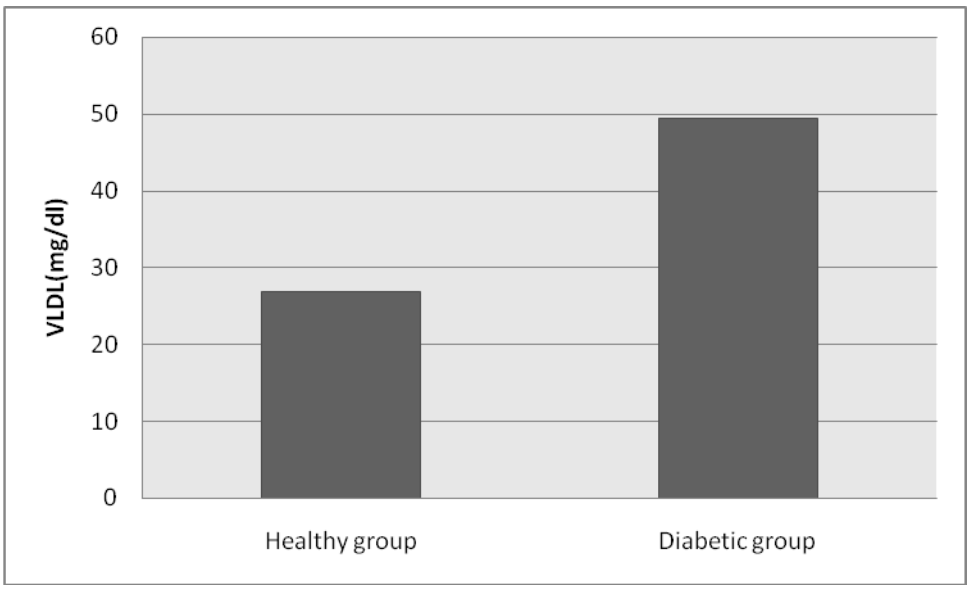
Graph-9(a) Comparison of total cholesterol between study and control groups



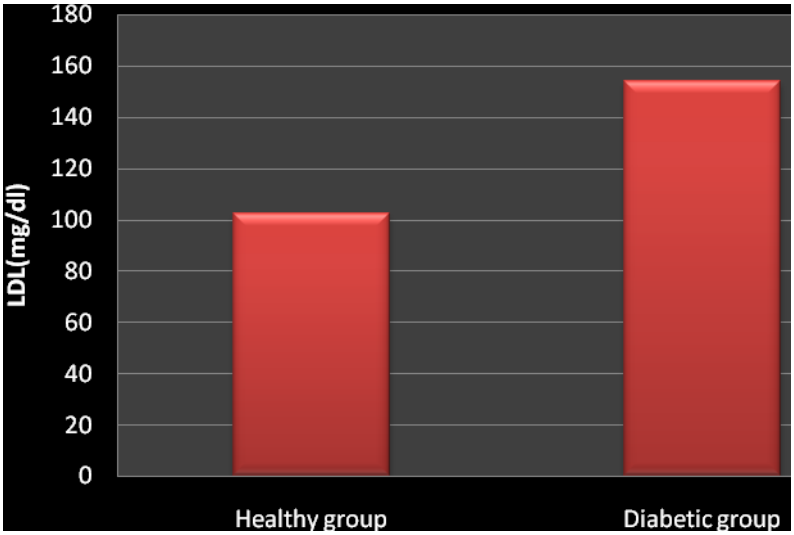
Graph-9(b) Comparison of HDL-C between study and control groups



Graph-9(c) Comparison of triglycerides between study and control groups



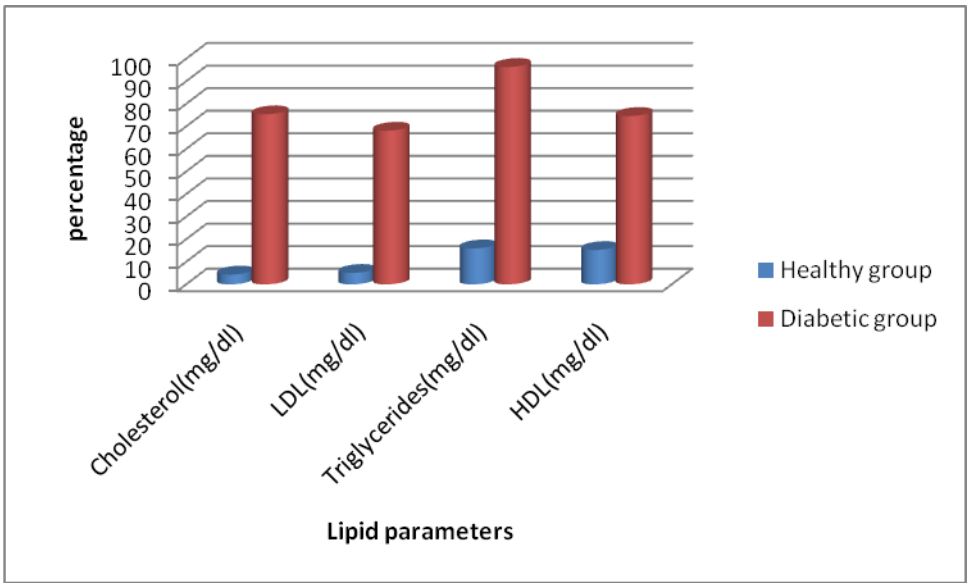
Graph -9(d) Comparison of VLDL between study and control groups.



Graph-9(e) Comparison of LDL-C between study and control groups

Table 10: Comparison of abnormal levels of Lipid Profile

Lipid parameters	Healthy group (n=138)	Diabetic group (n=138)	P value
Cholesterol (>200mg/dl)	06(4.34%)	104(75.36%)	<0.001**
HDL (<40 mg/dl)	21(15.21%)	103(74.63%)	<0.001**
Triglycerides(>150 mg/dl)	22(15.94%)	133(96.37%)	<0.001**
LDL (>130mg/dl)	7(5.07%)	94(68.11%)	<0.001**



Graph- 10 Comparison of lipid profiles between study and control groups.

Table 11: Correlation of BMI with BP, FBS and Lipid profile between study and control groups.

Pair	Diabetic	Healthy
	r value	r value
BMI vs Cholesterol	0.05	-0.01
BMI vs HDL	-0.03	0.05
BMI vs Triglycerides	0.05	-0.06
BMI vs VLDL	0.05	-0.04
BMI vs LDL	0.04	-0.02
BMI vs FBS	0.07	0.001
BMI vs PPBS	0.006	0.001
BMI vs SBP	0.003	-0.02
BMI vs DBP	0.05	-0.10

Table 12: Correlation of Waist circumference (WC) with BP, FBS and Lipid profiles between study and control groups.

Pair	Diabetic	Healthy
	r value	r value
WC vs Cholesterol	0.08	-0.17
WC vs HDL	-0.07	0.024
WC vs Triglycerides	0.12	-0.13
WC vs VLDL	0.09	-0.08
WC vs LDL	0.17	-0.16
WC vs FBS	0.08	-0.15
WC vs PPBS	0.02	0.06
WC vs SBP	0.03	-0.22
WC vs DBP	0.05	-0.11

DISCUSSION

In the present study, anthropometric measures (BMI, WC), BP and lipid profile were compared and their correlation was studied in patients with type2DM and normal healthy subjects of age group 40-65 years.

The present study showed a significant increase in BMI ($p<0.001$)) and WC ($p<0.001$) in diabetic group. BMI and WC were significantly higher in male diabetic group compared to male healthy group and similarly BMI and WC were significantly increased in female diabetic group compared to female healthy group. Both SBP (<0.001) & DBP ($p<0.001$) showed a significant increase in diabetic group. Serum levels of total cholesterol ($p<0.001$), Triglycerides ($p<0.001$), VLDL ($p<0.001$), LDL-C ($p<0.001$), FBS ($p<0.001$) and PPBS(<0.001) were significantly higher and serum HDL-C ($p<0.001$) was lower in diabetic group. Also there was a positive correlation of BMI and WC with Total cholesterol, Triglycerides, LDL and VLDL and negative correlation with HDL-C in diabetics. Both BMI and WC had a positive correlation with FBS and PPBS. WC and BMI positively correlated with SBP and DBP in diabetic group.

BMI and WC are predictors of cardiovascular disease. BMI has been used as a measure of general obesity and WC has been used as a measure of abdominal obesity. In the present study, diabetic group had significantly higher BMI when compared to healthy group with $p<0.001$ reflecting general obesity in diabetics. Also 47.82% of diabetics had high WC $\geq$ 102cm suggesting that abdominal obesity was higher in diabetic group when compared to healthy group. Obesity, particularly abdominal obesity increases the risk of CAD. Abdominal obesity increases the risk of other risk factors or atherosclerosis: high blood pressure, type 2 diabetes and high cholesterol levels (61). Both BMI and WC independently contributes to the prediction of total,

and abdominal subcutaneous and visceral fat and thus recommends health care practitioners routinely use both anthropometric variables to identify those at increased health risk (62).

Dyslipidemia is one of the major risk factors for cardiovascular disease in diabetes mellitus. The characteristic features of diabetic dyslipidemia are a high plasma triglyceride concentration, low HDL cholesterol concentration and increased concentration of small dense LDL-cholesterol particles. The lipid changes associated with diabetes mellitus are attributed to increased free fatty acid flux secondary to insulin resistance.

Accelerated coronary and peripheral vascular atherosclerosis is one of the most common and serious complications of long term diabetes mellitus (14). Also with other risk factors such as hypertension, smoking, obesity increasing importance has been given to secondary dyslipidemias in the causation of accelerated atherosclerosis. Dyslipidemia as a metabolic abnormality is frequently associated with diabetes mellitus. Its prevalence is variable, depending on the type and severity of diabetes glycemic control, nutritional status, age and other factors.

This study clearly shows that all lipid fractions (TG, TC, LDL-C, and VLDL-C) are abnormally elevated in type2 DM when compared to controls except HDL-C. This study is in accordance with previous studies which also showed that in type2DM there is significant elevation of TG, TC, LDL-C, VLDL-C and decrease in HDL-C (89-91). The reasons for increased lipid fractions in type2DM patients are due to increase in the incidence of the obesity, sedentary life, lack of physical activity and the diet.

Hypertension (HTN) is a very common co morbid condition in diabetes and accounts for up to 85% of excess CVD risk. When HTN coexist with diabetes, the risk of development of CVD is doubled. Patients of type-2DM are prone to develop hypertension which accelerates cardiac, renal, and cerebral dysfunctions which are leading causes of death. 70% of deaths in diabetics occur due to macro vascular complications like myocardial infarction (MI), unstable angina, heart failure (HF), sudden cardiac death, stroke, and neglected gangrene due to occlusion of large arteries of extremities (71). In the present study, there was significant increase in SBP and DBP in diabetics when compared to healthy individuals. This finding of the study is in accordance with earlier work of Ram B Singh et al who showed the high prevalence of hypertension and CAD in type2DM when compared to normal's (92).

The association between lipid profile and body fat are important predictors for metabolic disturbances like dyslipidaemia, hypertension, diabetes, CVD and hyperinsulinemia. Any alteration in the level of lipids in the body makes the individuals more prone to develop these diseases. In the present study, correlation of both BMI and WC were studied with lipid profile, BP, FBS and PPBS. Both BMI and WC showed a positive correlation with TC, triglycerides, VLDL, FBS, and PPBS. Also BMI showed a negative correlation with HDL-C. This study is in agreement with previous studies that also showed a negative correlation of body fat with HDL-C and a positive correlation with VLDL, TC, FBS and triglycerides (93-96). In the present study, correlation of BMI and WC with blood pressure showed a significant positive correlation of WC and BMI with BP. This finding of the study is in accordance with Bekatas Murat et al who in their linear regression study to identify which anthropometric measures are closely related to blood pressure showed that WC was found to be an independent risk factor for blood pressure in men (72).

Some of the possible mechanisms contributing to dyslipidemia support the concept that visceral adipocytes release an excess amount of FFAs and which are very resistant to antilipolytic effect of insulin. FFAs are important regulators of glucose metabolism and that elevated FFAs are associated with insulin resistance at the level of liver and muscles. It is been postulated a preferential influx of FFAs via portal circulation to the liver can induce or augment hepatic insulin resistance, in particular by enhancing gluconeogenesis. Also a recent study showed that obese persons have greater release of FFAs and glycerol into portal circulation than do non obese persons. This easily mobilized fat draining directly into portal vein could lead to increased synthesis of VLDL. Over production of triglyceride- rich lipoproteins and impaired clearance by lipoprotein lipase leads to hypertriglyceridemia in diabetes (32, 93-95).

Some of the possible physiological mechanisms contributing to HTN in type2DM include insulin is a vasodilator with secondary effects on sodium reabsorption in the kidney. However, in the setting of insulin resistance, the vasodilatory effect of insulin is lost, but the renal effect on sodium reabsorption is preserved. Also in the endothelium, this may cause an imbalance between the production of Nitric Oxide and secretion of endothelin-1, leading to decreased blood flow (3).

Thus from this study it is evident that type2DM is often associated with obesity, hypertension and dyslipidemia when compared to healthy group which in turn increases the risk factor for development of CVD. As good glycemic control in diabetes is shown to keep the lipid levels in near normal range, it appears important to aim at critical control of diabetes mellitus to prevent or at least postpone the onset of various complications As is wisely said “prevention is better than cure” we need cautious evaluation of various risk factors so that appropriate measures can be taken

in order to prevent grave sequelae later on. Thus efforts should be made to continuously educate the diabetics about its management, dietary and lifestyle modifications.

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CONCLUSION

We conducted a cross-sectional study to evaluate Body mass index, Waist circumference, Blood pressure and Lipid profile in Type 2 Diabetes mellitus patients (n=138) in the age group of 40-65years. The study was compared with age and sex matched non-diabetic (control) subjects (n=138) and same was analysed for statistical significance.

1. We observed that Body mass index, Waist circumference were significantly higher in type 2 Diabetes mellitus patients when compared to healthy individuals.

2. Systolic blood pressure and Diastolic blood pressure were significantly higher in type 2 Diabetes mellitus patients when compared to healthy individuals.

3. We found that total cholesterol, triglycerides, LDL-C, VLDL except HDL-C were significantly higher in type 2 Diabetes mellitus patients compared to healthy individuals and

4. We also observed that Body mass index and Waist circumference had a positive correlation with total cholesterol, triglycerides, LDL-C, VLDL, FBS, PPBS and negative correlation with HDL-C in diabetics. Also, Waist circumference and Body mass index had a positive correlation with both Systolic blood pressure and Diastolic blood pressure in diabetic patients.

SUMMARY

This study consisted of 138 type2DM and 138 normal healthy subjects in the age group of 40-65 years. The BMI, WC, BP and lipid profile were compared between type2DM patients and healthy normal individuals. Also, the correlation between BMI with lipid profile and WC with lipid profile was studied in both type2DM and normal individuals.

In the present study, diabetic group had significantly higher BMI and WC when compared to healthy group suggesting the higher prevalence of general obesity in particular abdominal obesity in diabetic group.

This study clearly shows that all lipid fractions (TG, TC, LDL-C, and VLDL-C) are abnormally elevated in type2DM when compared to controls except HDL-C suggesting that type2DM has a real impact on lipid metabolism.

In the present study, there was significant increase both in SBP and DBP in diabetics when compared to healthy individuals.

In the present study, BMI and WC had a positive correlation with total cholesterol, triglycerides, LDL-C, VLDL, FBS, PPBS and negative correlation with HDL-C in diabetics. Also, WC and BMI had a positive correlation with both SBP and DBP in diabetic patients.

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ANNEXURE-I

B.L.D.E.U'S SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586103
INSTITUTIONAL ETHICAL COMMITTEE

Ethical Committee BIJAPUR-586103 OUT WARD No 12310 Date 19/10/10 Shri B. M. Patil Med. Coll.

DR.M.S.BIRADAR
CHAIRMAN .I.E.C.
BLDEU'S SHRI: B.M.PATIL MEDICAL COLLEGE
BIJAPUR-586103

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 19-10-2010 at 10-30 am to scrutinize the Synopsis/Research projects of post graduate student/undergraduate student/Faculty members of this college from ethical clearance point of view. After scrutiny the following original/corrected & revised version Synopsis of the Thesis/Research project has been accorded Ethical Clearance.

Title A Study of correlation between body mass index, waist circumference, blood pressure and lipid profile in Type 2 diabetes mellitus.

Name of P.G. /U.G.Student /Faculty member Dr. Santosh Kumar Palekar Dept of Physiology.

Name of Guide Dr. Manjunatha Aithal Assoc Prof Physiology

DR.M.S.BIRADAR
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE

Following documents were placed before E.C.for securitization:
1) Copy of Synopsis/Research project
2) Copy of informed consent form
3) Any other relevant document's

ANNEXURE-II

B. L. D. E. A'S SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, BIJAPUR

RESEARCH INFORMED CONSENT FORM

Title of the project: **" A STUDY OF CORRELATION BETWEEN BODY MASS INDEX , WAIST CIRCUMFERENCE , BLOOD PRESSURE AND LIPID PROFILE , IN TYPE 2 DIABETES MELLITUS ".**

Principal investigator/ P.G.Guide's name: DR. MANJUNATHA AITHALA MD

PROFESSOR AND HEAD

DEPARTMENT OF PHYSIOLOGY

1: PURPOSE OF RESEARCH:

I have been informed that this study will test influence of body fat distribution on lipid profile in Type 2 Diabetes Mellitus patients . This study will be useful academically as well as for clinically to find out association between Glycaemic status with Body mass index , Waist circumference , Blood pressure and Lipid profile in Type 2 Diabetes Mellitus .

2: PROCEDURE:

I understand that , the procedure of the study will involve recording of various physiological & physical parameters. The procedure will not interfere with any of my physiological parameters and they are non invasive , excepting for determination of Fasting blood sugar level(FBS), post prandial blood sugar (PPBS) and Lipid profile .

3: RISK AND DISCOMFORTS:

I understand a study on correlation between body mass index , blood pressure , waist circumference and lipid profile on Type 2 Diabetes Mellitus patients will not cause any discomfort to me and do not involve any risk to my health.

4: BENEFITS:

I understand that my participation in the study may not have a direct benefit to me but this may have a potential beneficial effect in the field of DIABETOLOGY in future.

5: CONFIDENTIALITY:

I understand that medical information produced by this study will become part of institutional records and will be subject to the confidentiality and privacy regulation of the said institute. Information of a sensitive personal nature will not be a part of medical record, but will be stored in investigators research file and identified only by a code number. The code key connecting name two numbers will be kept in a separate secured location.

If the data are used for publication in the medical literature and for teaching purposes no names will be used and other identities such as photographs, audio and video tapes will be used only with my special written permission. I understand I may see the photographs and the video tapes and have the audio tapes before giving this permission.

6: REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Concerned researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study which might influence my continued participation. If during the study or later, I wish to discuss my participation in all concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful re-reading.

7: REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that researcher may terminate my participation in this study at any time after she/he has explained the reasons for doing so and had helped arrange for my continued care by my physician or physical therapist if this is appropriate.

8: INJURY STATEMENT:

I understand that in unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, then medical treatment will be available to me, but no further compensation would be provided.

I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____(subject's/relevant guardian) the purpose of the research, the procedure required and the possible risk and benefits to the best of my ability.

Investigator/ PG (Guide) Date

I confirm that _____ (Name of the P.G. Guide /Chief researcher) has explained to me the purpose of research, the study procedure that I will undergo, and the possible risk and discomforts as well as benefits that I may experience. Alternative to my participation in the study have also been to give my consent from. Therefore I agree to give consent to participate as a subject and this research project.

Participant / Guardian Date:

Witness to signature Date:

Modified from Portney L.G. Watkins M.P., in Foundation of Clinical Research, Second Edition, New Jersey, Prentice Hall Health 2000. (APPENDIX – E).

ANNEXURE-III

B. L. D. E. U'S SHRI B.M. PATIL MEDICAL COLLEGE , HOSPITAL AND RESEARCH CENTRE, BIJAPUR

DEPARTMENT OF PHYSIOLOGY

CLINICAL PROFORMA

"A STUDY OF CORRELATION BETWEEN BODY MASS INDEX, WAIST CIRCUMFERENCE, BLOOD PRESSURE AND LIPID PROFILE IN TYPE 2 DIABETES MELLITUS "

Name: **Case No. :**

Age: **IP/OP No.:**

Sex

Address:

Occupation: **phone no –**

HOPI:

Past history:

Family history:

Drug history:

Personal history:

General Physical Examination:

Built **Temperature** **Pallor**

Cyanosis **Edema** **Abnormal Pigmentation**

JVP **Lymphadenopathy**

PR(beats/min): **BP(mm of Hg):** **RR(cycles/min):**

Wt(cm): **Ht(cm):** **Waist: Circumference(cm)** **Hip: circumference(cm)**

Waist:hip ratio:

Systemic Examination:

Cardiovascular system:

Respiratory system:

Central nervous system:

Per abdomen:

PARAMETERS FOR STUDY

1. Body mass index(kg/m^2):

2. Waist circumference(cm):

3. Blood pressure(mm of Hg)

4. Lipid profile:

*** Total Cholesterol (mg/dl):**

*** HDL Cholesterol (mg/dl):**

*** Triglycerides (mg/dl):**

*** VLDL (mg/dl):**

*** LDL(mg/dl):**

Other investigations:

FBS(mg/dl):

PPBS(mg/dl):

Signature of Guide **Signature of PG student** **Signature of HOD**

ANNEXURE IV
List of Diabetics

Sl no	Name	Age yrs	sex	Duration (yrs)	Wt (kg)	Ht (cm)	BSA	BMI Kg/m2	PR	RR	SBPmm/Hg	DBPmm/Hg	WC cm	HC cm	W:H	FBS mg/dl	PPBS mg/dl	CHO mg/dl	HDL mg/dl	TRI mg/dl	VLDL mg/dl	LDL mg/dl	LDL:HDL mg/dl	Family history
1	Mallappa	56	M	8	68	160	1.88	26.56	82	12	142	88	104	95	1.09	139	204	306	32	326	65.2	208.8	6.52	f/h F
2	yusuf	55	M	10	63	158	1.64	25.3	72	32	128	86	110	96	1.14	140	196	220	26	228	45.6	148.4	5.7	No f/h
3	Gouramma	52	F	7	60	145	1.5	28.57	82	20	148	98	100	98	1.02	136	300	251	32.4	310	62	156.6	4.83	No f/h
4	Suresh	50	M	10	70	163	1.74	26.4	74	20	146	94	98	102	0.96	175	159	225	37	220	44	144	3.89	No f/h
5	Drakkshani	45	F	9	65	169	1.76	22.8	76	18	150	94	92	100	0.92	112	132	233	35	350	70	128	3.65	f/h M
6	Shantamma	50	F	12	66	161	1.7	25.48	76	30	138	90	90	95	0.95	120	181	289	26	260	52	211	8.11	No f/h
7	Ashok	45	M	10	64	165	1.72	23.52	82	18	146	92	90	95	0.94	172	347	210	31	249	49.8	129.2	4.16	Nof/h
8	B.M.Nimbargi	48	M	8	76	167	1.88	27.33	76	24	154	92	106	98	1.08	147	276	222	25	398	79.6	117.4	4.69	f/h of HTN
9	Prabha	55	F	9	67	169	1.76	23.5	82	24	126	86	82	98	0.83	135	200	236	37	168	33.6	165.4	4.47	Both+
10	Siddharedd y	59	M	5	75	170	1.88	25.95	70	18	124	86	98.5	98	1	143	333	256	31	260	52	173	5.58	Both +
11	Daddamani	42	M	6	72	168	1.82	25.53	66	24	142	84	109	98	1.11	153	165	225	30	255	51	144	4.8	F +
12	Mallikarjun	60	M	10	65	165	1.72	23.89	74	20	148	94	88	95	0.92	193	342	306	34	326	65.2	206.8	6.082	No f/h
13	Shailaja	55	F	9	76	173	1.9	25.41	76	24	130	84	105	94	1.11	160	319	242	35	285	57	150	4.28	No f/h
14	Hemanth	40	M	4	68	164	1.74	23.52	82	16	126	86	90	108	0.83	117	262	144	25	304	60.8	58.2	2.32	No f/h
15	Mahesh	42	M	10	81	164	1.9	30.22	76	20	120	90	88	90	0.97	229	339	249	26	256	51.2	171.8	6.6	M+
16	Girish	46	M	15	78	168	1.88	24.11	86	20	134	84	102	100	1.02	300	184	200	42	264	52.8	105.2	2.504	No f/h
17	Siddramesh	44	M	1	68	163	1.72	25.66	72	18	130	90	94	92	1.02	108	153	255	46	260	52	157	3.413	Nof/h
18	Manjunath	54	M	10	81	160	1.84	31.64	80	20	126	84	97	93	1.04	194	309	231	36	180	36	159	4.416	f/h of HTN
19	Suryakanth	56	M	4	54	167	1.58	19.42	78	22	120	84	88	98	0.89	154	342	244	30	186	37.2	176.8	5.893	Both+
20	Sittaya	58	M	12	67	163	1.72	25.28	74	18	134	86	90	78	1.15	159	252	248	22	228	45.6	180.4	8.2	f/h F
21	Kamlesh.G.	60	M	4	56	148	1.48	25.57	72	22	112	74	110	94	1.17	104	279	132	40	154	30.8	61.2	1.53	No f/h
22	Balakrishn a	50	M	6	65	152	1.6	28.13	80	20	118	80	104	90	1.15	170	281	160	47	228	45.6	67.4	1.434	F+
23	Rajshekar	64	M	5	48	154	1.44	20.25	80	30	112	84	88	94	0.93	130	153	230	40	178	35.6	154.4	3.86	f/h M
24	Rajiv	62	M	8	58	159	1.58	23.01	82	18	140	74	94	102	0.92	160	309	238	46	349	69.8	122.2	2.656	No f/h
25	Ganesh	64	M	8	65	149	1.56	29.27	84	20	140	86	102	94	1.08	142	342	193	30	352	70.4	92.6	3.086	No f/h
26	Sunil	58	M	2	68	154	1.66	28.69	84	32	144	84	110	98	1.12	102	252	162	24	230	46	92	3.83	No f/h
27	Yograj	52	M	6	65	158	1.64	26.1	74	20	134	80	97	102	0.95	224	279	446	34	195	39	373	10.97	Nof/h
28	Anwar	50	M	15	69	148	1.62	31.5	80	12	144	84	98	102	0.96	142	281	252	43	176	35.2	173.8	4.041	f/h of HTN F
29	Sangamesh. k	54	M	3	67	155	1.68	27.91	84	12	140	90	102	100	1.02	128	196	203	30	332	66.4	106.6	3.55	Both+
30	Srijeeth	64	M	4	47	157	1.44	19.1	82	24	140	74	99	105	0.94	126	227	144	40	232	46.4	57.6	1.44	Both +
31	Mohan	59	M	12	57	151	1.52	25	74	24	130	90	108	95	1.13	146	167	222	39	150	30	153	3.923	No f/h
32	Prabhuling	64	M	8	48	148	1.4	21.91	72	18	126	80	85	100	0.85	120	161	156	44	281	56.2	55.8	1.268	Nof/h
33	Sandeep	58	M	7	50	150	1.44	22.22	84	12	136	84	84	100	0.84	168	321	156	38	180	36	82	2.157	f/h of HTN m
34	Shrikanth	60	M	10	58	150	1.52	25.77	60	20	130	84	104	95	1.09	126	184	225	40	170	34	151	3.775	Both+

35	**Umesh.J.**	**50**	**M**	**10**	**72**	**154**	**1.7**	**30.37**	**78**	**24**	**128**	**86**	**94**	**98**	**0.95**	**120**	**209**	**130**	**44**	**160**	**32**	**54**	**1.227**	**f/h F**
36	**Pandurang**	**44**	**M**	**8**	**69**	**176**	**1.82**	**22.33**	**80**	**14**	**138**	**88**	**96**	**98**	**0.97**	**160**	**193**	**200**	**38**	**150**	**30**	**132**	**3.47**	**f/h F**
37	**Chandrakanth**	**58**	**M**	**6**	**84**	**170**	**1.94**	**29.06**	**72**	**18**	**154**	**86**	**98**	**94**	**1.04**	**180**	**181**	**300**	**50**	**220**	**44**	**206**	**4.12**	**No f/h**
38	**Basavaraj. M**	**40**	**M**	**5**	**65**	**168**	**1.72**	**23.04**	**76**	**28**	**134**	**80**	**82**	**100**	**0.82**	**180**	**269**	**350**	**40**	**290**	**58**	**252**	**6.3**	**No f/h**
39	**Somshekar**	**54**	**M**	**7**	**74**	**169**	**1.82**	**25.96**	**82**	**22**	**140**	**80**	**100**	**92**	**1.08**	**104**	**329**	**400**	**29**	**308**	**61.6**	**309.4**	**10.66**	**No f/h**
40	**Raseed**	**56**	**M**	**20**	**70**	**156**	**1.7**	**28.8**	**82**	**16**	**144**	**70**	**94**	**98**	**0.95**	**140**	**330**	**300**	**30**	**280**	**56**	**214**	**7.13**	**f/h M**
41	**Wasim.P**	**48**	**M**	**3**	**68**	**158**	**1.68**	**27.3**	**76**	**18**	**130**	**84**	**102**	**94**	**1.08**	**120**	**196**	**240**	**36**	**233**	**46.6**	**157.4**	**4.372**	**No f/h**
42	**Allimudin**	**46**	**M**	**7**	**60**	**154**	**1.58**	**25.31**	**82**	**14**	**142**	**84**	**96**	**94**	**1.02**	**208**	**274**	**220**	**41**	**160**	**32**	**147**	**3.58**	**F+**
43	**Amog.H**	**40**	**M**	**12**	**65**	**166**	**1.74**	**23.63**	**84**	**18**	**150**	**86**	**86**	**96**	**0.89**	**134**	**296**	**212**	**34**	**154**	**30.8**	**147.2**	**4.32**	**f/h M**
44	**Sushil**	**50**	**M**	**14**	**64**	**163**	**1.68**	**24.15**	**66**	**18**	**130**	**74**	**80**	**100**	**0.8**	**222**	**204**	**372**	**36**	**352**	**70.4**	**265.6**	**7.37**	**No f/h**
45	**Ambekar**	**42**	**M**	**12**	**68**	**165**	**1.76**	**25**	**76**	**16**	**110**	**86**	**102**	**100**	**1.02**	**174**	**258**	**273**	**40**	**226**	**45.2**	**187.8**	**4.69**	**No f/h**
46	**Azimsab**	**40**	**M**	**6**	**70**	**166**	**1.78**	**25.45**	**76**	**18**	**150**	**84**	**92**	**96**	**0.95**	**134**	**288**	**257**	**39**	**223**	**44.6**	**173.4**	**4.446**	**F +**
47	**Bhimanna**	**50**	**M**	**10**	**76**	**170**	**1.9**	**26.29**	**72**	**16**	**140**	**88**	**104**	**102**	**1.01**	**154**	**300**	**208**	**32**	**243**	**48.6**	**127.4**	**3.98**	**No f/h**
48	**Wishwa**	**48**	**M**	**4**	**60**	**162**	**1.64**	**22.9**	**78**	**30**	**152**	**88**	**82**	**95**	**0.86**	**184**	**269**	**226**	**32**	**230**	**46**	**148**	**4.62**	**No f/h**
49	**Devgouda**	**60**	**M**	**5**	**68**	**161**	**1.72**	**26.25**	**72**	**12**	**144**	**86**	**108**	**98**	**1.1**	**273**	**285**	**208**	**28**	**238**	**47.6**	**132.4**	**4.72**	**No f/h**
50	**Anand**	**54**	**M**	**4**	**81**	**165**	**1.9**	**29.77**	**80**	**22**	**128**	**70**	**102**	**98**	**1.04**	**144**	**210**	**300**	**34**	**326**	**65.2**	**200.8**	**5.9**	**M+**
51	**Jagadish**	55	M	12	60	146	1.52	28.16	84	16	148	88	106	95	1.11	238	304	443	32	229	45.8	365.2	11.41	F+
52	**ShankarGouda**	52	M	15	65	142	1.54	32.33	74	18	142	90	104	108	0.96	140	284	293	32	310	62	199	6.21	M+
53	**Sushma**	54	F	6	58	146	1.48	27.23	84	18	146	96	100	100	1	139	184	308	28	220	44	236	8.42	M+
54	**Farooq**	50	M	12	68	156	1.66	27.98	74	16	154	94	98	93	1.05	175	311	219	34	250	50	135	3.97	both
55	**Girija**	45	F	9	69	153	1.68	29.48	8	16	138	96	94	78	1.21	98	210	251	32	264	52.8	166.2	5.19	F+
56	**Gourishankar**	46	M	14	47	149	1.38	21.17	78	22	144	90	86	100	0.86	122	304	225	37	248	49.6	138.4	3.74	F+
57	**Sanganna**	45	M	10	56	145	1.46	26.66	72	22	155	90	102	102	1	172	284	233	35	390	78	120	3.428	no f/h
58	**Narayana**	48	M	8	58	152	1.54	20.56	76	20	140	94	87	100	0.87	148	184	289	26	298	59.6	203.4	7.82	M+
59	**Siddaram**	60	M	8	69	168	1.78	24.82	72	18	128	98	90	108	0.83	124	311	220	45	160	32	143	3.17	F+
60	**Ramesh**	57	M	12	65	167	1.74	26.74	74	16	130	90	96	105	0.91	130	210	226	26	258	51.6	148.4	5.7	F+
61	**Gulshanbe**	59	M	10	62	156	1.6	23.93	76	20	130	92	90	100	0.9	103	320	138	20	300	60	58	2.9	no f/h
62	**Laxmi**	54	F	4	72	161	1.74	27.79	76	20	124	94	102	96	1.06	140	339	224	24	249	49.8	150.2	6.258	M+
63	**Gurulingappa**	55	M	8	68	159	1.72	26.98	70	28	124	86	106	80	1.32	155	274	140	21	250	50	69	3.285	F+
64	**Mallinath**	46	M	7	78	165	1.86	28.67	74	18	122	86	90	88	1.02	106	191	186	34	264	52.8	99.2	2.917	M+
65	**Satish.R.**	44	M	10	77	168	1.86	27.3	84	18	136	96	100	94	1.06	194	203	188	38	260	52	98	2.57	F+
66	**Mallamma**	50	F	10	72	168	1.82	25.53	86	16	124	84	91.5	100	0.91	104	199	205	64	260	52	89	1.39	both
67	**Devendrappa**	45	M	4	78	162	1.82	29.77	80	14	130	94	97	93	1.04	193	185	234	38	263	52.6	143.4	3.773	no f/h
68	**Nagraj**	64	M	12	83	164	1.9	30.97	74	22	112	70	109	110	0.99	110	321	251	50	239	47.8	153.2	3.064	M+
69	**Laxmibai**	55	F	3	62	158	1.62	24.89	80	14	120	84	78	95	0.82	80	181	230	34	207	41.4	154.6	4.547	F+
70	**Chiranjeevi**	54	M	6	50	164	1.54	18.65	84	22	142	86	78	88	0.88	137	187	128	38	246	49.2	40.8	1.073	both
71	**Kasthuri**	54	F	10	58	150	1.54	25.77	84	12	110	80	86	80	1.07	155	240	198	30	80	16	152	5.066	F+
72	**Ramappa**	61	M	8	52	173	1.6	17.39	80	16	124	82	80	90		90	315	190	41	247	49.4	99.6	2.429	M+
73	**Vittal**	63	M	15	67	152	1.68	29	82	26	110	70	108	94	0.88	108	291	243	29	228	45.6	168.4	5.806	M+
74	**Vishwanath**	60	M	20	69	146	1.6	32.39	72	20	130	81	124	93	1.33	165	280	150	36	286	57.2	56.8	1.577	F+
75	**Ashok.J**	52	M	8	59	155	1.58	24.58	80	14	138	88	80	95	0.84	174	250	128	39	150	30	59	1.512	F+
76	**Sadashiv**	64	M	12	52	146	1.42	24.41	80	16	128	86	98	96	1.02	122	340	200	44	174	34.8	121.2	2.754	both
77	**Koppa**	63	M	4	74	148	1.64	33.78	76	12	142	86	84	88	0.95	128	311	220	41	228	45.6	133.4	3.253	no f/h
78	**Nagappa**	50	M	7	86	174	2	28.47	82	16	122	83	91	100	0.91	155	243	200	32	204	40.8	127.2	3.97	F+
79	**Nagayya**	65	M	12	72	166	1.8	26.18	76	16	110	81	94	96	0.98	157	274	220	34	177	35.4	150.6	4.43	M+
80	**Rakesh**	48	M	10	71	159	1.74	28.17	84	14	148	92	100	91	1.09	162	239	220	44	224	44.8	131.2	2.98	F+

81	**Rukumuddin**	62	M	5	64	157	1.64	26.01	76	16	140	84	97	105	0.92	138	258	232	34	345	69	129	3.79	both
82	**Surayya**	64	M	9	63	154	1.62	26.58	72	14	122	86	91	91	1	154	320	194	40	204	40.8	113.2	2.83	F+
83	**Sridevi**	53	F	8	73	162	1.76	27.86	72	28	136	90	96	100	0.96	98	300	289	34	348	69.6	185.4	5.45	M+
84	**Neelamma**	60	F	12	68	168	1.76	24.11	84	14	136	86	92	95	0.96	151	280	200	38	214	42.8	119.2	3.136	no f/h
85	**Mallikarjun**	55	M	10	69	160	1.72	26.95	84	20	140	90	90	108	0.83	200	340	258	40	226	45.2	172.8	4.32	M+
86	**Sangawwa**	54	F	4	67	164	1.72	25	86	14	134	100	112	110	1.01	140	196	270	43	191	38.2	188.8	4.39	F+
87	**Rangawwa**	55	F	3	79	166	1.88	28.72	72	16	130	92	98	102	0.96	138	214	443	36	330	66	341	9.47	No f/h
88	**Kammallawwa**	64	F	6	70	168	1.82	24.82	82	16	134	80	94	93	1.01	70	181	216	41	170	34	141	3.44	F+
89	**nanda**	50	F	10	78	169	1.9	27.36	84	14	140	72	98	98	1	124	291	200	30	284	56.8	113.2	3.77	No f/h
90	**Gurappa**	54	M	8	73	167	1.8	26.25	66	14	130	100	104.5	100	1.05	158	196	247	39	324	64.8	143.2	3.67	F+
91	**Sangamma**	51	F	15	73	169	1.84	25.61	76	20	130	78	96.5	94	1.03	100	214	197	34	304	60.8	102.2	3	M+
92	**Mutappa**	58	M	20	68	167	1.78	24.46	78	20	146	90	88	94	0.93	190	291	240	47	254	50.8	142.2	3.02	M+
93	**Kammala**	54	F	8	79	169	1.92	27.71	80	18	146	96	96	102	0.94	136	248	278	32	276	55.2	190.8	5.963	F+
94	**Indra**	40	F	12	63	172	1.74	21.35	74	18	144	90	86	94	0.91	154	290	304	36	254	50.8	217.2	6.03	No f/h
95	**Gangamma**	50	F	4	84	163	1.9	31.69	74	16	144	92	98	94	1.04	120	206	235	32	289	57.8	145.2	4.53	F+
96	**Krishnappa**	58	M	7	71	163	1.76	26.79	78	14	122	94	96.5	88	1.1	142	314	254	28	164	32.8	193.2	6.9	M+
97	**Rathnabai**	48	F	10	62	167	1.7	22.3	76	18	136	84	92	90	1.02	203	274	290	30	276	55.2	204.8	6.82	M+
98	**Dharmanna**	54	M	10	84	162	1.86	32.06	90	26	124	84	102	102	1	134	188	254	34	396	79.2	140.8	4.14	F+
99	**Srinivas.N**	48	M	4	50	159	1.5	19.84	88	16	144	86	91	100	0.91	130	306	367	36	288	57.6	273.4	7.59	both
100	**Boramma**	46	F	12	63	166	1.72	22.9	84	12	154	88	90	18	0.83	146	214	234	32	347	69.4	132.6	4.14	both
101	**Chandrakanth.G**	52	M	8	60	165	1.66	22.05	72	20	150	94	86	94	0.91	218	310	267	24	248	49.6	193.4	8.05	M+
102	**Balaram**	48	M	5	68	147	1.6	31.48	76	12	134	92	98	80	1.2	170	340	224	34	222	44.4	145.6	4.28	F+
103	**S.B.Methri**	44	M	10	52	151	1.46	22.8	84	20	144	94	100	98	1.02	170	284	253	30	258	51.6	171.4	5.71	both
104	**Ramanna**	45	M	5	61	183	1.8	18.26	76	20	110	96	98	104	0.94	130	291	204	30	219	43.8	130.2	4.34	M+
105	**S.A.Goudar**	48	M	9	68	148	1.64	31.05	72	18	136	70	88	94	0.93	120	212	400	32	348	69.6	298.4	9.32	F+
106	**Panchamani**	50	F	4	71	153	1.7	30.34	72	24	154	80	98	98	1	150	200	222	24	239	47.8	150.2	6.25	M+
107	**Basawwa**	44	F	1	68	147	1.6	31.48	84	20	156	94	94	95	0.98	98	197	286	36	221	44.2	205.8	5.71	No f/h
108	**Mahesh**	56	M	5	72	154	1.72	30.37	84	22	140	96	102	102	1	180	312	204	34	226	45.2	124.8	3.67	F+
109	**Meenakshi**	48	F	7	70	156	1.72	29.53	80	30	144	90	96	95	1.01	170	191	230	35	312	62.4	132.6	3.78	F+
110	**Chanappa.L**	58	M	13	50	150	1.44	22.22	78	20	148	96	104	93	1.11	210	305	217	38	234	46.8	132.2	3.47	M+
111	**Parwathi.D**	54	F	4	60	147	1.54	27.77	86	24	140	90	102	92	1.1	140	214	226	31	226	45.2	149.8	4.83	No f/h
112	**Shakuntala**	58	F	8	48	145	1.38	22.85	88	24	140	92	98	90	1	140	230	442	32	286	57.2	352.8	11.02	M+
113	**Kasebgouda**	50	M	12	53	149	1.46	23.87	80	18	146	90	112	108	1.03	144	351	250	27	330	66	157	5.81	F+
114	**Somnath**	54	M	7	61	153	1.58	26.06	82	24	130	96	106	95	1.11	161	289	287	34	260	52	201	5.91	both
115	**mammata**	58	F	7	75	175	1.9	24.5	74	24	148	90	108	98	1.1	190	260	216	36	264	52.8	127.2	3.53	M+
116	**Sainath.S**	40	M	6	72	169	1.82	25.26	74	18	144	92	110	98	1.12	154	268	243	36	169	33.8	173.2	4.81	both
117	**Veeresh**	58	M	12	87	167	1.96	31.29	76	22	126	80	96	100	0.96	144	314	308	34	310	62	212	6.235	No f/h
118	**Sudhir**	55	M	5	68	168	1.78	24.11	80	18	124	80	84	102	0.82	136	320	256	30	302	60.4	165.6	5.52	No f/h
119	**Harish**	49	M	6	74	157	1.74	30.08	86	12	156	90	114	96	1.18	148	244	260	38	262	52.4	169.6	4.463	M+

120	**Rajendra**	59	M	10	70	158	1.72	28.11	76	18	134	84	94	91	1.03	108	208	237	24	251	50.2	162.8	6.78	F+
121	**Suresh**	56	M	12	63	156	1.56	25.92	72	24	130	96	90	110	0.81	90	153	226	25	264	52.8	148.2	5.92	F+
122	**Ravindra**	54	M	8	62	153	1.58	26.49	80	30	120	86	124	100	1.24	200	230	144	46	208	41.6	56.4	1.22	M+
123	**veerendra**	44	M	9	72	161	1.74	27.79	78	16	130	94	96	88	1.09	190	251	224	29	154	30.8	164.2	5.66	No f/h
124	**Vineeth**	54	M	12	67	167	1.76	24.1	80	16	120	84	96	96	1	194	318	188	35	228	45.6	107.4	3.06	both
125	**Gurushanth**	53	M	8	68	159	1.7	26.98	84	16	130	84	104	105	0.99	84	324	232	32	176	35.2	164.8	5.15	M+
126	**Mudukappa**	62	M	5	66	163	1.72	24.9	72	14	140	88	86	100	0.86	170	212	220	40	340	68	112	2.8	M+
127	**Yellapa**	51	M	2	78	165	1.88	28.67	74	14	110	80	92	108	0.85	129	312	164	40	350	70	54	1.35	M+
128	**Arjun**	64	M	7	69	167	1.76	24.82	82	28	142	80	98	102	0.96	156	204	400	42	386	77.2	280.8	6.68	F+
129	**Musafir**	60	M	10	77	168	1.86	27.3	84	14	132	90	94	98	0.95	136	196	232	44	166	33.2	154.8	3.518	both
130	**Narendra**	63	M	7	72	166	1.82	26.18	76	16	126	78	102	94	1.08	96	220	190	36	220	44	110	3.05	No f/h
131	**Manjula**	50	F	2	72	168	1.82	25.53	82	20	120	90	98	102	0.96	160	230	154	44	84	16.8	93.2	2.11	M+
132	**Srinath**	54	M	5	67	171	1.78	22.94	80	18	152	96	102	94	1.08	120	196	420	40	340	68	312	7.8	M+
133	**Asif**	54	M	5	78	162	1.82	29.77	78	18	112	82	88	90	0.97	166	208	152	34	170	34	84	2.47	F+
134	**Sikandar**	50	M	3	62	162	1.64	23.66	74	24	140	80	96	94	1.02	154	300	150	30	230	46	74	2.46	M+
135	**Omkar**	64	M	12	83	166	1.9	30.18	82	20	130	86	98	80	1.23	136	176	240	42	156	31.2	166.8	3.97	No f/h
136	**Sharanu**	50	M	6	70	161	1.74	27.02	86	18	136	94	100	98	1.02	120	216	210	35	170	34	141	4.02	M+
137	**Shaklesh**	51	M	8	61	158	1.62	24.49	88	22	128	80	90	110	0.81	140	198	220	34	200	40	146	4.29	M+
138	**Rachappa**	56	M	6	83	165	1.9	30.51	84	20	140	84	96	92	1.04	150	220	216	30	180	36	150	5	F+

ANNEXURE V

list of Non-Diabetics

Names	Age (yrs)	sex	wt(kg)	Ht(cm)	BMI (kg/m2)	BSA	WC (cm)	HC (cm)	W:H	PR	RR	SBP	DBP	FBS (mg/dl)	PPBS (mg/dl)	CHO (mg/dl)	HDL (mg/dl)	TRI (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	LDL:HDL mg/dl
Shantesh	54	M	77	173	26.55	1.88	102	106	0.96	72	18	132	80	87	120	170	36	140	28	106	2.94
Maheboob	60	M	63	152	27.39	1.64	99	105	0.94	66	18	120	70	81	89	180	44	144	28.8	107.2	2.43
Gangadhar	60	M	60	159	23.8	1.62	94	96	0.97	72	18	110	70	70	85	184	46	138	27.6	110.4	2.4
Geetha	54	F	65	155	27.08	1.7	88	103	0.85	54	24	110	70	82	87	188.5	32.4	200.17	40.034	116.066	3.58
Shrikanth	50	M	54	165	20	1.54	82	89	0.92	80	20	130	80	80	110	180	48	130	26	107	2.22
Jayanthi	62	F	44	149	19.81	1.36	79	84	0.94	88	16	140	90	73	96	254	36.6	118	23.6	193.8	5.295
Jagadish	65	M	52	159	20.8	1.5	83	88	0.94	72	18	130	70	80	89	180	46	160	32	102	2.217
Ranjith	66	M	59	172	20	1.7	87	89	0.97	72	12	90	66	60	80	210	30	220	44	136	4.53
Kailash	43	M	70	164	26.11	1.78	97	97	1	80	24	130	70	80	86	250	38	224	44.8	167.2	4.4
Aravind	54	M	49	167	18.14	1.56	82	92	0.89	81	12	112	80	77	108	220	41	200	40	139	3.39
Sharik	60	M	47	157	19.1	1.44	77	83	0.92	84	24	120	80	65	75	180	43	290	58	79	1.837
Pavan	64	M	70	167	25.9	1.8	81	87	0.93	80	12	110	70	82	109	212	40	206	41.2	130.8	3.27
Ramya	53	F	42	152	18.26	1.34	88	94	0.93	80	24	120	90	82	94	160	43	190	38	79	1.83
Basavaraj	48	M	80	169	28.57	1.9	90	93	0.96	78	18	100	70	85	89	240	48	220	44	148	3.08
Timmapa	52	M	61	152	26.52	1.57	102	100	1.02	72	12	110	70	79	99	181	50	144	28.8	102.2	2.044
Sharada	45	F	59	155	24.58	1.58	90	96	0.93	60	30	110	70	89	102	200	41	101	20.2	138.8	3.38
Venkatappa	55	M	69	168	24.64	1.8	93	97	0.95	64	24	120	80	79	88	196	61	150	30	105	1.712
Ekeashwar	58	M	76	164	28.35	1.84	71	69	1.02	84	12	120	80	77	92	192	42	146	29.2	120.8	2.87
Divakar	50	M	40	152	17.39	1.34	88	89	0.98	80	24	124	88	68	75	151	59	142	28.4	63.6	1.07
Hemaraju	65	M	72	168	25.71	1.76	96	97	0.98	88	24	130	80	88	190	154	46	138	27.6	83.4	1.813
Karthik	52	M	85	175	27.72	2	88	100	0.88	84	12	100	60	73	83	158	50	134	26.8	81.2	1.62
Lokesh	56	M	60	150	26.66	1.53	82	87	0.94	60	18	140	90	80	94	161	55	113	22.6	83.4	1.516
Mubharak	54	M	45	162	17.18	1.44	98	98	1	72	18	100	70	71	95	167	58	126	25.2	83.8	1.45
Puttegouda	53	M	66	165	24.44	1.72	94	112	0.83	78	24	120	80	78	96	165	43	122	24.4	97.6	2.27
Ragavendra	52	M	60	155	25	1.6	100	102	0.98	82	18	100	60	86	90	172	47	118	23.6	101.4	2.16
Srinivas	46	M	69	155	28.75	1.69	77	79	0.97	60	20	120	70	74	80	176	53	114	22.8	100.2	1.89
Putteswami	65	M	40	152	17.39	1.3	104	107	0.97	74	24	110	80	71	78	180	60	110	22	98	1.63
Harsha	56	M	68	150	30.22	1.64	75	81	0.92	74	18	110	70	78	90	185	51	106	21.2	112.8	2.21
Thomas	56	M	41	150	18.22	1.32	85	95	0.89	80	20	110	80	75	85	189	57	102	20.4	111.6	1.96
Narasappa	58	M	65	167	24	1.74	76	86	0.88	64	16	120	80	98	110	194	65	145	29	100	1.53
Suresh	54	M	45	164	16.71	1.46	96	101	0.95	72	12	110	60	84	97	199	52	140	28	119	2.28
Ansar	58	M	70	167	25.92	1.8	71	80	0.88	80	24	130	80	105	112	152	45	135	27	80	1.77
Nagaraj	55	M	42	152	18.26	1.34	98	104	0.97	72	24	100	70	104	109	156	48	129	25.8	82.2	1.71
Vijaykumar	56	M	80	169	28.57	1.92	96	102	0.94	64	24	114	70	89	99	162	44	124	24.8	93.2	2.12

Chandrashekar	54	M	61	152	26.52	1.58	88	92	0.89	72	24	120	70	80	97	166	55	119	23.8	87.2	1.58
Saleem	55	M	59	155	24.58	1.58	98	97	1.01	80	14	140	88	91	84	171	63	113	22.6	85.4	1.36
Devakumar	48	M	69	168	24.64	1.8	108	114	0.94	72	24	136	86	89	140	177	68	108	21.6	87.4	1.28
Raguram	55	M	76	164	28.35	1.84	70	68	1.02	80	24	120	80	78	86	182	48	103	20.6	113.4	2.36
Madhu	51	F	40	152	17.39	1.32	94	99	0.94	72	24	126	80	100	110	197	62	109	21.8	113.2	1.825
lokesh.M	53	M	72	168	25.71	1.82	102	109	0.93	80	24	110	70	103	108	179	67	116	23.2	88.8	1.325
Madhan	47	M	85	175	27.77	2	88	106	0.83	82	18	116	80	92	128	183	64	123	24.6	94.4	1.47
Dinesh	46	M	70	175	22.87	1.84	84	102	0.82	88	18	140	84	78	88	190	65	131	26.5	98.5	1.515
Dharmendra	51	M	65	155	27.08	1.62	93	103	0.9	82	16	134	84	86	94	193	42	144	28.8	122.2	2.91
Doddamani.M	58	M	65	153	27.77	1.62	83	108	0.76	60	16	118	86	85	99	186	31.2	136	27.2	127.6	4.08
Avinash	50	M	50	167	17.98	1.54	89	112	0.79	72	16	130	84	75	90	179	30	127	25.4	123.6	4.12
Shivanand	57	M	65	164	24.25	1.7	78	90	0.87	72	22	128	88	76	100	159	32.4	117	23.4	103.2	3.185
Krishna	49	M	61	160	23.82	1.64	95	107	0.88	72	18	130	88	87	95	153	29	107	21.4	102.6	3.53
Pavan	54	M	55	155	22.91	1.54	79	89	0.88	74	14	140	86	83	102	163	36.6	141	28.2	98.2	2.68
Kailash	56	M	50	165	18.38	1.54	72	84	0.85	68	16	146	70	86	120	169	30	139	27.8	111.2	3.7
Siddapa	52	M	65	155	27.08	1.64	78	94	0.82	74	14	110	80	91	100	183	30	143	28.6	124.4	4.146
Thimmailu	56	M	49	150	21.77	1.42	99	112	0.88	60	22	142	84	78	104	178	40.2	149	29.8	108	2.686
Basavaraj	45	M	42	152	18.18	1.34	82	94	0.87	82	14	140	84	79	86	188	42	133	26.6	119.4	2.84
Praveen	53	M	53	160	20.7	1.54	70	86	0.81	86	22	134	86	85	96	168	40	112	22.4	105.6	2.64
Suresh	56	M	70	150	31.11	1.64	92	100	0.92	74	16	118	84	76	84	160	46	121	30.25	83.75	1.82
Sandeep	54	M	55	173	18.39	1.64	80	100	0.8	74	14	130	84	65	86	170	42	130	26	102	2.42
Somnath.S	52	M	45	150	20	1.36	77	97	0.79	84	28		86	83	92	180	31.2	123	24.6	124.4	3.98
Srinath	50	M	65	164	24.25	1.7	71	91	0.78	86	22	136	74	86	94	155	40	156	31.2	83.8	2.09
Manjula	54	F	65	160	25.39	1.68	72	88	0.81	88	14	122	86	80	99	157	42	119	23.8	91.2	2.17
Vijaykumar	65	M	50	167	17.98	1.55	91	106	0.85	84	22	140	84	82	110	166	46	148	29.6	90.4	1.96
Yellappa.M	52	M	44	162	16.79	1.42	76	92	0.82	80	22	128	80	83	120	164	48	150	30	86	1.79
Mujawar	48	M	45	157	18.29	1.4	69	86	0.8	76	14	140	84	70	80	170	43	142	28.4	98.6	2.29
Arjun	50	M	65	167	23.38	1.7	83	100	0.83	64	16	116	84	89	104	184	48	136	27.2	108.8	2.26
Lakshmi	53	F	53	163	20	1.54	93	110	0.84	68	16	140	86	87	114	195	42	127	25.4	127.6	3.03
Saritha	51	F	43	152	18.61	1.34	85	98	0.86	86	22	134	90	110	116	175	40	146	29.2	105.8	2.64
Poornima	58	F	50	165	18.38	1.4	91	106	0.85	82	16	118	80	86	100	198	40	148	29.6	128.4	3.21
Mallinath	50	M	70	160	27.34	1.74	97	105	0.92	86	18	130	84	109	104	187	42.6	157	31.4	113	2.65
Mallamma.S	57	F	50	150	22.22	1.48	83	97	0.85	86	20	142	86	90	96	184	44	113	22.6	117.4	2.66
Nanda	49	F	62	151	27.19	1.58	85	101	0.84	64	16	140	88	86	104	192	46	102	20.4	125.6	2.73
Ratnabai	54	F	63	159	28	1.64	93	102	0.91	74	16	134	86	84	114	153	44	125	25	84	1.9
Methri	44	M	60	154	25.31	1.58	88	102	0.86	76	14	118	80	84	108	173	48	119	23.8	101.2	2.1
Shrinivas.d	51	M	52	145	24.76	1.44	105	114	0.92	84	20	130	80	94	110	184	56	130	26	102	1.82
Sunil	56	M	70	161	27.02	1.74	69	74	0.93	64	20	140	70	95	90	164	46	130	26	92	2
Virpaksha	45	M	70	163	26.41	1.76	68	73	0.93	78	20	136	84	85	94	152	40	122	24.4	87.6	2.19
Sayed	48	M	46	151	20.17	1.4	85	94	0.9	78	20	120	84	94	144	164	40	178	35.6	88.4	2.21
Nazera	56	M	44	150	19.55	1.36	89	94	0.94	84	12	126	86	83	98	183	42.6	156	31.2	109.2	2.56
Prakash	54	M	57	170	19.72	1.64	90	102	0.88	66	20	110	70	84	90	192	40	119	23.8	128.2	3.2
Sujal	52	M	70	160	27.34	1.74	98	110	0.89	74	20	136	84	69	82	151	42	151	30.2	78.8	1.87
Srinidhi	47	F	60	160	23.43	1.64	88	102	0.86	84	22	122	84	105	114	175	40	172	34.4	100.6	2.515
Tippanna	54	M	86	170	29.75	1.98	78	108	0.72	66	20	140	86	93	96	195	40	128	25.6	129.4	3.23
Yashoda	49	F	65	170	22.49	1.76	84	108	0.77	74	16	128	86	108	110	152	44	138	27.6	80.4	1.82

	52																			
Sarvanand		M	60	150	26.66	1.54	80	96	0.83	84	16	140	84	102	136	176	31	103	20.6	124.4	4.01
Ramu.S	55	M	60	154	25.31	1.58	90	107	0.84	66	14	128	88	70	90	196	40	123	24.6	131.4	3.28
Mahesh.M	48	M	47	162	17.93	1.48	74	90	0.82	78	14	130	88	76	100	173	42	101	20.2	110.8	2.63
HarisChandra	53	M	58	160	22.65	1.6	74	86	0.86	84	14	140	86	84	106	194	29	152	30.4	134.6	4.64
	48																				
Arun		M	58	162	22.13	1.6	73	96	0.76	74	20	146	70	118	96	172	45	110	22	105	2.33
Surekanth	56	M	50	154	21.09	1.46	95	110	0.86	84	16	110	84	126	106	193	46	123	24.6	122.4	2.66
Sanjay	58	M	55	160	21.48	1.56	80	98	0.82	76	12	126	76	90	100	167	44	116	23.2	99.8	2.268
	54																				
Komal		M	60	150	26.66	1.55	74	90	0.82	84	14	130	84	103	108	190	48	109	21.8	120.2	2.5
Mantesh	45	M	50	150	22.22	1.44	88	102	0.86	76	12	116	74	84	124	169	56	190	38	75	1.33
Gouramma.D.	48	F	43	150	19.11	1.34	84	104	0.81	84	20	120	74	118	104	192	46	105	21	125	2.71
	54																				
Srilekha		F	54	164	20.14	1.58	72	97	0.74	78	12	118	74	70	110	188	42	129	25.8	120.2	2.86
Rani	50	F	65	145	30.95	1.56	72	96	0.75	86	20	128	84	86	90	164	46	129	25.8	92.2	2
Ramesh.H	57	M	50	172	16.94	1.58	71	90	0.78	84	14	142	92	82	110	186	48	123	24.6	113.4	2.36
Kashinath	49	M	45	155	18.75	1.4	92	106	0.87	62	12	126	74	84	90	162	43	105	21	98	2.27
	54																				
Hemalatha		F	60	160	23.43	1.62	71	94	0.75	74	26	100	70	118	90	184	48	148	29.6	106.4	2.21
	56																				
Rishikesh		M	60	154	25.31	1.59	70	88	0.8	74	20	124	74	76	112	159	43	117	23.4	92.6	2.15
Badrinath	50	M	50	160	19.53	1.5	84	102	0.82	76	12	126	84	108	100	182	40	128	25.6	116.4	2.91
	45																				
Kamlesh.H		M	46	160	17.96	1.44	94	112	0.84	70	20	120	84	90	112	157	30	138	27.6	99.4	3.31
Ranjith.S	52	M	46	155	19.16	1.48	84	102	0.82	76	12	114	74	90	112	180	29	116	23.2	127.8	4.4
Ravindra.M	56	M	64	164	23.88	1.69	92	106	0.87	68	14	118	94	122	126	155	30	142	28.4	96.6	3.22
	54																				
Parashuram		M	50	160	19.53	1.5	96	108	0.89	88	14	100	76	70	86	178	42	130	26	110	2.61
Kisshan	47	M	40	150	17.77	1.3	84	96	0.87	84	20	114	76	86	106	153	48	150	30	75	1.56
	54																				
Namdev		M	50	168	17.73	1.58	86	102	0.84	76	14	112	74	126	120	176	43	114	22.8	110.2	2.56
Umesh.M	49	M	65	160	25.39	1.7	92	104	0.88	78	14	118	86	90	120	151	42	167	33.4	75.6	1.8
	52																				
Shivanand.R		M	52	152	22.51	1.46	84	102	0.82	88	18	118	80	103	110	130	40	126	25.2	64.8	1.62
	43																				
Bassappa		M	60	150	26.66	1.55	106	112	0.95	80	14	120	86	90	108	143	40	101	20.2	82.8	2.07
Anand.S	65	M	60	155	25	1.58	70	78	0.89	82	14	128	84	122	100	180	44	118	23.6	112.4	2.55
Laxman.H	62	M	62	152	26.83	1.56	72	82	0.88	86	12	114	70	90	106	186	46	130	26	114	2.47
	53																				
Rasid.H		M	50	144	24.15	1.38	84	96	0.88	82	16	130	70	108	116	189	44	130	26	119	2.7
	55																				
Rajshekar		M	65	160	25.39	1.7	90	102	0.88	78	16	112	78	82	110	154	48	123	24.6	81.4	1.69
	46																				
Dileep.m		M	65	165	23.89	1.72	92	106	0.87	88	16	126	84	94	112	138	56	121	24.2	57.8	1.03
	58																				
Sheetal.S		F	42	152	18.18	1.34	100	112	0.89	66	18	114	66	122	94	177	46	156	31.2	99.8	2.16
	53																				
Shubham		M	50	165	18.38	1.52	83	104	0.79	70	14	120	84	76	96	187	52	130	26	109	2.09
Seetha	44	F	72	154	30.37	1.7	73	104	0.7	84	16	114	74	108	140	149	40	103	20.6	88.4	2.21
Vishal.H	54	M	54	158	21.68	1.55	80	106	0.75	84	16	114	84	90	112	177	40	168	33.6	103.4	2.58
Sanju.R	51	M	82	170	28.37	1.92	72	98	0.73	82	20	120	74	94	94	188	42	129	25.8	120.2	2.86
Virat	46	M	63	165	23.16	1.7	92	112	0.82	82	16	114	86	84	84	187	44	120	24	119	2.7
Wasim	47	M	42	160	16.4	1.38	76	96	0.79	68	14	128	86	90	120	174	30	105	21	123	4.1
Naser	50	M	54	160	21.09	1.54	76	90	0.84	76	14	110	80	108	100	149	29	124	24.8	95.2	3.28

Appasaheb	61	M	62	155	25.83	1.6	75	112	0.67	78	12	110	84	82	112	167	45	168	33.6	88.4	1.96
Sachin	58	M	54	152	23.37	1.48	90	112	0.8	86	12	120	72	84	124	156	46	117	23.4	86.6	1.88
Subhash	50	M	47	150	20.88	1.4	84	102	0.82	66	18	140	74	118	140	130	44	116	23.2	62.8	1.43
Mounesh.H	57	M	52	162	19.84	1.56	72	96	0.75	80	14	130	76	102	120	178	48	124	24.8	105.2	2.19
Shardhamma	47	F	64	144	30.91	1.56	86	104	0.83	80	14	116	84	70	136	167	56	126	25.2	85.8	1.53
Anil.Rathod	54	M	52	170	17.99	1.58	86	110	0.78	86	12	134	74	76	100	188	46	132	26.4	115.6	2.51
Amog	49	M	46	154	19.4	1.4	74	102	0.73	68	18	118	86	94	106	130	42	101	20.2	67.8	1.61
Maruthi.H	53	M	64	164	23.88	1.7	74	100	0.74	76	16	128	84	82	100	143	46	154	30.8	66.2	1.43
Pralad	54	M	58	152	25.1	1.54	72	96	0.75	86	18	112	82	94	108	178	48	118	23.6	106.4	2.21
prajwal	44	M	62	152	26.83	1.56	94	110	0.85	68	12	130	72	122	124	167	43	103	20.6	103.4	2.4
Parvathi.M	42	F	54	162	20.61	1.54	74	100	0.74	80	14	128	80	87	136	143	48	130	26	69	1.43
Sunandha	56	F	44	154	18.56	1.38	74	94	0.79	86	20	122	86	81	100	180	46	121	24.2	109.8	2.38
Rekha	45	F	60	160	23.43	1.62	82	104	0.79	76	18	136	88	86	106	154	42	116	23.2	88.8	2.11
Pasha	52	F	48	158	19.27	1.46	96	110	0.87	80	14	110	86	100	120	138	46	125	25	67	1.45
Gangabai	47	F	44	154	18.56	1.38	84	104	0.81	86	16	118	84	103	112	190	56	119	23.8	110.2	1.96
Yamunna	49	F	52	168	18.43	1.56	94	110	0.85	78	16	124	80	90	144	169	51	154	30.8	87.2	1.7
Shailaja	52	F	48	154	20.25	1.42	92	108	0.85	86	14	104	90	110	134	103	48	117	23.4	31.6	0.65
Saraswathi	54	F	57	152	24.67	1.54	86	100	0.86	84	16	122	84	109	140	122	41	105	21	60	1.46
Sruthi	50	F	52	158	20.88	1.55	84	102	0.82	64	16	116	82	94	100	174	39	128	25.6	109.4	2.8
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