“STUDY OF LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS’’

By

Dr. NAGABUSHAN HESARUR

Dissertation submitted to BLDE University, Bijapur

In partial fulfilment of the requirements for the degree of

MD

IN

GENERAL MEDICINE

Under the guidance of

Dr. R.C.BIDRI M.D

Professor

DEPARTMENT OF MEDICINE,

BLDEU’S Shri B.M. Patil Medical College,

Bijapur.

2014

B. L. D. E. U'S

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &

RESEARCH CENTRE, BIJAPUR.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation/thesis entitled **" STUDY OF LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS''** is a bonafide and genuine research work carried out by me under the guidance of **DR.R.C.BIDRI,M.D**, Professor, Department of Medicine, Shri B.M. Patil Medical College, Bijapur

Date:

Place: Bijapur **Dr. NAGABUSHAN HESARUR**

B. L. D. E. U'S

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &

RESEARCH CENTRE, BIJAPUR.

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"STUDY OF LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS''** is a bonafide and genuine research work carried out by **Dr. NAGABUSHAN HESARUR** in partial fulfilment of the requirement for the degree of MD in General medicine.

Date:

Place: Bijapur

Dr. R.C.BIDRI M.D.
Professor
Department of Medicine,
Shri B.M. Patil Medical College,
Bijapur.

B. L. D. E. U'S

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &

RESEARCH CENTRE, BIJAPUR.

ENDORSEMENT BY THE HOD, PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"STUDY OF LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS''** is a bonafide research work done by **Dr**. **NAGABUSHAN HESARUR** under the guidance of, **Dr. R.C.BIDRI** **M.D**, Professor, Department of Medicine, Shri BM Patil Medical College, Bijapur.

Seal & Signature of
HOD of Medicine
Dr. M.S. MULIMANI **MD**
BLDEU's Shri B.M. Patil
Medical College, Hospital &
Research Centre, Bijapur

Date:
Place: Bijapur

Seal and signature of
the principal
DR.M.S.BIRADAR **MD**
BLDEU's Shri B.M. Patil
Medical College, Hospital &
Research Centre, Bijapur.

Date:
Place: Bijapur

B. L. D. E. U'S

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &

RESEARCH CENTRE, BIJAPUR.

COPYRIGHT DECLARATION BY THE CANDIDATE

I hereby declare that the BLDE University, Karnataka shall have the rights to preserve, use and disseminate this dissertation / thesis in print or electronic format for academic / research purpose.

Date:

Place: Bijapur **Dr. NAGABUSHAN HESARUR**

ACKNOWLEDGEMENT

I wish to express my deep sense of gratitude and regards to my guide **Dr. R.C.Bidri** MD, Professor of Medicine, for his able guidance and valuable suggestions, constant supervision and encouragement which he rendered in pursuit of my postgraduate studies and during the preparation of this dissertation.

I would also like to express my sincere thanks to our principal and Professor **Dr. M.S. Biradar** MD, for his kind support.

I am extremely grateful to Professors of Medicine, **Dr. S.N. Bentoor** MD, **Dr.S.S.Devarmani** MD**, Dr. L.S.Patil** MD**, Dr. S.R. Badiger** MD & **Dr.M.S. Mulimani** MD for their valuable advice and guidance in completing this work.

I am also thankful for the support extended **by Dr. G.S.Mahishale** MD, Associate Professor of Medicine**. Dr. S.B.Nadagouda** MD, Associate Professor of Medicine.

I would also like to thank my junior **Dr. Saurabh Sadekar** for his kind help in completing this work.

My sincere thanks to all the staff of the Department of Biochemistry, Shri B.M. Patil Medical College Hospital & Research Centre, Bijapur who helped me in the laboratory investigation work.

I thank **Mr. Shankar Javali**, statistician who kindly obliged & helped me with the statistical work.

I thank all my friends and colleagues for their kind cooperation.

I would also like to thank my parents **Dr. Mahalingappa Basalingappa** and **Smt. Umadevi**, without their constant encouragement and moral support, my studies would have been a distant dream.

I would like to thank the Almighty god who gave me the energy, skill and the enthusiasm to complete this as well as the other tasks in my life & also for continuing to shower his blessings upon me.

Last but not the least; I extend my sincere thanks to all the patients who willingly cooperated in this study.

Date: **Dr. NAGABUSHAN HESARUR**

Place: Bijapur

LIST OF ABBREVIATIONS

TC	-	Total cholesterol
VLDL	-	Very low density lipoprotein
LDL	-	Low density lipoprotein
TG	-	Triglycerides
HDL	-	High density lipoprotein
IDL	–	Intermediate density lipoprotein
Lp(a)	–	Lipoprotein A
ADA	-	American DiabetesAssociation
Apo(B)	-	Apoprotein B
Apo(E)	-	Apoprotein E
HbA1C	-	Glycosylated haemoglobin
CAD	–	Coronary artery disease
MODY	–	Maturity onset diabetes mellitus of young
NIDDM	–	Non Insulin dependent diabetes mellitus
BMI	-	Body mass index
IGT	-	Impaired glucose tolerance
LPL	-	Lipoprotein lipase
LDL-R	-	Low density lipoprotein receptor
LCAT	-	Lecithin cholesterol acyl transferase
FFA	-	Free fatty acid
HSL	-	Hormone sensitive lipase
PVD	-	Peripheral vascular disease
AAP	-	Amino anti pyrine
CBC	-	Complete blood count
CTEP	-	Cholesteryl ester transfer protein

ABSTRACT

BACKGROUND

Diabetes mellitus is a major public health problem worldwide. Around the world 200 million people have diabetes and it is predicted to increase to 300 million by 2020. Type 2 diabetes mellitus has shown an alarming upward trend among the Indian population and it is labeled as Diabetes Capital amongst the developing nations. In type 2 diabetes mellitus, lipid abnormalities are almost the rule and is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities that are all recognized as major risk factors for coronary artery disease and other macro vascular complications. Levels of all lipid fractions except HDL are abnormally elevated in type 2 diabetes mellitus when compared to non-diabetics.

OBJECTIVES:

To study the lipid profile in type 2 diabetic individuals in comparison with non-diabetic individuals.

METHODOLOGY:

The material for the present study will be collected from patients who attend the Outpatient department and Inpatient department in BLDEU'S Shri. B.M.Patil Medical College Hospital and Research Centre, Bijapur over a period 2 years from January 2013 to June 2014. The sample size was 250 of which 125 were type 2 diabetes mellitus patients who were studied as cases and 125 non diabetics were taken as controls and there lipid profile were estimated and the results obtained were statistically computed.

RESULTS:

In the present study the results obtained are in cases (type 2 diabetes mellitus) values are as follows – LDL 117.99 ± 49.28, TC 196.77 ± 73.6, TG 186.05 ± 128.32, HDL 38.72 ± 12.5,

VLDL 34.06 ± 19.65.These values are much higher as compared to controls.

CONCLUSION

In the Present study it is observed that in type 2 diabetes mellitus patient's lipid profile will be significantly altered as compared to non-diabetic patients and hence morbidity and mortality in this group of patients is more as compared to non-diabetic patients.

KEYWORDS:

HDL; LDL; TC; TG; VLDL

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

INTRODUCTION

Diabetes mellitus is the most prevalent metabolic disease in the world. Diabetes mellitus has been known since antiquity. In India, diabetes is not an epidemic anymore but has turned into a pandemic. The International Diabetes Federation has declared diabetes mellitus as a 'Tsunami'. According to the International journal of Diabetes, India has been labeled as 'The Diabetes Capital' amongst the developing countries.

Around the world about 200 million people have diabetes and is predicted to increase to 300 million by 2020[1]. The incidence of diabetes mellitus has shown an alarming upward trend among the Indian population. The International Diabetes Federation estimated that the number of diabetic patients in India will be more than doubled from 19 million in 1995 to 40.9 million in 2007. It is projected to increase to 80 million by 2030.

The largest increase of the diabetic population occurs in the most economically productive age group. Currently up to 11% of India's urban population and 3% of rural population above the age of 15 has diabetes mellitus. The most prevalent is type 2 diabetes mellitus, which constitutes 95 percent of diabetic population in the country.

In type 2 diabetes mellitus, lipid abnormalities are almost the rule and is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities that are all recognized as major risk factors for coronary artery disease and other macro vascular complications. These lipid abnormalities are not only quantitative but also qualitative abnormalities of the lipoproteins which are potentially atherogenic.

Typical findings are elevation of Total cholesterol, Triglycerides, VLDL cholesterol, LDL Cholesterol and Apolipoprotein (Apo B), with lowering of HDL cholesterol.

In type 2 diabetes mellitus, due to obesity and insulin resistance, lipolysis and free fatty acid flux from adipocytes are increased, leading to increased VLDL and triglyceride synthesis in hepatocytes. This is responsible for the dyslipidemia found in type 2 diabetes Mellitus.

This dyslipidemia may be present at the time of diagnosis of type 2 diabetes mellitus and is a part of metabolic syndrome. Insulin resistance and obesity combine to cause dyslipidemia and are likely to contribute to the risk of coronary artery disease and other macro vascular complications and the determination of serum lipid levels in people with type 2 diabetes mellitus is now considered as a standard of the diabetes care.

Realizing the most of diabetics have a high probability of developing coronary artery disease and cerebrovascular diseases due to abnormalities in lipid metabolism. This study intends to show the differences in lipid profile in type 2 diabetic individuals as compared to non-diabetic individuals.

AIMS AND OBJECTIVES

To study the lipid profile in type 2 diabetic individuals in comparison with non-diabetic individuals.

REVIEW OF LITERATURE

Diabetes is perhaps as old as mankind. The earliest mention of diabetes like illness by polyuria can be traced to Egyptian papyrus dating back to around 1550BC.

Diabetes was recognized as a disease entity in ancient Indian Ayurveda[2], Charaka samhitha authored in the pre Buddhist period (600BC) contains graphic description of the disorder from a physician point of view. By 400BC, Sushrutha, an astute clinician and a deft surgeon, supplemented the earlier information and presented a comprehensive picture of diabetes.

The disorder was named as Madhumeha (Rain of Honey) because of the sweet taste of urine attracting ants and insects.

Aretaeus, 1st century AD according to some historians of Cappedocia is generally believed to have coined the term 'Diabetes', conveying the same meaning. Although Demetrios (2nd century AD) has been alluded to have first linked the excessive water drinking and polyuria of this disorder to "Flowing through a siphon".

Progress in the Diabetes front was nominal during medieval period (600-1500AD). Chhuan of China (7th century AD) and Arabian Physician Aricenna (960-1037AD) had described the features of diabetes appropriately.

During the period of Industrial revolution and renaissance (1780-1850AD), the observation of Dobson, the hitherto held idea of attributing diabetes to defects of kidneys and the urinary system was no longer tenable.

Thomas Cowley in 1788 observed shrunken pancreas riddled with stones in a patient of diabetes at an autopsy. By 1798, John Rollo, Surgeon General in British Artillery, based on findings of Dobson, assumed hyperglycemia to be a feature of diabetes.

In 1815, Chevrenil reported that the sugar in blood was chemically akin to grape sugar (Glucose). During the next century of the modern era (1850-1950) there has been exponential growth of experimental medicine as well as progress in knowledge about diabetes.

Medicine first recognized the existence of abnormal fatty content of the circulating blood through the milky appearance observed during the days when bloodletting was widely practiced. The term lipemia was formulated by Babington in the 18th century, when he showed that fats were responsible for giving this milky appearance to the serum.

The presence of lactescent serum with diabetes was first noted by Mariet in 1799 and in 1958 by Thannhauser.S.J.

Albrink et al [3] (1963) studied 139 diabetics over a period of 30 years with regard to the serum lipid concentration and the vascular complications of diabetes mellitus and found a trend towards increasing serum triglyceride concentrations and increasing incidence of atherosclerosis in coronary and other arteries.

Bagdade et.al [4] (1967) studied five patients with chronic symptomatic diabetes and minimal ketoacidosis who had marked hyperlipidemia and concluded that diabetic lipemia can be considered to be a reversible form of dietary fat induced lipemia secondary to chronic insulin deficiency.

Chance et.al [5] (1969) studied serum lipids and lipoproteins in 135 diabetic children prior to treatment and found elevated serum total lipids in 64% of the patients and elevated cholesterol in 43%. Abnormal lipoprotein patterns were found in 77%, the commonest anomaly being increase in pre P-lipoprotein.

Sharma D et.al [6] (1970) studied serum lipid profile in type 2I diabetic patients below 40 years of age and found significant elevations in the level of serum

cholesterol, phospholipids, esterified fatty acids and triglyceride as compared to a control group.

Nikkila EA [7] and Hormila P (1978), concluded that the average serum lipid and lipoprotein pattern of insulin treated chronic diabetic patients was not more atherogenic than non-diabetic subjects of similar age and sex. On the contrary the increase in HDL-C levels which they found should make them less liable to develop coronary heart disease. Thus they felt that the increased incidence of cardiovascular disease in type 2 diabetes must be accounted for by some other factors.

V.J. Retnam et al [8] (1983) reported hyperlipoproteinemia in a study of 152 adult diabetics on treatment. They found that 20 out of 70 controlled patients and 48 of 82 uncontrolled patients had hyperlipoproteinemia.

Bijlani et al [9] (1984) 40 found HDL-C to be significantly lower in obese diabetics when compared to normal weight diabetics.

Hokanson JE et al [10] (1996) stated that plasma triglyceride is an independent risk factor for the development of cardiovascular disease.

In a study of the significance of blood lipid alterations in diabetes mellitus**,** Mazzone.T et al [11] (2000) measured plasma triglyceride and cholesterol levels in a large series of diabetic and non-diabetic subjects of all ages. Their results showed that plasma triglycerides increase with age in diabetics but not in non-diabetics, while cholesterol levels increase with age in both groups.

Chaturvedi et al [12] (2001) found elevation in triglyceride rich VLDL to be a common abnormality.

A study conducted by Joshua A Becham [13], Mark A Creager and P Libby in 2002, showed that there is increased incidence of lipid abnormalities in diabetes mellitus, which was the risk factor for cardiovascular morbidity and mortality.

A study done by Allan D. Sniderman et al [14] showed that 40% of their subjects had elevated Apo-B and therefore elevated LDL particle matter.

A large population based survey from the Swedish National Diabetes register by Mats Eriksson et al [15] showed that out of 75,048 type 2 diabetics, pronounced hypertriglyceridemia was seen in 3.4% of the patients. Total cholesterol, LDL-Cholesterol, HDL-cholesterol and non-HDL cholesterol were generally higher, and LDL-Cholesterol/HDL-cholesterol and Non HDL cholesterol/HDL cholesterol were lower in women.

A study conducted by Kayode et al [16] in 2010 showed that 57 diabetic patients out of 113 diabetic study subjects were having at least one lipid value or the other outside the clinical target, giving it a prevalence of 50.4%. The most frequent lipid combination was total cholesterol plus HDL-cholesterol. The mean total cholesterol and LDL-cholesterol were higher among female subjects.

A study of correlation between lipid profile and waist to hip ratio in patients with diabetes mellitus by Hardev singh sandhu [17], Shyamal Koley and Karanjit singh sandhu showed that there is a strong correlation with waist to hip ratio and lipid profiles in patients with diabetes mellitus.

A study conducted by Samatha P [18], Venkateshwarlu M and Siva prabodh V in Adilabad, Andhrapradesh showed that the mean total cholesterol, triacylglycerols, LDL-Cholesterol and the fasting blood sugar levels were highly significant in the diabetics as compared to those in the controls. Thus indicating that diabetic patients were more prone for dyslipidemia, which could cause cardiovascular disorders.

Study conducted by Kursheed et al [19] in 2011, depicted the picture of hyperlipidemia as the commonest complication of diabetes mellitus and it predisposes them to premature atherosclerosis and macro vascular complications. In this study,

males had higher levels of LDL-cholesterol as compared to females. 19% males had LDL-cholesterol > 160mg/dl.

A study done by Yadav et al [20] in 2012 showed that there was a high prevalence of dyslipidemia in diabetics.

In the study done by Fatima Hussain Kanani [21] and Junaid Mehmood Alam s in Karachi, showed that raised apolipoprotein B was the most frequent lipid disorder in type 2 diabetics, occurring in 56.7% of the studied patients. This was followed by high serum triglycerides levels in 55.8% and low HDL cholesterol levels in 55 % of the patients.

Howard et al [22] in the strong heart study, showed that dyslipidemia in diabetes is a major risk factor for cardiovascular disease. In this study, greater adverse differences in those with diabetes versus those without diabetes were observed in women than in men for HDL-cholesterol, apolipoprotein (Apo) B, apo A1, fibrinogen and LDL size.

A study conducted by M.Nakhjavani [23], A.R.Esteghamati, F.Esfahanian and A.R.Heshmat in Iran showed that there is high prevalence of dyslipidemia in type 2 diabetic patients. The most prevalent was hypertriglyceridemia (63.4%) and the least frequent was elevated total cholesterol (35%) and diabetic women has more atherogenic lipid profile as compared to diabetic men.

In the Framingham heart study [24], the prevalence of high plasma triglyceride levels in individuals with diabetes mellitus (19% in men and 17% in women) was significantly higher than those without diabetes mellitus (9% in men and 8 % in women).

In the study conducted by Gilani et al [25] in Abbotabad in 2010, showed that out of 150 diabetic patients enrolled for the study, 82.7% (124/150) had increased

serum triglyceride. Raised LDL was found in 54.7% (82/150) and 64.6% had low HDL levels.

A study done by Amin-ul-haq [26], Jamil ur Rehman, Rashid Mahmood, Abdul Jalal Safi, Zahoor Ahmed and Saatea Arif showed that the levels of serum triglycerides, total cholesterol, LDL-C and VLDL-C was higher in diabetic patients as compared to the control group, while HDL-C showed the opposite pattern.

The findings in this study done by Dr.Ratna Manjula Songa [27], Siddhartha K and Dr.Sudhakar K in Vijayawada, Andhra Pradesh showed that the obese type 2 diabetes mellitus patients had significantly higher serum triglycerides, LDL-C levels and serum VLDL-C levels; with significant lower HDL-C levels when compared to obese non diabetic cases.

In a study done by Khursheed Muhammad Uttra et al [28], males had higher levels of LDL-C as compared to females. 19% diabetic males had LDL-C >160mg/dl and 14% diabetics had HDL-C <35mg/dl. Low HDL-C was a common associated finding with raised serum TG, serum cholesterol and LDL-C.

A study conducted by Ramu Kandula [29] and Vinayak E.Shegokar in Hyderabad showed that there is a significant increase in serum Total Cholesterol, Triglycerides and LDL-C along with a significant decrease in HDL-C among diabetics as compared to non-diabetics.

Udawat et al [30] reported dyslipidemia in 89% of type 2 diabetic patients. LDL hyper lipoproteinemia more than 100mg/dl in 73% and HDL dyslipidemia less than 35 mg/dl in the study group.

Bhu et al [31] observed higher level of cholesterol and LDL level in diabetic individuals.

Hardas et al [32] found only higher triglycerides levels in diabetics as compared to non-diabetics.

A study done by Daniel Nii Aryee Tagoe [33] and Philip Amo-Kodieh in Ghana showed type 2 diabetes mellitus patients in this study had elevated levels of TG, reduced levels of HDL with either normal or elevated levels of LDL.

Lorenzo Gordon [34], Dalip Ragoobirsingh, Errol Y St A Morrison, Eric Choo-Kang, Donovan McGrowder, E Martorell showed that the mean TC, TG, VLDL-C, LDL-C and HDL concentrations, and TG/HDL and LDL/HDL ratios were higher in type 2 diabetics and hypertensive type 2 diabetic patients, compared with non-diabetic and hypertensive non-diabetic control subjects, respectively.

An observation by Tahmeen Jameel [35], Raisa Faheem and Syed Mahmood Ahmed in Hyderabad indicated that there is derangement of lipoprotein metabolism in patients with NIDDM. There was increase in serum triglycerides, cholesterol, LDL and VLDL levels. HDL cholesterol levels are lowered. The most characteristic lipid abnormality in diabetics is hypertriglyceridaemia which is a risk factor for major vascular events in diabetics. It has been conclusively shown that reducing LDL cholesterol is beneficial in reducing CAD risk, with lowering of LDL cholesterol being a primary target in the prevention of CAD.

An observational study by Mumtaz Ali Shaikh [36], Santosh Kumar, Rafi Ahmed Ghouri showed that majority of type 2 diabetes mellitus had uncontrolled blood sugar levels and most had their lipid levels deranged.

The PROCAM [37] study showed that hypertriglyceridemia was present in 39% of diabetics as compared to non-diabetics.

Sapna smith et al [38] reported that LDL-C was higher in diabetic group when compared to control and it was significantly elevated in diabetic males when compared to diabetic female.

Survyavanshi et al [39] showed that HDL-C was significantly lower in diabetics when compared to non-diabetics.

Gambhir et al [40] found that low HDL-C were independent risk factor for premature coronary artery disease.

A study done by Abdul Hamid Zargar [41] , Farooq Ahmad Wandroo , Mool Brahman Wadhwa, Bashir Ahmad Laway, Shariq Rashid Masoodi , Nissar Ahmad Shah showed that obese diabetics when compared to obese control subjects showed statistically significant increase in the levels of serum total lipids, serum total cholesterol, serum triglycerides, serum LDL-cholesterol and serum phospholipids . Serum HDL – cholesterol levels did not differ significantly in the two groups.

A study done by Mranali Sharma [42], Menu Rai, Pushpendra Singh demonstrated that there was significant elevation of serum total cholesterol, triglycerides, LDL-cholesterol, VLDL-cholesterol, except HDL-cholesterol which is decreased significantly, in all diabetic patients when compared with those of control groups. Female patients had higher level, of total cholesterol triglycerides, LDL-cholesterol, VLDL-cholesterol and low HDL-cholesterol, as compared to age matched male patients.

In the United Kingdom Prospective Diabetes Study (UKPDS) [43], an altered plasma lipid profile was observed in type 2 diabetics. Women with type 2 diabetes had markedly higher levels of total cholesterol than women who were non diabetic.

DIABETES MELLITUS

Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia.

It is defined as a group of metabolic diseases that are characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both.

Diabetes may present with symptoms suggestive of hyperglycemia such as polyuria, polydipsia, polyphagia, and weight loss or may present with one of the acute or chronic complications or may be detected incidentally in hospitalized patients.

Diabetes may be seen intermittently only as in pregnancy.

CLASSIFICATION [44]

Diabetes mellitus is the most prevalent metabolic and non-communicable disorder in the world. There has been a lack of uniformity in manifestation, complications, management and genetics. This had led to epidemiological agencies to put forth varieties of classifications for this syndrome of chronic hyperglycemia.

I. Type 1 diabetes (beta cell destruction, usually leading to absolute insulin deficiency)
   a) Immune mediated
   b) Idiopathic

II. Type 2 diabetes (may range predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)

III. Other specific types of diabetes

A. Genetic defects of beta cell function characterized by mutations in :

1. Hepatocyte nuclear transcription factor (HNF)4alpha (MODY 1)
2. Glucokinase (MODY 2)
3. HNF-1 alpha (MODY 3)
4. Insulin promoter factor-1(IPF-1: MODY 4)
5. HNF-1beta (MODY 5)
6. Neuro D1 (MODY 6)
7. Mitochondrial DNA
8. Subunits of ATP sensitive potassium channel
9. Proinsulin or insulin

B. Genetic defects in Insulin action

1. Type A insulin resistance
2. Leprechaunism
3. Rabson-Mendenall syndrome
4. Lipodystrophy syndromes

C. Diseases of the exocrine pancreas- pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, haemochromatosis, fibrocalculous pancreatopathy mutations in carboxyl ester lipase.

D. Endocrinopathies – acromegaly, cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma.

E. Drug or chemical induced- glucocorticoids, pentamidine, nicotinic acid, Beta adrenergic agonists, thiazides, alpha interferons, protease inhibitors, anti psychotics (Atypicals and others)

F. Infections- congenital rubella, cytomegalovirus, coxsackie virus

G. Uncommon forms of immune mediated diabetes – "Stiff person" syndrome, anti-insulin receptor antibodies.

H. Other genetic syndromes sometimes associated with diabetes- Wolfram's syndrome, Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Friedreich's ataxia, Huntington's chorea, Laurence –Moon Biedl syndrome, mytonic dystrophy, Prader Willi syndrome

IV. Gestational diabetes Mellitus (GDM)

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS:

The International Expert Committee with members appointed by the American Diabetes Association (ADA), the European Association for the study of Diabetes and the International Diabetes Federation has issued diagnostic criteria for Diabetes mellitus based on the following premises:

1) The Fasting Plasma Glucose, the response to an oral glucose challenge (OGTT-oral glucose tolerance test) and HbA1c differ among individuals and
2) Diabetes mellitus is defined as the level of glycemia at which diabetes specific complications occur rather than on deviations from a population based mean.

Criteria for the diagnosis of Diabetes Mellitus[44]:

1. A random blood glucose concentration > 200mg/dl (11.1 mmol/l) accompanied by classic symptoms of diabetes mellitus (polyuria, polydipsia, weight loss). **(**Random is defined as without regard to time since last meal.)

OR

2. Fasting plasma glucose (FPG) > 126 mg/dl (7.0 mmol/l) (Fasting is defined as no caloric intake for at least 8 hours).

OR

3. HbA1C > 6.5%

OR

4. Two-hour plasma glucose > 200mg/dl (11.1mmol/l) during an oral glucose tolerance test. (The test should be performed using a glucose load containing the equivalent of 75gm anhydrous glucose dissolved in water, not recommended in routine use.)

EPIDEMIOLOGY

The worldwide prevalence of diabetes mellitus has risen dramatically over the past two decades, from an estimated 30 million cases in 1985 to 285 million in 2010. Based on current trend, the International Diabetes Federation projects that 438 million individuals will have diabetes by the year 2030 [45].

Although the prevalence of both type 1 and type 2 diabetes mellitus is increasing worldwide, the prevalence of type 2 diabetes mellitus is rising much more rapidly because of increasing obesity, reduced activity levels as countries become more industrialized and the aging of population.

Type 2 diabetes is the predominant form of diabetes worldwide, accounting for 90% of cases globally. An epidemic of Type 2 diabetes mellitus is underway in both developed and developing countries, although brunt of the disorder is felt disproportionately in Non-European populations. This is primarily due to rapid transition occurring in these countries as a consequence of urbanization, industrialization and globalization.

The epidemiology of diabetes in India, the second largest country with a population of over 1 billion is of prime importance as the prevalence of diabetes is growing rapidly not only in urban area but also in rural areas.

In 2000, India (31.7 million) topped the world with the highest number of people with diabetes mellitus followed by China (20.8 million) with the United States (17.7 million) in second and third place respectively.

According to the recent WHO report, India has the highest number of people with diabetes in the world, with an estimated 32 million in 2000 which is set to increase to a staggering 80 million by 2030.

There are however patterns of diabetes incidence that are related to the geographical distribution of diabetes in India. Rough estimates show that the prevalence of diabetes in rural populations is one-quarter that of urban population for India and other Indian sub-continent countries such as Bangladesh, Nepal, Bhutan, and Sri Lanka [46,47].

Preliminary results from a large community study conducted by the Indian Council of Medical research (ICMR) revealed that a lower proportion of the population is affected in states of Northern India (Chandigarh 0.12 million) Jharkhand (0.96 million) as compared to Maharashtra (9.2 million) and Tamil Nadu (4.8 million).

The National Urban Survey conducted across the metropolitan cities of India reported similar trend: 11.7 % in Kolkata (Eastern India), 6.1 % per cent in Kashmir Valley (Northern India), 11.6 % in New Delhi (Northern India), and 9.3 % per cent in West India (Mumbai) compared with (13.5 % per cent in Chennai (South India), 16.6 % per cent in Hyderabad (south India), and 12.4 % per cent Bangalore (South India).

Obesity is one of the major risk factors for diabetes, yet there has been little research focusing on this risk factor across India [48]. Despite having lower overweight and obesity rates, India has a higher prevalence of diabetes compared to western countries suggesting that diabetes may occur at a much lower body mass index (BMI) in Indians compared with Europeans [49].

Therefore, relatively lean Indian adults with a lower BMI may be at equal risk as those who are obese. Furthermore, Indians are genetically predisposed to the development of coronary artery disease due to dyslipidaemia and low levels of high density lipoproteins [50]. These determinants make Indians more prone to development of the complications of diabetes at an early age (20-40 years) compared with

Caucasians (>50 years) and indicate that diabetes must be carefully screened and monitored regardless of patient age within India.

An upsurge in number of early-onset diabetes cases is also responsible for the development of various diabetic complications due to longer disease duration, however data on the prevalence on diabetic complications across the whole of India is scarce [51].

Both macro vascular and micro vascular complications cause significant morbidity and mortality among diabetic subjects [52]. The Chennai Urban Population Study (CUPS) [53] and CURES [54] provided valuable data from India on the complications related to diabetes.

The prevalence of coronary artery disease was 21.4 % among diabetic subjects compared to 9.1 per cent in subjects with normal glucose tolerance. The prevalence of CAD in IGT subjects were 14.9% in the same study. It was also seen that the diabetic subjects had increased subclinical atherosclerosis as measured by intimal medial thickness (IMT) at every age point compared to subjects with normal glucose tolerance.

A recent study showed that carotid intima medial thickness increased with worsening grades of glucose tolerance as well as with increase in the number of components of metabolic syndrome [55].

The prevalence of peripheral vascular disease (PVD) was 6.3 % among diabetic subjects compared to 2.7 % in non-diabetic subjects [56], and these figures are lower than the prevalence reported in western populations. This is probably due to lower age at onset for diagnosis of type 2 diabetes in India. It is well known that PVD is more common in older individuals.

The CURES Eye study [57] is the largest population based data on the prevalence of diabetic retinopathy done in India. This study showed that the overall prevalence was 17.6 per cent, which was lower when compared to the reports from the West. A recent population based study reported that the prevalence of overt nephropathy was 2.2 per cent in Indians while microalbuminuria was present in 26.9 %.Glycated haemoglobin, duration of diabetes and systolic blood pressure were independently associated with diabetic nephropathy [58].

Overall, Asian Indians appear to have a greater predilection for cardiovascular complications whereas the prevalence of microvascular complications appears to be lower than in Europeans.

Diabetes has become one of the world's most important public health problem and is a major cause of mortality. In the United States, diabetes was listed as the seventh leading cause of death in 2007. A recent estimate suggested that diabetes was the fifth leading cause of death worldwide and was responsible for almost 4 million deaths in 2010.

BRIEF ACCOUNT OF THE LIPID CHEMISTRY

The lipids are a heterogenous group of compounds including fats, oils, steroids, waxes and related compounds that are related more by their physical than chemical properties.

The lipids have following functions.

1. They are important dietary constituents not only because of their high energy value, but also because of the fat soluble vitamins and essential fatty acids contained in the fat of natural foods.
2. Fat is stored in adipose tissue, where it also serves as a thermal insulator in the subcutaneous tissues and around certain organs.
3. Non polar lipids act as electrical insulators, allowing rapid propagation of depolarization waves along myelinated nerves.
4. Combination of lipid and protein (lipoproteins) serve as the means of transporting lipids in the blood.

The lipids are classified as given below [59].

A) Simple lipids (esters of fatty acid with various alcohols)

• Fats: esters of fatty acids with glycerol. Oils are fats in liquid state

• Waxes: esters of fatty acids with higher molecular weight monohydric alcohols.

B) Complex lipids: These are esters of fatty acids containing groups in addition to an alcohol and a fatty acid groups)

• Phospholipids: lipids containing in addition to fatty acids and an alcohol, a phosphoric acid residue.

• Glycolipids: lipids containing a fatty acid, sphingosine and carbhohydrate.

Other complex lipids**:** lipids such as sulfolipids and aminolipids. Lipoproteins may also be placed in this category.

C) Derieved lipids: These include fatty acids, glycerol, steroids, other alcohols, fatty aldehydes, hydrocarbons, lipid soluble vitamins and hormones.

FATTY ACIDS

They are present as such in minute concentrations in plasma cells. Fatty acids occur mainly as esters in natural fats and oils but do occur in the unesterified form as free fatty acids, a transport form found in the plasma. Fatty acids that occur in natural fats are usually straight chain derivatives containing an even number of carbon atoms.

Lipid Chemistry

Fatty acids are of two types

a. Saturated fatty acids - Those which contain no double bonds.

b. Unsaturated fatty acids- Those which contain one or more double bonds.

Saturated fatty acids having 10 or less carbon atoms are called "Lower fatty acids"

e.g. - Acetic acid, butyric acid

Saturated fatty acids having more than 10 carbon atoms are

Called "higher fatty acids". e.g. - Palmitic acid, stearic acid.

Unsaturated fatty acids are classified according to degree of saturation.

1. **Mono unsaturated fatty acids** - These are those fatty acids which contain one double bond. E.g. Oleic acid.

2. **Polyunsaturated fatty acids** - These are fatty acids which contain more than one double bond.

eg. Linoleic acid series

Linolenic acid series

Arachidonic acid series

Essential fatty acids are those which cannot be synthesized in body and must be provided in the diet. Lack of these essential fatty acids in diet can produce growth retardation and other deficiency syndromes. Free fatty acids are immediately available energy source and provide much of the energy requirements of body. Normal value ranges from 250 - 400 mg / dl.

CHOLESTEROL

Cholesterol is widely distributed in all cells of the body. It is the best known steroid because of its association with atherosclerosis and heart disease.

It occurs as a white or faintly yellow, almost odorless, pearly leaflets or granules. It is insoluble in water. Sparingly soluble in alcohol and soluble in ether, chloroform, hot alcohol, ethyl acetate alcohol and vegetable "oil.

Cholesterol is found in largest amounts in normal human adult brain and nervous tissue of about 20%, in liver - 0.3%, skin - 0.3%, intestinal mucosa 0.2% and certain endocrine glands namely adrenal cortex contain about 10%.

The normal level of serum total cholesterol in adults varies from 150-250 mg/dl

TRIGLYCERIDES (NEUTRAL FATS)

These molecules are used to provide energy. In the body, stored fat in adipose tissue is the storage form of energy.

Important sites of adipose tissue are subcutaneous tissue around some internal organs and omentum. Fat under the skin prevent heat loss in winter and the intestinal organs get support from fat around them. The triglycerides constitute the body's main caloric reserve. Normal value ranges from 40-150 mg %.

PHOSPHOLIPIDS

Phospholipids are compound lipids. They contain in addition to fatty acids and glycerol one more alcohol or phosphatidic acid, residue nitrogen containing base and other substituents.

Phosphatidic acid is important as an intermediate in the synthesis of triacylglycerols as well as phosphoglycerols but is not found in any great quantity in tissues.

They are classified into 3 groups.

1. **Glycerophopsholipid** - Here glycerol is the alcohol group.

eg - Lecithin, Cephalin

2. **Phospho inositides** - Here inositol is the alcohol group.

eg - phosphatidylinositol

3. **Sphingophospholipids** - Here sphingol is the alcohol group.

eg - Sphingomyelin

The lecithins are the most abundant phospholipids at the cell membrane and represent a large portion of body's store of choline.

Dipalmitoyl lecithin is a very effective surface active agent and a major constituent of the surfactant preventing adherence, due to surface tension of the inner surface of lungs.

Phosphatidylethanolamine (cephalin) and phosphatidyl serine differ from phosphatidylcholine ony in that ethanolamine or serine, respectively replaces choline.

Sphingomyelins are found in large quantities in brain and nerve tissue.

On hydrolysis, the sphingomyelins yield a fatty acid, phosphoric acid, choline and a complex amino alcohol, sphingosine. No glycerol is present. The combination of sphingosine plus fatty acid is known as ceramide, a structure also found in the glycophospholipids.

GLYCOLIPIDS

Glycolipids are widely distributed in every tissue of the body, particularly in nervous tissue such as brain. They occur particularly in the outer leaflet of the plasma membrane, where they contribute to cell surface carbhohydrates.

The major glycolipids found in animal tissues are glycosphingolipids. They contain ceramide and one or more sugars. Galactosylceramide is a major glycosphinglipid of brain and other nervous tissue, found in relatively low amounts elsewhere. It contains a number of characteristic C24 fatty acids, eg, cerebronic acid.

Galactosylceramide can be converted to sulfogalactosylceramide (sulfatide), present in high amounts in myelin. Glucosylceramide is the predominant simple glycosphingolipid of extraneural tissues, also occurring in the brain in small amounts.

Gangliosides are complex glycosphingolipids derived from glucosylceramide that contain in addition tone or more molecules of sialic acid. Neuraminic acid is the principal sialic acid found in human tissues. Gangliosides are present in nervous systems in high concentration.

LIPO PROTEINS OF PLASMA

In plasma, cholesterol and triglycerides form integral component of macromolecule complex called lipoprotein which are conjugated proteins. Lipid part is the prosthetic group and lipid free protein are designated as apolipoproteins or apo proteins. Protein separation including electrophoresis and ultracentrifugation shows progress in lipoprotein chemistry.

Teselius et al in 1941 reported existence of two lipoprotein classes separated by moving boundary electrophoresis.

In 1954 Gofmen et al separated lipoproteins by ultra centrifugation into five major density classes

OVERVIEW OF LIPOPROTEINS AND LIPOPROTEIN METABOLISM

Lipoproteins are microemulsions composed of lipids (cholesterol, cholesteryl ester, triglyceride and phospholipid) and proteins (apoproteins). Their function is to transport non water soluble cholesterol and triglycerides in plasma. Lipoproteins are spherical particles containing a central core of non-polar lipids (primarily triglycerides and cholesteryl ester) and a surface monolayer of phospholipids and apoproteins

Lipoproteins have been classified on the basis of their densities during ultracentrifugation into [60]:

1) Chylomicrons
2) Very low density lipoprotein (VLDL)
3) Low density Lipoprotein (LDL)
4) High density lipoprotein
5) Lipoprotein a

CLASSIFICATION OF LIPOPROTEINS [60]

Lipoprotein	source	Diameter (nm)	Density (g/ml)	Composition		Main lipid components
				Protein (%)	Lipid (%)	
Chylomicrons	Intestine	90-1000	<0.95	1-2	98-99	Triacylglycerol
VLDL	Liver	30-90	0.95-1.006	6-8	92-94	Triacylglycerol
LDL	VLDL	20-25	1.019-1.063	21	79	Cholesterol
Lp(a)		25-35	1.040-1.063	36	55	Cholesterol
HDL	Liver, intestine, VLDL chylomicrons					Phospholipids Cholesterol
HDL1		20-25	1.019-1.063	32	68	
HDL2		10-20	1.063-1.125	33	67	
HDL3		5-10	1.125-1.210	57	43	

CHYLOMICRONS

Chylomicrons are the largest of lipoproteins. They measure about 90-1000nm in diameter and they have the least density as compared to other classes of lipoproteins [60].

These are particles that are primarily triglyceride bearing and are produced by the intestine after exogenous fat undergoes digestion. These are responsible for the transport of dietary triglycerides and cholesterol. Dietary triglycerides are hydrolyzed in the gut, releasing monoglycerides and fatty acids that are then reesterified to form triglycerides in the intestinal mucosal cell.

These triglycerides are assembled with newly absorbed cholesterol, apoprotein B48 and the A apoproteins. Upon secretion from the enterocyte, these assembled particles enter the lymphatic circulation and then the bloodstream, where they acquire C apoproteins and apo E by transfer from HDL.

As chylomicrons enter the plasma, the triglycerides are rapidly hydrolyzed by the enzyme lipoprotein lipase (LPL), which resides on the surface of capillary endothelial cells. LPL is synthesized primarily in adipose tissue and striated muscle. It is secreted and transported to the endothelial surface, where it acts on triglyceride rich particles. Its action requires the presence of apo CII on the surface of the lipoprotein, whereas apo CIII inhibits LPL.

As triglyceride is depleted from the chylomicrons, phospholipids and A and C apoproteins are transferred to HDL. The residual chylomicron particle, which has lost 80 to 90 % of its triglyceride and is now relatively cholesterol enriched is called chylomicron remnant.

VERY LOWDENSITY LIPOPROTEINS

VLDLS are synthesized in the endoplasmic reticulum of hepatocytes and are composed of endogenous triglyceride derived from plasma free fatty acids, chylomicron remnants and from de novo lipogenesis [60].

Nascent VLDLs are secreted into the circulation; contain apoB100 and small amounts of apo C and apo E. After VLDLs enter the circulation, they are metabolized in the same manner as chylomicrons by the enzyme LPL, with the fatty acids that are liberated following the same fate as those liberated from chylomicrons. After secretion, VLDLs acquire more C and E apoproteins by transfer from HDL. In addition, free cholesterol is progressively exchanged to HDL, where it is esterified and the cholesteryl ester is returned to VLDL.

As VLDLs become progressively depleted of triglyceride, a portion of the surface, including cholesterol, apolipoproteins C and E and phospholipids is removed and contributes to nascent HDL particles.

LOW DENSITY LIPOPROTEINS

LDLs are products of the metabolism of VLDL. They have got a diameter of 18-25nm. The only apoprotein in LDL is apo B and only one molecule of apo B is present per particle of LDL. Clearance of LDL is mediated by a specific receptor present on the surface of both liver and peripheral cells. Once it is bound to the receptor, the lipoprotein is internalized by an endocytic process. The vesicle then fuses with a lysosome, where enzymes degrade the apoB and hydrolyze the cholesteryl ester to free cholesterol.

The smaller remnants of VLDL are triglyceride depleted, cholesterol rich particles, some of which are isolated in the IDL compartment, although some remain in the VLDL compartment.

VLDL particles are believed to be secreted in a spectrum of sizes with various degrees of triglyceride enrichment. The larger VLDL particles appear to be more rapidly cleared and less likely to be converted to LDL. On the other hand, smaller VLDL particles that are richer in cholesterol may be preferentially converted to LDL.

All peripheral cells express the LDL-receptor (LDL-R), and recycle it to the cell surface upon need for cholesterol. Cholesterol is delivered to these cells through binding of LDL to LDL-R, which triggers endocytosis (internalization) of both species. When the need for cholesterol is satisfied, the recycling of LDL-R is discontinued. Normally, an LDL particle stays in circulation for no more than a few days before being consumed by a cholesterol needing cell.

However, under conditions of sustained cholesterol excess, the particle stays in circulation for longer periods of time, and becomes more vulnerable to undesired modifications (e.g. oxidation). As high levels of oxidized LDL are commonly found in atherosclerotic plaques, they are thought to be the major inducer of atherosclerotic lesions. Hence, LDL became known as bad cholesterol

HIGH DENSITY LIPOPROTEINS

HDLs also are represented by a spectrum of particles of various sizes and densities. HDLs are 18-25nm in diameter.

HDL is synthesized in the liver and intestine as a nascent, discoid-shaped particle that contains predominantly apoA-I, and some phospholipids [60]. Upon maturation, HDL assumes a spherical shape, and the composition of its core lipids becomes very similar to that of LDL. However, the relative higher protein content in HDL renders the particle denser and more resistant to undesired modifications.

Unlike LDL, HDL is not recognized by LDL-R, and cannot deliver cholesterol to tissue cells. Instead, it has the ability to remove excess peripheral cholesterol and

return it to the liver for recycling and excretion. This process, called reverse cholesterol transport, is thought to protect against atherosclerosis. Observational studies over the last 2 decades have consistently shown strong correlation between elevated HDL levels and low incidents of coronary artery disease (CAD). Hence HDL has been dubbed “good” cholesterol.

LIPOPROTEIN (a)

Lipoprotein (a) or Lp (a) has been established as an independent CAD risk factor. The structure is similar to that of an LDL molecule linked by a disulphide bridge to apoprotein A.

Lp (a) levels range from 1-100mg /dl with the largest number of values below 20 mg/dl.

Although Lp (a) is structurally similar to LDL, the former appears to be regulated independently and carries an independent relation to overall coronary risk. If serum levels of both LDL and Lp (a) are elevated the risk of CAD is markedly increased.

The mechanism by which high levels of Lp (a) are related to coronary atherosclerosis is unclear. It has been suggested that because of the structural similarities of Lp (a) to plasminogen, high levels of Lp (a) may inhibit the thrombolytic activity of naturally occurring tissue plasminogen activity.

An alternative explanation for the association between elevated Lp (a) levels and atherosclerosis in that Lp (a) may somehow alter the LDL mediated delivery of cholesterol to the atherosclerotic plaque.

APO PROTEINS

Apo proteins are key lipoprotein components that serve both as enzymatic cofactors and as recognition elements that bind to specific receptors on peripheral tissues, including the vascular endothelial cells.

It is the Apo E component of the chylomicron remnant that is recognized by receptors on the hepatocyte. This apoproteins are distinguished alphabetically and numerically as Apo A I through Apo E.

A great deal of research has been conducted in the use of apoproteins as CAD markers. Some investigations have found that the concentration of Apo AI and Apo B 100 are better predictors of CAD then are measurements of total plasma lipids or lipoprotein.

Apoprotein – A

Apo AI, the prototype of Apo A, is a major protein in HDL and also is seen in chylomicrons.

Apo AII is a minor constituent of HDL and does not appeal to be present in all species. Human Apo AII is of hepatic origin. Apo AII maybe an activator of hepatic triglyceride lipase which hydrolyses triglyceride and phospholipid while utilizing HDL2 as its preferred substrate. Apo A IV is synthesized in the gut and is present in HDL, chylomicrons and as a free protein. It may also be an activator of LCAT.

The genetic codes for Apos AI, A IV and CIII are close together on the long arm of chromosome II. Combined AI CIII deficiency is associated with severe premature atherosclerosis.

Apoprotein - B

Apo B occurs in two forms namely Apo B 100 and B28. Apo B 100 is found in VLDL, IDL, and LDL. Apo B 100 is the primary apoprotein of LDL and accounts

for 25% of its weight. It is also the recognition site for the LDL, or Apo B/E receptor on cell surfaces. The gene for Apo B 100 has been localized to chromosome 2.

The structure in the amino acid sequence of human Apo B 100 and the corresponding CDNA messenger CID has recently been determined. A unique editing mechanism introduces a stop codon into the mRNA for Apo B by means of single base change. This allows the biosynthesis of two proteins from a single gene and mRNA with either Apo B -100 or Apo B 48 being synthesized. Apo B 48 is synthesized by small intestine and Apo B 100 is secreted by the liver.

Apoprotein - E

Apo E accounts for about 15 percent of the protein content of VLDL, 7 percent of chylomicron remnants and 2 percent of protein content of HDL.

It can be recognized by the LDL or Apo B/E receptor and by specific Apo E receptors in the liver whose function appears to be the removal of chylomicron remnants Apo E is polymorphic and contains three major alleles, Apo E2, E3 and E4. The various combinations results in homozygote are Apo E2/2 EG/4. Also Apos E2/3, E2/4 and E3/4 exist in the heterozygous state. The polymorphism of Apo E has been determined on a molecular basis and results from the substitution of an amino acid at residues 112 and 158 in the protein. Apo E isoform may account for as much as 15 percent of variability of cholesterol and LDL levels in the population.

Recent Finnish studies suggest that Apo E4 may be associated with increased cholesterol absorption in the GI tract.

CHARACTERISTICS AND MECHANISMS OF DYSLIPIDEMIA IN DIABETES

Dyslipidemia is a relatively common problem in patient with diabetes mellitus as compared to non-diabetics. Patients with diabetes typically have an atherogenic lipid profile characterized by elevated triglycerides, increased LDL, VLDL and cholesterol and decreased HDL. There are several reasons for this association:

First, insulin plays an important role in the regulation of intermediary lipid metabolism and fluctuations in the degree of diabetic control thus produce a variable effect on plasma lipoprotein metabolism.

Secondly, many non-insulin dependent diabetic patients are obese, and obesity leads to the development of hyperlipidemia.

Increased LDL

There is an increased proportion of small dense LDL particles. Total LDL levels may be modestly increased but are comparable with those of the general population, although an individual with type 2 diabetes mellitus and a total LDL of 100 mg/dl may actually have many more circulating small, dense LDL particles than an individual with normal insulin sensitivity and the same LDL level.

Hypertriglyceridemia

Triglycerides come from the diet (exogenous) or are newly synthesized by the liver (endogenous) using dietary carbohydrate precursors and re esterified fatty acids absorbed from peripheral tissues. Exogenous triglycerides circulate as chylomicrons, while endogenous triglycerides combine with hepatic cholesterol to form VLDL particles, which are secreted into the circulation by hepatocytes.

Abnormalities of LPL activity, HSL activity, and fatty acid metabolism all contribute to baseline and often extreme postprandial hypertriglyceridemia associated with type 2 diabetes and insulin resistance.

Although fasting LPL levels are typically increased in the setting of obesity because of the large number of adipocytes, insulin resistance at the level of the fat cell causes decreased LPL activity and therefore, an abnormal response of LPL to a glucose load.

Diminished LPL activity leads to an accumulation of atherogenic LDL precursors, such as VLDL in the circulation. HSL activity is increased in type 2 diabetes, which causes increased circulating FFA.

In the setting of insulin resistance, adipocytes take up less circulating FFA. This situation, called reduced fatty acid trapping, allows excess FFA delivery to the liver, which, in turn, causes increased hepatic secretion of VLDL particles. This is most pronounced and prolonged after a meal [61,62,63].

Decreased HDL

The decreased HDL in NIDDM is mostly reflected in decreases in the HDL2 subfraction [64].

Although it is not completely understood how hepatic lipase acts in the regulation of HDL, it is possible that the lower HDL concentrations in NIDDM may in part be attributable to higher hepatic lipase activity. Hepatic lipase is elevated in obese female NIDDM subjects and increased in thin male NIDDM individuals, the activity in the latter group decreasing after normalization of glycemia with insulin therapy.

Reduced HDL leads to diminished clearance of cholesterol from peripheral tissues. The actions of CETP illustrate why hypertriglyceridemia and reduced HDL typically go hand in hand in patients with type 2 diabetes. In the setting of elevated circulating triglycerides, CETP allows an increased influx of VLDL triglycerides into HDL particles. This occurs as an exchange reaction, with a simultaneous efflux of

cholesteryl ester out of HDL particles. This process leads to reduced HDL levels owing to increased clearance of HDL particles.

There is also reduced production of HDL particles in type 2 diabetes owing to abnormal LPL activity, causing decreased conversion of dense, triglyceride- rich HDL to more buoyant particles.

Qualitative Changes in LDL: LDL Particle Size

As already mentioned, although the total LDL level may be normal or only modestly elevated in patients with type 2 diabetes and obesity there are frequently qualitative changes in LDL particles that confer increased risk of CAD.

As individuals proceed from normal insulin sensitivity to insulin resistance, VLDL particles become larger and LDL particles become smaller. Individuals with type 2 diabetes have been shown to have smaller, denser LDL particles even after adjusting for elevated triglyceride levels and lower HDL levels.

LDL particles are divided into two subclasses based on particle size and atherogenicity. Pattern A particles are buoyant and pattern B particles are small, dense and more atherogenic. Pattern B molecules are formed by a lipid exchange process similar to that that occurs in HDL particles in the setting of elevated triglycerides. Through the actions of CETP, triglyceride from VLDL is exchanged with cholesteryl ester from LDL. Hydrolysis of the triglyceride-rich LDL particle produces smaller, denser LDL particles.

Pattern B particles are considered more atherogenic than pattern A particles for several reasons: they adhere to and penetrate the arterial wall more easily, they are more toxic to endothelial cells, they exert a procoagulant effect by causing greater production of plasminogen activator inhibitor-1 by endothelial cells, and they are oxidized more easily.

Studies done in the general population and in diabetic populations have shown that individuals with elevated levels of pattern B particles are at higher risk of CAD.

Glycosylation of LDL

Epidemiological data have demonstrated that higher HbAlC levels are associated with higher rates of CAD. Severe hyperglycemia may worsen diabetic dyslipidemia via glycosylation of LDL particles. Glycosylated LDL particles are thought to have increased atherogenicity. Advanced glycation end products may modify LDL particles such that they have reduced affinity for hepatic LDL receptors and thereby a prolonged half-life.

LDL particles that "live longer" may have a greater likelihood of becoming oxidized or taken up by macrophages, in turn leading to the formation of foam cells [65,66].

Increased VLDL:

The most common alteration of lipoproteins in NIDDM is an elevation in VLDL, as reflected by either increased total triglyceride or VLDL triglyceride concentrations. Abnormalities in both production and clearance of VLDL triglyceride have been reported in NIDDM. Several studies have observed an overproduction of VLDL triglyceride. Although there are fewer studies of VLDL Apo B metabolism in NIDDM, results indicate that there is a clearance defect similar to that for VLDL triglyceride, whereas VLDL Apo B production may be influenced primarily by obesity. Subjects with type 2 diabetes mellitus have a decrease in fractional catabolic rate (FCR) for VLDL Apo B [67].

Haffner et al [68] found slower clearance of chylomicron Apo B in hyperlipidemic subjects with NIDDM. The proportional decrease in clearance of VLDL Apo B was similar to that observed for VLDL triglyceride [69].

There appear to be changes in the composition of VLDL in NIDDM which may either reflect or be the cause of alterations in VLDL metabolism. Several studies have suggested that diabetics may have a large, triglyceride rich VLDL [70].

There are multiple alterations in VLDL metabolism in non-insulin-dependent diabetes mellitus. NIDDM appears to induce an overproduction of VLDL triglyceride and to a lesser extent, of VLDL Apo B. FCR for both VLDL triglyceride and Apo B are lower and are associated with lower activities of LPL.

Finally, there are indications that the VLDL particle in NIDDM has altered composition. The mechanism for the overproduction of VLDL is not clear. The most likely explanation is that it is a result of the increased flow of substrates, particularly glucose and free fatty acids, to the liver.

ATHEROSCLEROSIS AND DIABETES

Accelerated coronary and peripheral vascular atherosclerosis is one of the most common and serious chronic complications of long term diabetes. Coronary artery disease mortality is two to four times higher in diabetic individuals than in non-diabetic individuals and is the leading cause of death in both type 1 and type- 2 diabetics.

Although diabetic patients have a higher incidence of other cardiovascular risk factors such as hyperlipidemia, hypertension and obesity, even when these risk factors are taken into consideration, diabetics still experience an increase in cardiovascular disease compared to non-diabetics [71]. This increased risk is more in women.

In the Framingham heart study, the relative risk of cardiovascular death in diabetic versus non-diabetic subjects was 21 for men and 49 for women. Although the Framingham study included mostly type-2 diabetic patients, increased risk of CAD is also found in type-2 diabetes.

The Joslin clinic in Boston followed a cohort of 292 patients with type 2 diabetes for 20 to 40 years. They found a large excess of cardiovascular mortality in their patients which also was impressive in females than males.

By age 55, the cumulative mortality due to CAD was 35 % compared with 8% in men and 4% in women for non-diabetics in the Framingham Heart Study.

Many factors contribute to the increase in atherosclerosis in diabetics, which include alterations in platelet function, clotting factors, and arterial smooth muscle cell metabolism and blood pressure regulation. Lipid abnormalities represent an important cardiovascular risk factor in type 2 diabetes which is accelerated by obesity. Also associated changes in plasma lipid and lipoprotein levels in diabetes remain important to explain the accelerated atherosclerosis.

Similar to the non-diabetic population, increases in total cholesterol and LDL and decrease in HDL are more prevalent in diabetics CAD than those without CAD.

In addition increases in plasma triglycerides in some studies have been better predictors of CAD in diabetics than increase in cholesterol.

The risk of CAD may be correlated with increases in blood glucose levels. Even mild abnormalities of glucose metabolism, such as patients with impaired glucose tolerance increases the risk of CAD mortality.

LIPIDS AND ATHEROSCLEROSIS

Several evidences have contributed to our current understanding of the relationship between increase in plasma cholesterol and development of CAD. 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 CAD.

In the Framingham study, for individuals below 50 years, cholesterol level was directly related to cardiovascular mortality. The Framingham study highlights the profound effects of lipoprotein abnormalities on incidence of coronary artery disease in diabetics compared to non-diabetics [72].

Although micro vascular disease is the specific lesion associated with diabetes, atherosclerosis accounts for majority of deaths in diabetic patients.

Atherosclerosis proceeds more rapidly and is more extensive in diabetic than non-diabetic individuals [73].

The onset of atherosclerosis occurs early in life with diffuse regular thickening of the arterial intima in childhood. The smooth appearance of the arterial tree is usually lost during the teenage years with formation of nodular aggregates or cushions of fibro-elastic tissue, termed fatty streaks [74].

Fatty streaks are collections of lipid, mainly cholesterol esters in macrophages and smooth muscle cells deposited in the intima of the artery. These fatty streaks are the precursors of the hallmark of atherosclerosis, the fibrous atheromatous plaque. The fibrous plaques are white lesions that usually protrude into the vessel lumen and consists of a core of cholesterol, cholesterol ester, phospholipid and necrotic cells covered by a fibrous cap of elastin and collagen. There is also proliferation of the smooth muscle cells into the media. Another important component is the foam cells, which contain a large quantity of lipid [75].

In the Framingham study, the presence of diabetes further increased the risk of a given cholesterol level. Because most cholesterol in plasma is transported in LDL, it is believed that it is responsible for the correlation between plasma cholesterol and CAD.

Another important predictor of cardiovascular risk is HDL level. The cholesterol content of HDL is relatively small fraction of plasma cholesterol (20-25%). This fraction is inversely related to cardiovascular risks HDL decreases, the risk of CAD increases and vice-versa.

In the Framingham study, average HDL level in middle aged men was 45 mg/dl and 55 mg/dl in the female counterparts.

The relationship between high triglyceride and CAD remains controversial. Some patients with hypertriglyceridemia are at increased cardiovascular risk others are not. However epidemiological studies suggest that the increased risk seen be due to other risk factors such as obesity, low HDL and hypercholesterolemia.

Hypertriglyceridemia also maybe a marker of an individual with a genetic defect of lipoprotein metabolism, such as accumulation of VLDL remnants which are associated with premature atherosclerosis.

An important factor for the development of atheromatous plaques is oxidized LDL, which causes increased chemotaxis of monocytes, and increased uptake of oxidized LDL by macrophages [76].

NATIONAL CHOLESTEROL EDUCATION PROGRAM GUDELINES [77]

In 1984, National Institute of Health convened a Consensus Development Conference on lowering blood cholesterol. After reviewing the evidence relating cholesterol levels to coronary heart disease, the panel unanimously concluded that elevated blood cholesterol levels is a major cause of CAD and that lowering blood cholesterol levels will reduce the risk of atherosclerosis. They recommended treating moderate risk adults with a low cholesterol diet and that high-risk adults be considered for drug therapy if not responsive to low cholesterol diet.

In 1985, The National Cholesterol Education Program was initiated by the National Heart Lung and Blood institute to reduce the prevalence of "elevated blood cholesterol levels.

The NCEP ATP III recommended that all adults over the age of 20 years and children of families with premature atherosclerosis should be tested for their serum cholesterol level. This initial blood sample for serum cholesterol screening can be obtained at any time of the day and does not require fasting.

Acute illness, myocardial infarction or surgery can lower cholesterol levels, so such patients should have their serum lipids measured again after 8-12 weeks later.

Current recommendations by NCEP ATP III are as follows:

Cholesterol levels

Desirable: <200 mg/dl

Borderline high: 200-239 mg/dl

High: >240 mg/dl

If serum cholesterol is less than 200 mg/dl, no further evaluation is required and patient asked to repeat the test in 5 years. Patients with borderline high cholesterol levels should be advised to follow a cholesterol lowering diet and repeat

the test in 1 year. Patients with cholesterol levels>200 mg/dl and CHD other cardiovascular risk factors, or with cholesterol>240 mg/dl should have a complete assessment of their lipid status. This requires a fasting sample of blood and measurement of total cholesterol, triglycerides, HDL and LDL (calculated as total cholesterol-HDL-TG/5).

The NCEP ATP III recommends that decisions about treatment of patients with hypercholesterolemia be based on

LDL levels as follows:

Desirable: < 100 mg/dl

Near or above optimal: 100-129 mg/dl

Borderline high: 130-159mg/dl

High: 160-189 mg/dl

Very High: ≥190 mg/dl

High-risk patients should begin a program of intensive dietary therapy. High risk patients are described by the NCEP as given above. Patients are considered to have a high risk status if they have following risk factors:

Major Risk Factors (Exclusive of LDL Cholesterol)

1. Cigarette smoking
2. Hypertension (BP > 140/90 mmHg or on antihypertensive medication)
3. Low HDL cholesterol (< 40 mg/dl)
4. Family history of premature CHD (CHD in male first degree relative <55 years; CAD in female first degree relative < 65 years)
5. Age (men >45 years; women >55 years).

INITIATE THERAPEUTIC LIFESTYLE CHANGES (TLC) IF LDL IS ABOVE GOAL [77]

1) TLC Diet:

— Saturated fat <7% of calories, cholesterol <200 mg/day

— Consider increased viscous (soluble) fiber (10-25 g/day) and plant stanols/sterols (2g/day) as therapeutic options to enhance LDL lowering

2) Weight management

3) Increased physical activity.

DRUG THERAPY

Drug therapy should be reserved for patients who after an adequate therapeutic lifestyle change therapy still have:

1) LDL> 129 mg/dl

2) High risk status and HDL<40 mg/dl.

The goal of the therapy is to reduce LDL to below 100 mg/dl and in high risk patients less than 70 mg/dl.

The major classes of drugs of consideration are

1) HMG COA reductase inhibitors:

a) Lovastatin (20-80 mg)

b) Pravastatin (20-40 mg)

c) Simvastatin (20-80 mg)

d) Fluvastatin (20-80 mg)

e) Atorvastatin (10-80 mg)

f) Cerivastatin (0.4-0.8 mg)

Lipid /Lipoprotein effects

LDL-C: decreased by 18-55%

HDL-C: increased by 5-15%

TG: decreased by 7-30%

Side Effects

Myopathy and Increased liver enzymes

Contraindications

Absolute:

- Active or chronic liver disease

Relative:

- Concomitant use of certain drugs

2) Bile acid sequestrants:

a) Cholestyramine (4-16 g)

b) Colestipol (5-20 g)

c) Colesevelam (2.6-3.8 g)

Lipid /Lipoprotein effects

LDL-C: decreased by 15-30%

HDL-C: increased by 3-5%

TG: no change or increase.

Side Effects

Gastrointestinal distress, Constipation and Decreased absorption of other drugs

Contraindications

Absolute:

- Dysbetalipoproteinemia
- TG >400 mg/dL

Relative:

- TG >200 mg/dL

3) Nicotinic acid

Immediate release (crystalline) nicotinic acid (1.5-3 gm), extended release nicotinic acid (Niaspan®) (1-2 g), sustained release nicotinic acid (1-2 g)

Lipid /Lipoprotein effects

LDL-C: decreased by 5-25% (may be increased with patients with high TG)

HDL-C: increased by 15-35%

TG: decreased by 20-50 %.

Side Effects

Flushing, Hyperglycemia, Hyperuricemia (or gout), Upper GI distress and hepatotoxicity

Contraindications

Absolute:

- Chronic liver disease
- Severe gout

Relative:

- Diabetes
- Hyperuricemia
- Peptic ulcer disease

4) Fibric acid derivatives

a) Gemfibrozil (600 mg BID)

b) Fenofibrate (200 mg)

c) Clofibrate (1000 mg BID)

Lipid /Lipoprotein effects

LDL-C: decreased by 5-20% (may be increased with patients with high TG)

HDL-C: increased by 10-20%

TG: decreased by 20-50 %.

Side Effects

Dyspepsia, Gallstones and Myopathy

Contraindications

Absolute:

- Severe renal disease
- Severe hepatic disease

It often takes 2-3 months of diabetic treatment to see the full effect on lipoprotein profiles and to restore tissue lipoprotein levels to normal.

Treatment of hypertriglyceridemia also remains controversial. The National Institute of Health Consensus Conference on Treatment of Hypertriglyceridemia recommended that fasting triglyceride level not be considered elevated unless >200 mg/dl. Hypertriglyceridemia always should be treated with better diabetic control, weight loss, alcohol restriction and fat restricted diet. Drug therapy is advised when TG levels are more than 200mg/dl.

Although the Level of HDL is inversely related to risk of CHD there is no evidence that raising isolated HDL levels with drugs in patients with otherwise normal plasma lipid levels reduces cardiovascular risk.

Reduction of high risk of CAD and peripheral arterial disease should be an essential part of diabetic management. Diet and glycemic control can improve serum lipid levels dramatically.

MATERIALS AND METHODS

Source of Data:

The material for the present study will be collected from patients who attend the Outpatient department and Inpatient department in BLDEU'S Shri.B.M.Patil Medical College Hospital and Research Centre, Bijapur over a period 2 years from January 2013 to June 2014. The sample size was 250 of which 125 were type 2 diabetes mellitus patients were studied as cases and 125 non diabetics were taken as controls and there lipid profile were computed.

Method of Collection of Data:

1. By detailed history
2. By detailed clinical examination
3. By relevant investigations like CBC, Urine for a) Albumin b) Sugar c) Microscopy, FBS, PPBS, HbA1c and fasting Lipid profile.

Inclusion Criteria:

1. **For cases**

Type 2 diabetes patients in the age group of 30-75 years and who are on either :

a) Oral hypoglycemic agents

b) Insulin

c) Both

2. **For controls**

Patients without type 2 diabetes mellitus age and sex matched.

Exclusion Criteria:

1. Known type 2 diabetics in the age group 30-75 years who are on oral hypolipidemic drugs.
2. Known type 2 diabetics in the age group 30-75 years who are suffering from

a) Chronic liver disease

b) Hypothyroidism

3. Known type 2 diabetics who are on drugs which cause hyperglycemia

a) Thiazides

b) Corticosteroids

c) Oral contraceptives

Blood sampling and preparation of serum.

The blood samples were drawn in the fasting state. 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 10 ml of blood was drawn using perfectly dry and sterile syringes and the blood was transferred to dried glass vials.

Serum was seperated within 2 hrs of collection to prevent artifical changes in concentration of HDL. The blood was centrifuged at 5000 rpm for 10 minutes. The supernatent clean serum was then pipetted out using dry piston pipettes with disposable tips and stored in dry thin walled vials at 4cc. The samples were analyzed the same day. Care was taken to exclude the haemolysed serum.

Laboratory procedure

Estimation of Blood Glucose

Glucose oxidase (GOD) oxidizes glucose to gluconic acid and hydrogen peroxide in presence of enzyme peroxidase, released hydrogen peroxide is coupled with phenol and 4-Aminoantipyrine (4-AAP) to form coloured quinoneimine dye. Absorbance of colored dye is measured at 505nm and is directly proportional to glucose concentration in the sample.

Estimation of Total Cholesterol (TC)

TC was determined by an enzymatic method. The cholesterol esters are hydrolyzed to free cholesterol by cholesterol esterase.The free cholesterol is then oxidized by Cholesteroloxidasetocholesten-3-one with the simultaneous production of hydrogen peroxide. The hydrogen peroxide produced couples with 4-AAP and phenol, in the presence of peroxidase, to yield a chromogenic with maximum absorbance at 505 nm.

Estimation of Total Triglycerides (TG)

In this direct colorimetric procedure, Serum triglycerides are hydrolyzed to glycerol and free fatty acids by lipoprotein lipase. In the presence of ATP and glycerol kinase (GK), the glycerol is converted to glycerol-3-phosphate, which then is oxidized by glycerol phosphate oxidase (GPO) to yield hydrogen peroxide. The oxidative condensation of ADPS and 4-aminophenazone in the presence of peroxidase (POD) and hydrogen peroxide produces a rose colored dye which is measured at 550 nm. The intensity of the colour formed is directly proportional to the triglycerides concentration in the sample.

Estimation of High-Density Lipoprotein Cholesterol (HDL)

HDL cholestrol was measured by an enzymatic method on the supernatant obtained after selective precipitation of apolipoprotein B-containing lipoproteins with phosphotungstic acid, in the presence of magnesium ions and centrifugation.

Estimation of Low-Density Lipoprotein Cholesterol (LDL)

Estimates LDL cholesterol using the Friedewald equation by subtracting the amount of cholesterol associated with other particles, such as HDL and VLDL, assuming a prolonged fasting state. LDL-C = TC- HDL- (TG/5)

Estimation of Very Low-Density Lipoprotein Cholesterol (VLDL)

In the absence of chylomicrons, only three forms of lipoproteins are present in the sera-VLDL, LDL and HDL. Since VLDL is the primary triglyceride carrying form in the fasting state, its concentration can be approximated by dividing the amount of plasma triglycerides by described by Friedwald formula in 1972**.**

VLDL= TRIGLYCERIDE/5

Statistical Analysis

a) Data will be presented by diaphragmatic presentation

   Mean $\pm$ SD

b) Results will be compared by

   x^2 test (Chi square test)

   't'- test

RESULTS

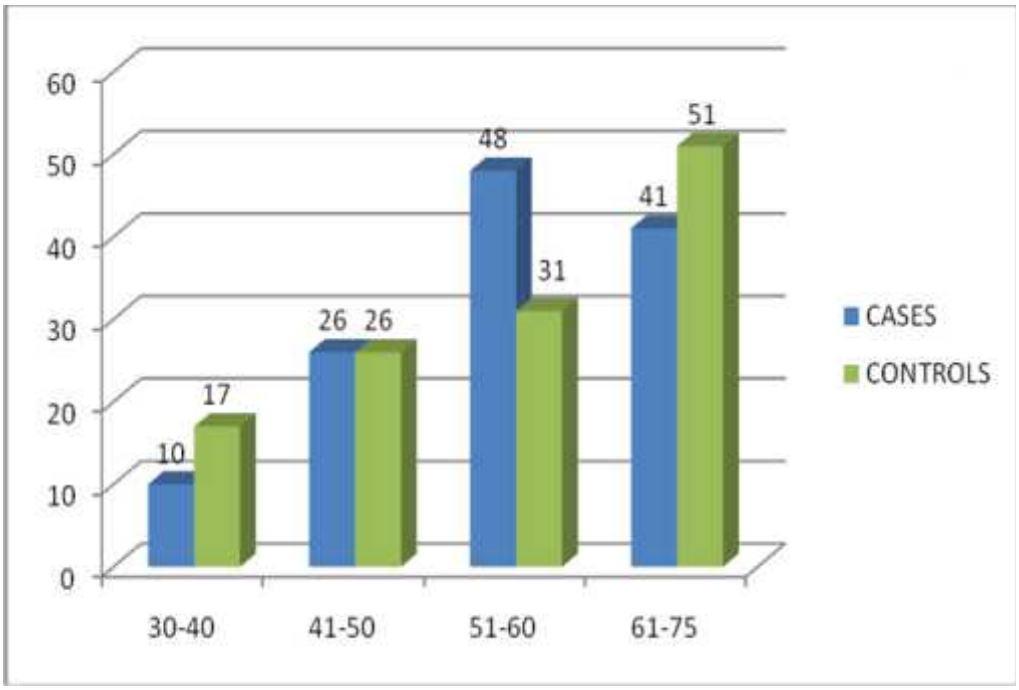
In present study, 125 type 2 diabetes mellitus patients and 125 non diabetics are considered as cases and controls respectively independent of sex.

AGE WISE DISTRIBUTION

Table no 1: Age wise distribution of patients

	Group		
Age (yrs)	Cases	Control	Total
30-40	10 (8%)	17(13.6%)	27(10.8%)
41-50	26(20.8%)	26(20.8%)	52(20.8%)
51-60	48(38.4%)	31(24.8%)	79(31.6%)
61-75	41(32.8%)	51(40.8%)	92(36.8%)
Total	125(100.0%)	125(100.0%)	250(100.0%)

Graph no 1: Age wise distribution of patients in cases & control



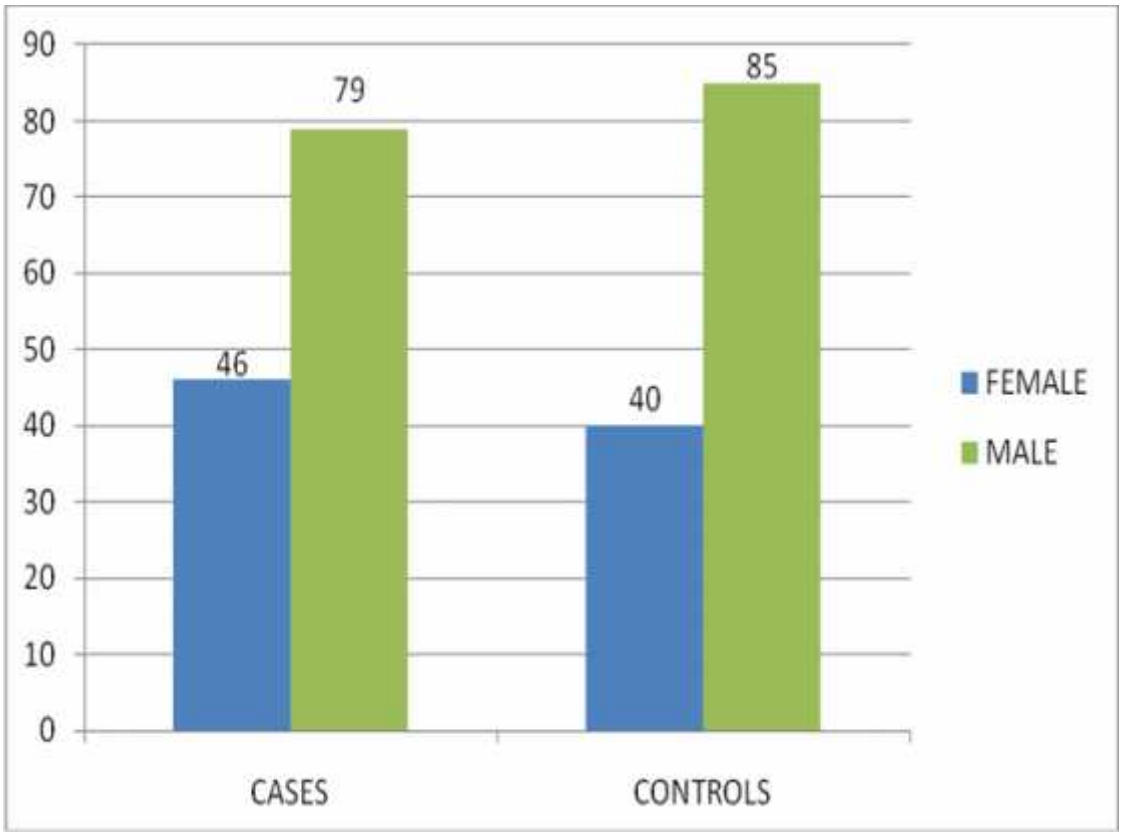
In the present study, 36.8 % of the patients (both cases and controls) ranged from 61-75 years. The mean age was 57 years in cases and 56.6 years in controls.

SEX WISE DISTRIBUTION

Table no 2: Sex wise distribution of patients in cases & controls

	Group		
	Cases	Control	Total
Female	46(36.8%)	40(32.0%)	86(34.4%)
Male	79(63.2%)	85(68.0%)	164(65.6%)
Total	125(100.0%)	125(100.0%)	250(100.0%)

Graph no 2: Sex wise distribution of patients in cases & controls



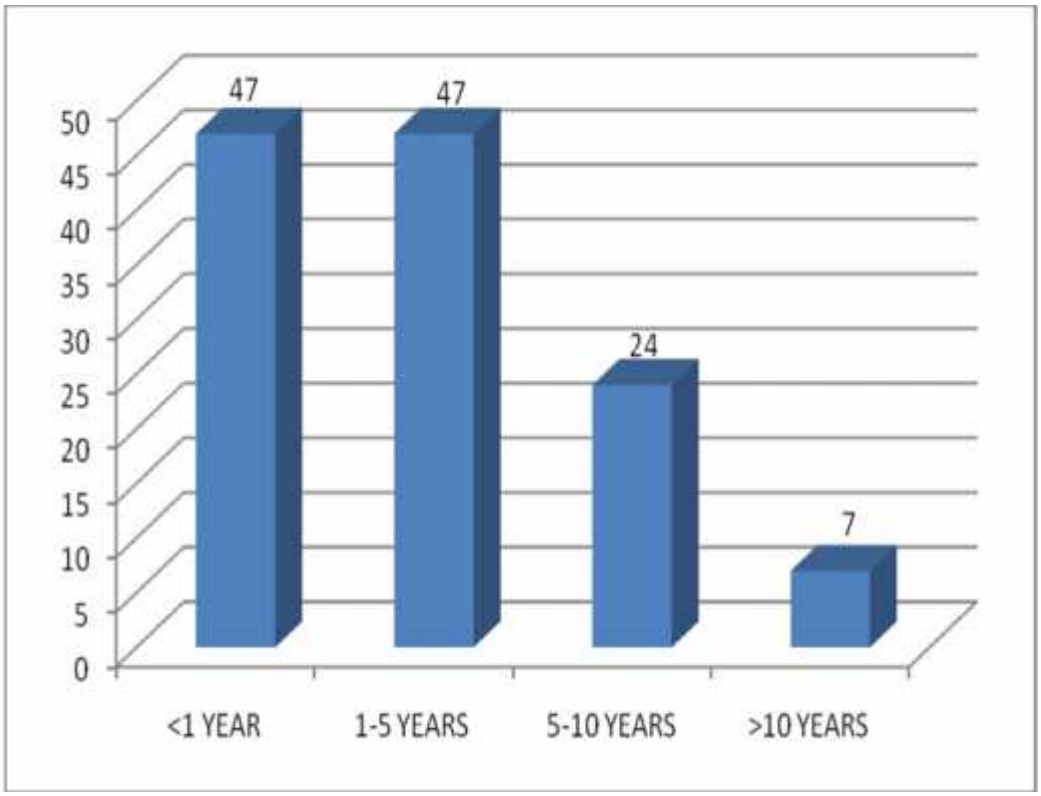
In the present study, cases and controls are taken independent of sex.

DURATION OF DIABETES:

Table no 3: Duration of diabetes in cases.

Duration(years)	Number	Percentage
<1	47	37.6 %
1-5	47	37.6 %
5-10	24	19.2 %
>10	07	5.6 %
Total	125	100 %

Graph no 03: Duration of diabetes in cases.



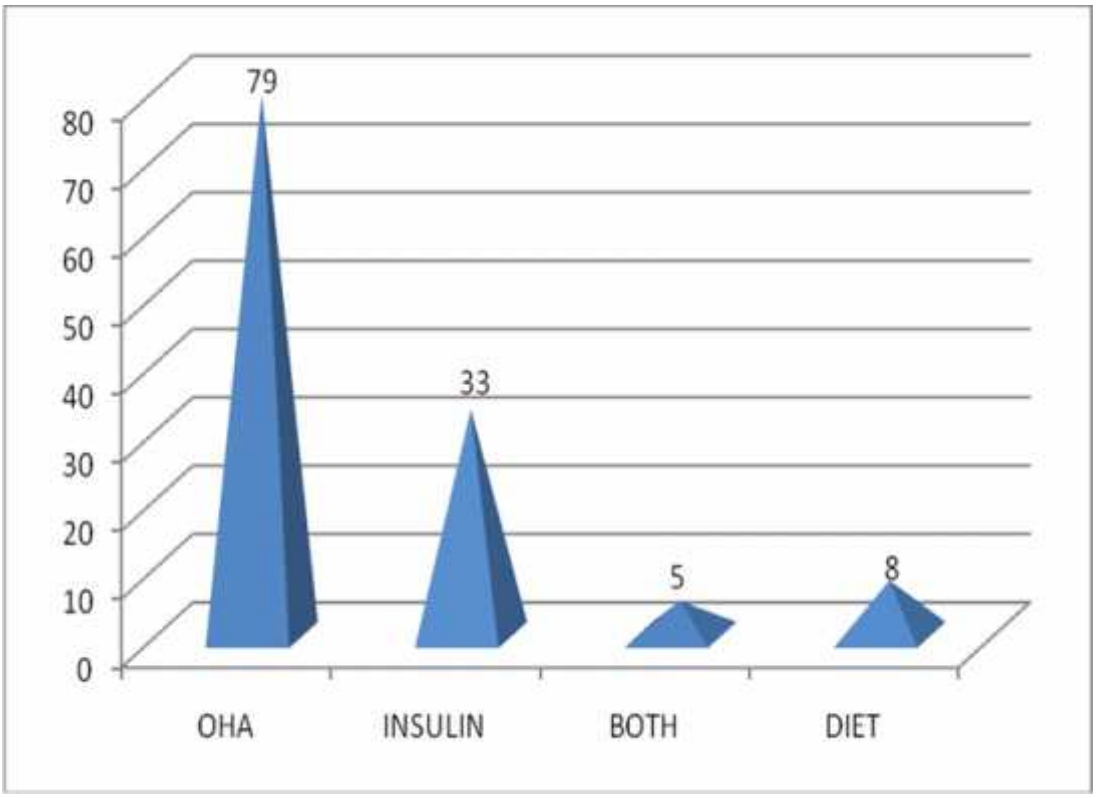
Out of 125 diabetic patients, duration of diabetes was <1 year in 47 patients, 1-5 years in 47 patients, 5-10 years in 24 patients and >10 years in 7 patients.

TYPE OF TREATMENT

Table no 4: Type of treatment

Treatment	Number	Percentage
OHA	79	63.2 %
INSULIN	33	26.4 %
BOTH	5	4.0 %
DIET	8	6.4 %
Total	125	100.0 %

Graph No 4: distribution of diabetes patients according to type of treatment.



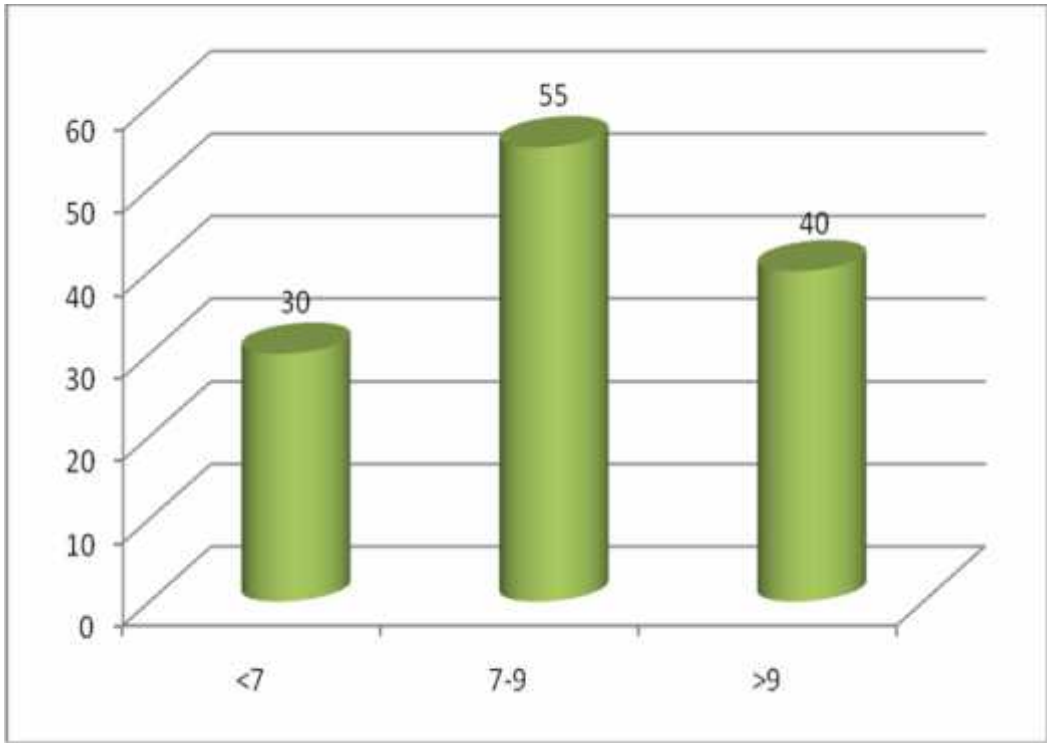
Out of 125 diabetics, 79(63.2%) were on Oral hypoglycemic agents, 33(26.4%) were on Insulin, 5(4%) were on both and 8(6.4%) on diet control.

DEGREE OF CONTROL OF DIABETES:

Table no 5: Distribution of patients according to HbA1C

HbA1C	Number	Percentage
<7	30	24.0 %
7-9	55	44.0 %
>9	40	32.0 %
Total	125	100.0 %

Graph no 5: distribution of patients according to HbA1C.



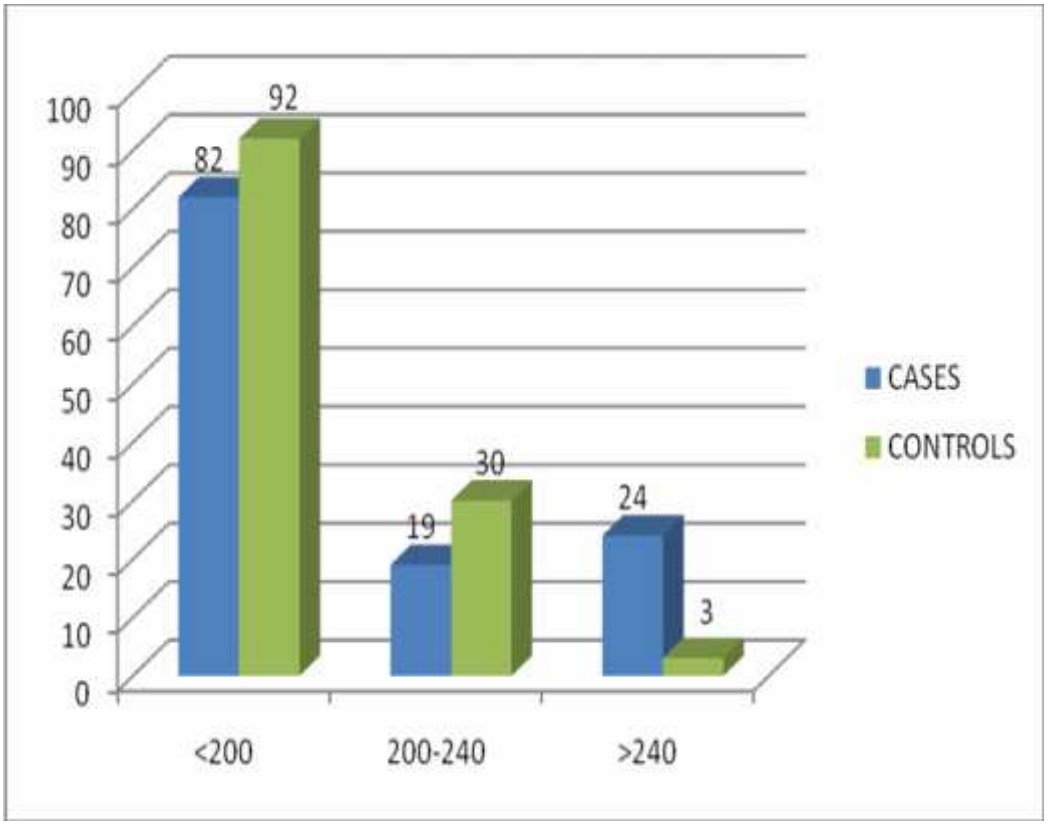
Out of 125 patients, 95 patients had poor glycemic control. 55 (44%) had HbA1c level of 7-9% and 40(32%) had HbA1c >9%.

TOTAL CHOLESTEROL IN CASES AND CONTROLS

Table no 6: distribution of patients according to Total Cholesterol

TC (mg/dl)	GROUP			Chi-square
	CASES	CONTROL	Total	P – value
<200	82(65.6%)	92(73.6%)	174(69.6%)	<0.0001
200-240	19(15.2%)	30(24.0%)	49(19.6%)	
>240	24(19.2%)	3(2.4%)	27(10.8%)	
Total	125 (100.0%)	125(100.0%)	250(100.0%)	

Graph no 6: Distribution of patients according to Total Cholesterol



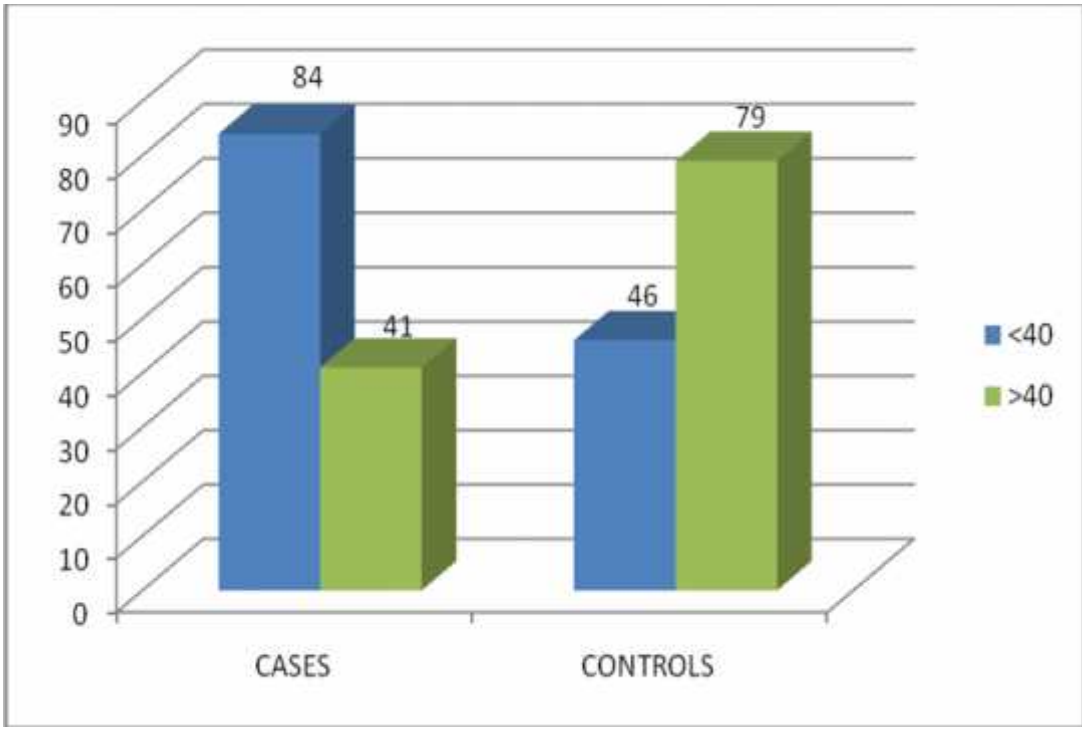
Total cholesterol are increased in cases as compared to controls with a $p < 0.0001$

HDL CHOLESTEROL IN CASES AND CONTROLS

Table no 7: Distribution of patients according to HDL

HDL (mg/dl)	GROUP		Total	Chi-square
	CASES	CONTROL		P - value
<40	84(67.2%)	46(36.8%)	130 (52.0%)	<0.0001
>40	41(32.8%)	79 (63.2%)	120(48.0%)	
Total	125(100.0%)	125(100.0%)	250(100.0%)	

Graph no7: Distribution of patients according to HDL.



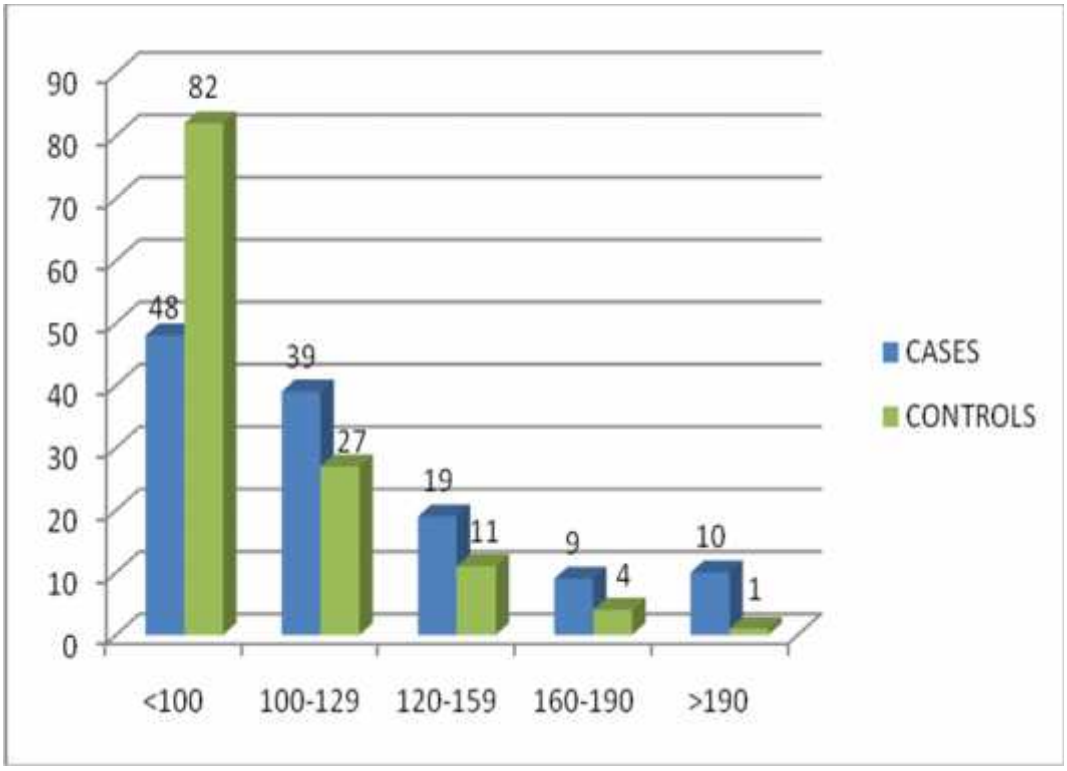
In the present study 84(67.2%) of cases have HDL <40mg/dl as compared to 46(36.8%) in control group.

LDL CHOLESTEROL IN CASES AND CONTROLS

Table no 8: Distribution of patients according to LDL

LDL (mg/dl)	GROUP			Chi-square
	CASES	CONTROL	Total	P - value
<100	48(38.4%)	82(65.6%)	130(52.0%)	<0.0001
100-129	39(31.2%)	27(21.6%)	66(26.4%)	
130-159	19(15.2%)	11(8.8%)	30(12.0%)	
160-190	9(7.2%)	4(3.2%)	13(5.2%)	
>190	10(8.0%)	1(0.8%)	11(4.4%)	
Total	125(100.0%)	125(100.0%)	250(100.0%)	

Graph no 8: Distribution of patients according to LDL



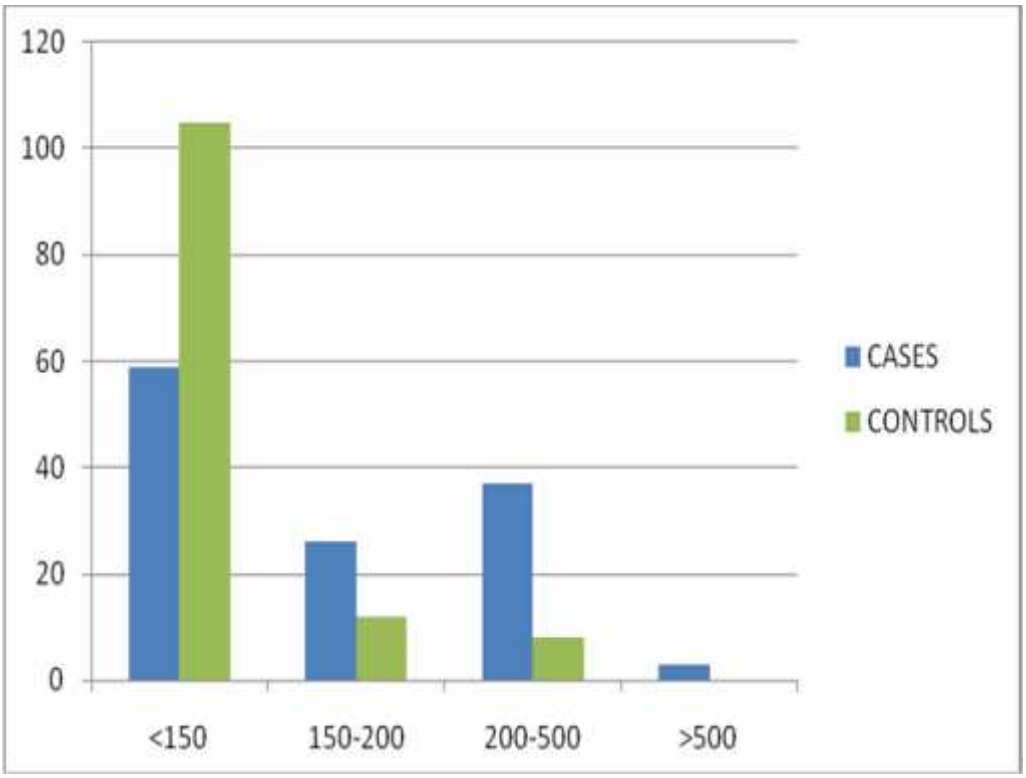
In the present study, levels of LDL is higher in cases as compared to control group.

TRIGLYCERIDES IN CASES AND CONTROLS

Table no 9: Distribution of patient's triglyceride level in case and control

TG (mg/dl)	GROUP			Chi-square
	CASES	CONTROL	Total	P – value
<150	59(47.2%)	105(84.0%)	164(65.6%)	<0.0001
150-200	26(20.8%)	12(9.6%)	38(15.2%)	
200-500	37(29.6%)	8(6.4%)	45(18.0%)	
>500	3(2.4%)	0(0%)	3(1.2%)	
Total	125(100.0%)	125(100.0%)	250(100.0%)	

Graph no 9: Distribution of patient's triglyceride level in case and control



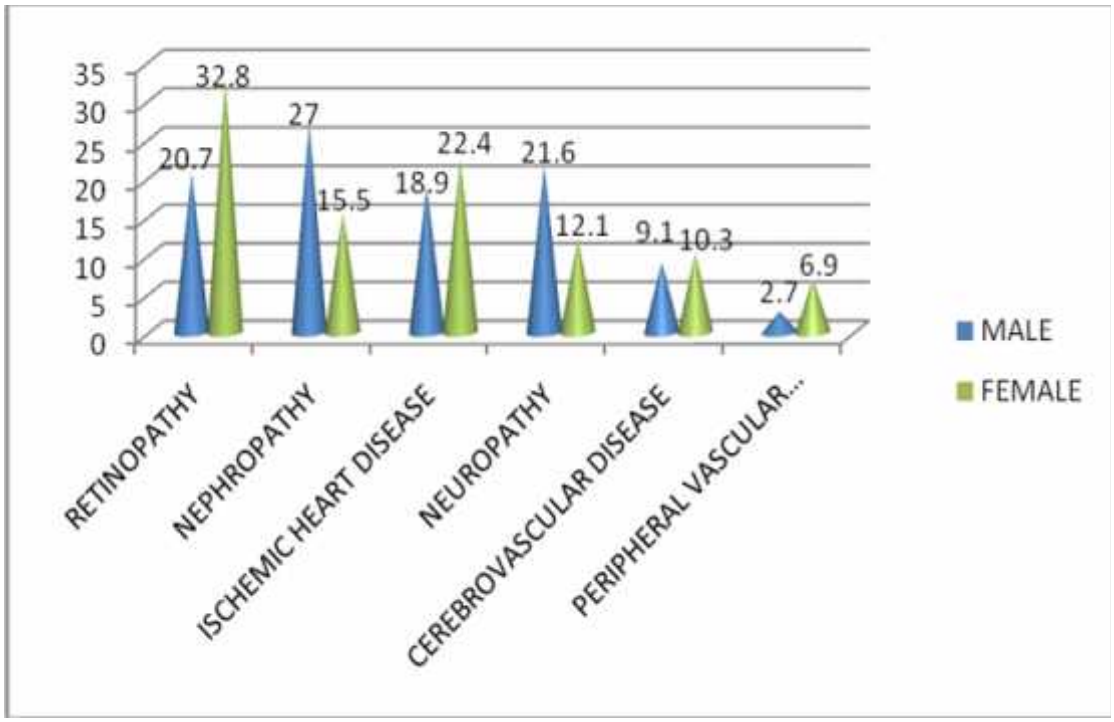
In the present study, triglyceride levels are higher in cases as compared to control group with a p value of < 0.0001.

INCIDENCE OF COMPLICATIONS

Table no 10: Incidence of complications in diabetic patients according to sex

	SEX			
COMPLICATIONS	MALE	%	FEMALE	%
RETINOPATHY	23	20.7	19	32.8
NEPHROPATHY	30	27.0	09	15.5
ISCHEMIC HEART DISEASE	21	18.9	13	22.4
NEUROPATHY	24	21.6	07	12.1
CEREBROVASCULAR DISEASE	10	9.1	06	10.3
PERIPHERAL VASCULAR DISEASE	03	2.7	04	6.9
TOTAL	111	100	58	100

Graph no 10: Incidence of complications in diabetic patients according to sex



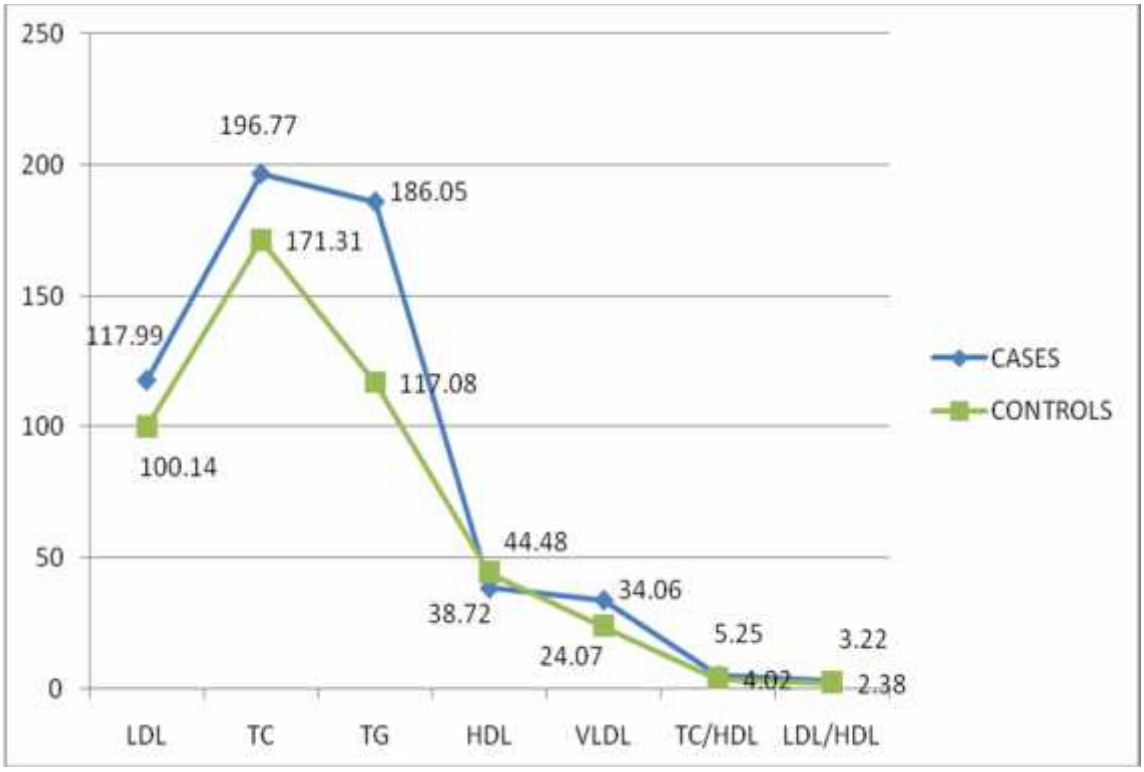
In the present study, Retinopathy and Nephropathy were the commonest complications. Next commonest was ischemic heart disease.

STUDY OF LIPID PROFILE IN CASES AND CONTROLS

Table No 11: lipid profiles in cases & control.

	GROUP						
	CASES			CONTROLS			P value
	MEAN	SD	SEM	MEAN	SD	SEM	
LDL	117.99	49.28	4.40	100.14	26.22	2.34	<0.0001
TC	196.77	73.60	6.58	171.31	37.3	3.33	<0.0001
TG	186.05	128.32	11.47	117.08	48.01	4.29	<0.0001
HDL	38.72	12.57	1.12	44.48	11.3	1.01	<0.0001
VLDL	34.06	19.65	1.75	24.07	10.96	0.98	<0.0001
TC/HDL	5.25	1.61	0.14	4.02	1.11	0.09	<0.0001
LDL/HDL	3.22	1.38	0.12	2.38	0.86	0.07	<0.0001

Graph No 11: Mean lipid profile in cases & control.



The above graph shows lipid profile in cases and controls overall – patient with type 2 diabetes mellitus studied have a higher level of:

LDL	–	mean	117.99 ± 49.28	cases
			100.14 ± 26.22	controls
TC	-	mean	196.77 ± 73.6	cases
			171.31 ± 37.3	controls
TG	-	mean	186.05 ± 128.32	cases
			117.0 ± 48.01	controls
HDL	-	mean	38.72 ± 12.57	cases
			44.48 ± 11.30	controls
VLDL	-	mean	34.06 ± 8.6	cases
			24.07 ± 10.96	control
TC/HDL-C	-	mean	5.25 ± 1.61	cases
			4.02 ± 1.11	controls
LDL/HDL-C	-	mean	3.22 ± 1.38	cases
			2.38 ± 0.86	control

Overall the values of TC, LDL and TG are increased in type 2 diabetes mellitus patients and HDL values are decreased in the same patients as compared to non-diabetics which is statistically significant with a p value of <0.001.

DISCUSSION

In the present study conducted at BLDEU'S Shri B.M.Patil Medical College Hospital and Research Centre, Bijapur which included a total of 250 subjects in which 125 cases of type 2 diabetes mellitus and 125 controls. The results obtained are computed with available study as follows.

A) Total cholesterol:

In the present study, serum Total cholesterol levels were raised in cases as compared to control groups, with a p value < 0.001 which is statistically significant. Aminul haq et al [26], Zargar AH et al [41] and Songa RM et al [27] have similar observations.

TC	Aminul haq et al [26]	Songa RM et al [27]	Zargar AH et al [41]	In the present study
CASES	191.61 ± 18.6	189.4 + 33.82	263.02 + 18.01	196.77 ± 73.6
CONTROLS	182.61 ± 17.6	181.9 + 32.36	215.9 + 33.3	171.3 ± 37.3

The levels of total cholesterol in the above studies are comparable to the present study with a P value of < 0.001 which is statistically significant.

B) Triglycerides:

In the present study, the serum triglycerides levels were significantly higher in cases as compared to controls (P <0.0001). Studies done by Aminul haq et al [26], Kandula R et al [29] and Songa RM et al [27] have similar observations.

TG	Aminul haq et al [26]	Songa RM et al [27]	Kandula R et al [29]	In the present study
CASES	170.15 ± 10.31	225.76 + 139.9	171.9 + 43.46	186.05 ± 128.32
CONTROLS	159.01 ± 10.11	167.6 + 65.61	169.09 + 26.33	117.08 ± 48.01

C) LDL cholesterol:

In the present study LDL cholesterol levels are significantly increased in cases as compared to controls (P <0.001.)

LDL-C	Aminul haq et al [26]	Zargar AH et al [41]	Songa RM et al [27]	In the present study
CASES	113.12 ± 11	164.96 + 16.8	104.02 + 35.04	117.99 ± 49.28
CONTROLS	102.69 ± 11.17	135.32 + 35.9	102.28 + 35.26	100.14 ± 26.22

The values of LDL-C are significantly raised in present study and it is comparable to above mentioned studies and both are having P < 0.001 which is statistically significant.

D) VLDL cholesterol:

VLDL-C levels are significantly increased in cases as compared to controls with a P value < 0.001 in the present study.

VLDL-C	Aminul haq et al [26]	Zameel T et al [35]	Songa RM et al [27]	In the present study
CASES	49.39.3 ± 6.43	47.6 + 7.62	46.14 + 28.56	34.06 ± 19.65
CONTROLS	31.80 ± 2	44.93 + 7.44	33.62 + 13.26	24.07 ± 10.96

Studies done by Zameel et al [35], Aminul haq et al [26] and Songa RM et al [27] have showed a significant increase in VLDL level in cases as compared to controls and these studies are comparable to our study.

E) HDL cholesterol

The present study showed the level of serum HDL-C is significantly decreased in cases as compared to controls with a p value of <0.001.

HDL-C	Aminul Haq et al [26]	Kandula R et al [29]	Songa RM et al [27]	In the present study
CASES	49.39 ± 6.43	36.65 + 3.23	40.86 + 8.45	38.72 ± 12.5
CONTROLS	57.69 ± 7.06	40.47 + 3.4	46.46 + 9.19	44.48 ± 11.3

Studies done by Aminul Haq et al [26], Kandula et al [29] and Songa RM et al [27] has similar results when compared with the present study.

CONCLUSION

The patients admitted to BLDEU'S Shri B.M.Patil Medical College Hospital and Research centre, Bijapur were selected for the present study. Total number of 250 subjects were studied which included 125 type 2 diabetes patients as cases and 125 non diabetics as controls.

Following are the important findings observed in the study

- Triglyceride levels were significantly high in cases compared to Controls
- LDL-C levels were significantly high in cases compared to controls.
- Total cholesterol levels were significantly high in cases compared to controls.
- HDL-C levels were significantly low in cases compared to controls.
- In conclusion type 2 diabetes mellitus patients are prone to develop dyslipidemia which is severe as compared to non-diabetic patients and hence the mortality and morbidity is increased in cases of type 2 diabetes mellitus.

SUMMARY

Diabetes mellitus is a major public health problem worldwide. In India the incidence is increasing very rapidly, particularly the incidence of type 2 diabetes mellitus. In type 2 diabetes mellitus, lipid abnormalities are almost the rule and is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities that are all recognized as major risk factors for coronary artery disease and other macro vascular complications. These lipid abnormalities are not only quantitative but also qualitative abnormalities of the lipoproteins which are potentially atherogenic.

In this study, the patients admitted to BLDEU'S Shri B.M.Patil Medical College Hospital and Research centre, Bijapur were selected and a total number of 250 subjects were studied which included 125 type 2 diabetes patients as cases and 125 non diabetics as controls.

There was significant increase in total cholesterol, LDL-C, Triglyceride, VLDL-C and decreased levels of HDL-C in cases as compared to the control group.

This study signifies the altered lipid levels in patients with type 2 diabetes mellitus as compared to non diabetics and early intervention of these patients will reduce the cardiovascular and cerebrovascular complications.

BIBLIOGRAPHY

1. Gopalan C. Raising incidence of obesity and diabetes, Nutrition Foundation of India;2006.
2. Tripathy BB, Chandalia HB, Das AK, Rao PV, Madhu SV. RSSDI Text book of Diabetes Mellitus.Vol 1. 2nd edition.
3. Albrink,.MJ et al. "Dietary and drug treatment of hyperlipidemia in diabetics". *Diabetes.*1974; 23:913-18
4. Bagdade J.D et al. "Diabetic lipemia. A form of acquired fat induced lipemia". *New England Journal of Medicine.*1967; *276:* 427-433.
5. Chance GW, Albutt EC, Edkins SM. Serum lipids and lipoproteins in untreated diabetics. Lancet.1969; 1:1126-28.
6. Sharma D, Bansal BC, Prakash C. Serum lipid studies in thin insulin dependent diabetics below the age of 30 years. J Indian Med Assoc 1970; 54 (9):416-20.
7. Nikkila EA and Hormila P. "Serum lipids and lipoproteins in insulin-treated diabetes.Demonstration ofincreased HDL concentrations". *Diabetes.*1978; 27: 1078-1086.
8. Retnam VJ, Nerurkar SV, Taskar SP, Bhandarkar SD. Lipid composition of plasma lipoproteins in treated diabetics. J Postgrad Med.1983; 29: 201.
9. Bijlani PK, Shah Kokila, Raheja BS. HDL cholesterol in diabetics. JAPI 1984; 32.
10. Hokanson JE, Austin MA, Edwards KL. Hypertriglyceridemia as a cardiovascular risk factor. Am J Cardiol. 1998 Feb 26; 81(4A):7B-12B.

11. Mazzone T et al. "Current concepts and controversies in the pathogenesis, prevention and treatment of the macrovascular complications of diabetes. *J Lab. Clin Med;* 135:437-443.
12. Chaturvedi N, Fuller JH and Taskinen MR. "Differing associations of lipid and lipoprotein disturbances with the macrovascular and micro vascular complications of type 2 diabetes". *Diabetes Care.* 2001; 24(12): 2071-2076.
13. Joshua A, Becham B and Libby P. Diabetes and Atherosclerosis.2002. JAMA; 287: 2570-2581.
14. Sniderman AD, Lamarche B, Tilley J, Seccombe D and Frohlich J. Hypertriglyceremic HyperapoB in Type 2 Diabetes. 2002. Diabetes care; 25 : 579-582
15. Eriksson M et al. blood lipids in 75,048 type 2 diabetic patients: a population based survey from the Swedish National diabetes register. European journal of cardiovascular prevention and rehabilitation; 18(1): 97-105.
16. Kayode et al. Lipid profile of type 2 diabetic patients at a rural tertiary hospital in Nigeria. Journal of diabetes and Endocrinology.2010.Nov; Vol.1(4): 46-51
17. Sandhu HS, Koley S and Sandhu KS. A study of correlation between lipid profile and Waist to Hip ratios in patients with diabetes mellitus. Anthropologist.2008; 10(3): 215-218.
18. Samatha P, Venkateswarlu M and Siva Prabodh V. Lipid profile levels in Type 2 diabetes mellitus from the tribal population of Adilabad in Andhra Pradesh,India. Journal of clinical and Diagnostic Research.2012.May; Vol-6(4): 590-592

19. Khursheed et al. Lipid profile of patients with *Diabetes Mellitus* (A multidisciplinary study). World Applied Sciences Journal.2011; 12(9):1382-1384.
20. Yadav et al. assessment of prevalence of cardiovascular risk in diabetic patients attending OPD of Chitwan Medical College Teaching Hospital. International journal of pharmaceutical and Biological Archives.2012; 3(3): 561-564.
21. Kanani FH, Alam JM. Apolipoprotein B in Type 2 diabetics- a cross sectional study in a tertiary care setup. Journal of Pakistan Medical association.2010; vol 60:653-656.
22. Howard BV, Cowan LD, Go O, Welty TK, Robbins DC and Lee ET. Adverse effects of diabetes on multiple cardiovascular disease risk factors in women. The strong heart study. Diabetic care. 1998; 21(8): 1258-1265.
23. Nakhjavani M, Esteghamati AR, Esfahanian F and Heshmat AR. Dyslipidemia in type 2 diabetes mellitus: more atherogenic lipid profile in women. ActaMed.Iranica. 2006; 44(2):111-118
24. Kannel WB. "Lipids, Diabetes and Coronary Artery Disease: insights from the Framingham study". *Am. Heart. J. 1985; 110:* 1100-17.
25. Gilani SY, Bibi S, Ahmed N, Ali Shah SR. Gender difference of Dyslipidemia in type 2 diabetics. Journal of Ayub Med College, Abbottabad. 2010; 22(3): 146-148.
26. Aminul haq et al. Pattern of lipid profile in Type 2 diabetes mellitus patients. JPMI. 2006; VOL 20 NO 4: 366-369.

27. Songa RM, Siddhartha K, Sudhakar K. Lipid profile in type 2 diabetes mellitus with obesity. Bulletin of Pharmaceutical and Medical Sciences (BOPAMS).2013; Vol.1. Issue.2 : 132-138
28. Uttra KM et al. Lipid Profile of Patients with *Diabetes mellitus* (A Multidisciplinary Study). World Applied Sciences Journal. 2011; 12 (9): 1382-1384.
29. Kandula R, Shegokar V. A study of lipid profile in type 2 diabetes mellitus. MRIMS Journal of Health Sciences. 2013. Jan-June; Volume 1. Issue 1: 23-26
30. Udavat H, Goal RKG, Maheswary A. Coronary risk and dyslipidemia in type 2 diabetes mellitus. JAPI. 2001; 49: 970-73.
31. Bhu N, Coyal V, Mangal S, Goyal S. blood lipids amont 350 non-insulin dependent diabetic subjects. SDMH J. 1998; 12:167-74.
32. Hardas SP, Bhatia SG, Doiphade DN. Lipid profile in diabetes mellitus. JAPI. 1991; 39: 99.
33. Aryee Tagoe DN and Amo-Kodieh P. Type 2 diabetes mellitus influences lipid profile of diabetic patients. Annals of Biological Research, 2013; 4 (6):88-92.
34. Gordon L, Ragoobirsingh D, Morrison EY, Choo-Kang E, McGrowder D, Martorell E. Lipid Profile of Type 2 Diabetic and Hypertensive Patients in the Jamaican Population. Journal of Laboratory Physicians .2010. Jan-Jun; Vol-2 / Issue-1: 25-30
35. Jameel T, Faheem R and Ahmed SM. Estimation of lipid profile in type 2 diabetes mellitus. *International Journal of Basic and Applied Medical Sciences ISSN: 2277-2103 (Online) An Online International Journal Available*

at http://www.cibtech.org/jms.htm 2013. September- December; Vol. 3 (3):24-38.

36. Shaikh MA, Kumar S, Ghouri RA. Type 2 Diabetes Mellitus and Lipid Abnormalities. JLUMHS 2010. SEPTEMBER-DECEMBER; Vol: 09 No. 03: 145-147.
37. Assamang G, Schute H. The prospective Cardiovascular Minister (PROCAM) study; Prevalence of hyperlipidemia in persons with hypertension and/or diabetes mellitus and the relationship to coronary heart disease: American Heart Journal 1988; 116:1713
38. Smith S, Lall AM. A study on Lipid profile levels of Diabetics and Non diabetics among Naini Region of Allahabad, India. Turkish J Biochem. 2008: 33(4) : 138-41
39. Suryawanshi NP, Bhutey AK, Nagdeote AN, Jadhavand AA, Manoorkar GS. Study of lipid peroxide and lipid profile in Diabetes mellitus. Indian J Clin Biochem.2006; 21(1) : 126-130.
40. Gambhir JK, Kaur H, Gambhir DS, Prabhu KM. Lipoprotein (a) as an independent risk factor for coronary artery disease in patients below 40 years of age. Indian Heart J 2002; 52: 411-5.
41. Zargar AH, Wandroo FA, Wadhwa MB, Laway BA, Masoodi SR, Shah NA. Serum Lipid Profile in Non-insulin-dependent Diabetes Mellitus Associated with Obesity**.** *INT. J. DIAB. DEV. COUNTRIES (1995), VOL. 15: 9-15*
42. Sharma M , Rai M , Singh P. Study of Serum Lipid Profile in Type 2 Diabetes Mellitus in Male and Female: More Apparent in Postmenopausal and Premenopausal Female**.** Int J Biol Med Res. 2014; 5(3): 4391-4395.

43. U.K.Prospective Diabetes Study. Plasma Lipids and Lipoproteins at diagnosis of NIDDM by age and sex. *Diabetes care.* 1997; 20: 1683-1687.
44. Longo, Fauci, Kasper, Hauser, Jameson, Loscalzo. HARRISONS PRINCIPLES OF INTERNAL MEDICINE, 18TH EDITION, 2969.
45. Melmed S, Polonnsky KS, Larsen PR, .Kronenberg HM. Williams Textbook of ENDOCRINOLOGY, 12TH EDITION: 1371.
46. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes-estimates for the year 2000 and projections for 2030. Diabetes Care. 2004;27(3):1047-53.
47. Anjana RM, Ali MK, Pradeepa R, Deepa M, Datta M, Unnikrishnan R, Rema M, Mohan V. The need for obtaining accurate nationwide estimates of diabetes prevalence in India - rationale for a national study on diabetes. Indian J Med Res. 2011; 133:369-80.
48. Rao CR, Kamath VG, Shetty A, Kamath A. A cross-sectional analysis of obesity among a rural population in coastal southern Karnataka, India. Australas Med J. 2011;4(1):53-57
49. Mohan V, Deepa R. Obesity and abdominal obesity in Asian Indians. Indian J Med Res. 2006; 123(5):593-96.
50. Misra A, Khurana L. Obesity-related non-communicable diseases: South Asians vs White Caucasians. Int J Obes (Lond). 2011; 35(2):167-87.
51. Mohan V, Shah S, Saboo B. Current glycemic status and diabetes related complications among type 2 diabetes patients in India: data from the A1chieve study. JAPI (Suppl). 2013; 61:12-15.

52. Mohan V, Seshiah V, Sahay BK, Shah SN, Rao PV, Banerjee S. Current status of management of diabetes and glycaemic control in India: Preliminary results from the Diab Care India 2011 Study. Diabetes. 2012; 61:a645-a677.
53. Shanthi Rani CS et al. THE CHENNAI URBAN POPULATION STUDY (CUPS) - METHODOLOGICAL DETAILS – (CUPS PAPER NO. 1). INT. J. DIAB. DEV. COUNTRIES (1999), VOL. 19: 149-155
54. Deepa M. The Chennai Urban Rural Epidemiology Study (CURES) - Study Design And Methodology (Urban Component) (CURES - 1). JAPI . SEPTEMBER 2003; VOL 51: 863-870.
55. Mohan V, Gokulakrishnan K, Sandeep S, Srivastava BK,Ravikumar R, Deepa R. Intimal media thickness, glucose intolerance and metabolic syndrome in Asian Indians- the Chennai Urban Rural Epidemiology Study (CURES - 22).*Diabet Med* 2006; *23* : 845-50.
56. Premalatha G, Shanthirani S, Deepa R, Markovitz J, MohanV. Prevalence and risk factors of peripheral vascular disease in a selected South Indian population: the Chennai Urban Population Study. *Diabetes Care* 2000; *23* : 1295-1300.
57. Rema M et al. Prevalence of Diabetic Retinopathy in Urban India: The Chennai Urban Rural Epidemiology Study (CURES) Eye Study, I. *IOVS,* July 2005; Vol. 46, No. 7: 2328-33.
58. Ranjit UI, Rema M, Pradeepa R, Deepa M, Shanthirani CS, Deepa R. Prevalence and risk factors of diabetic nephropathy in an urban Indian population - The Chennai Urban Rural Epidemiology Study (CURES) (in press).

59. Botham KM and Mayes PA. HARPER'S ILLUSTRATED BIOCHEMISTRY. 28TH EDITION. Page no 121.

60. Howard BV and Howard WJ. Pathophysiology and Treatment of Lipid Disorders in Diabetes. Joslin's DIABETES MELLITUS. FOURTEENTH EDITION, Page no: 563-582.

61. Panarotto D, Remillard P, Bouffard L, Maheux P. Insulin resistance affects the regulation of lipoprotein lipase in the postprandial period and in an adipose-tissue specific manner. Eur Clin Invest 2002;32:84-92.

62. Krentz AJ. Lipoprotein abnormalities and their consequences for patients with type 2 diabetes. Diabetes Obes Metab 2003;5:S19-S27.

63. Garber AJ, Vinik AI, Crespin SR. Detection and management of lipid disorders in diabetic patients: a commentary for clinicians. Diabetes Care 1992; 15:1068-1074.

64. Howard BV, Knowler WC, Vasquez B, Kennedy AL, Pettit DJ, and Bennett PH. Plasma and lipoprotein cholesterol and triglyceride in the Pima. Indian population. Comparison of diabetics and nondiabetics. 1984. *Arteriosclerosis.* 4: 462-471.

65. Bowie A, Owens D, Collins P. Glycosylated low density lipoprotein is more sensitive to oxidation: implications for the diabetic patient? Atherosclerosis 1993; 10:63-67.

66. Matsui J, Onuma T, Tamasawa N, Suda T. Effects of advanced glycation endproducts on the generation of macrophage-mediated oxidized low-density lipoprotein. J Diabetes Complications 1997; 11:338-342.

67. Taskinen, MR, Beltz WF, Harper I, Fields RM, Schonfeld G, Grundy SM, and Howard BV. 1986. The effects of noninsulin-dependent diabetes mellitus on

VLDL triglyceride and VLDL apoB metabolism: studies before and after sulfonylurea therapy. *Diabetes.* 35:1268-77

68. Haffner, SM, Foster DM, Kushwaha RS, and Hazzard WR. 1984. Retarded chylomicron apolipoprotein-B catabolism in type 2 (non-insulin-dependent) diabetic subjects with lipaemia. *Diabetologia.* 26: 349-354.
69. Howard BV, Abbott WF, Beltz WF, Harper I, Fields RM, Grundy SM and Taskinen RM. 1986. The effect of non-insulin-dependent diabetes on very low density lipoprotein and low density lipoprotein metabolism in man. *Metabolism.* In press.
70. Schonfeld, G, Birge C, Miller JP, Kessler G, and Santiago J. 1974. Apolipoprotein B levels and altered lipoprotein composition in diabetes. *Diabetes.* 23**:** 827-834.
71. Nesto, Richard.W. et al. "Heart Disease in Diabetes" Chp. 45 in International Edition Joslin's Diabetes Mellitus, Kahn, Ronald C and Gordon C, Ed W, Philadelphia:Waverly Company, 1994,837 pp.
72. Crall, FV and Roberts WC. "The extramural and intramural coronary arteries in juvenile diabetes mellitus. Analysis of nine necropsy patients aged 19 to 38 years with onset of diabetes before age 15 years". *Am* .J. *Med* 1978. 64:221-230
73. Stout LC and Thorpe LW. 1980: "Histology of normal aortas in nonhuman primates with emphasis on diffuse intimal thickening". *Atherosclerosis,* 35:165-180
74. Dodson PM and Horton RC. 1987: "The nature of atheroma". *Proc. Nutr. Soc,* 46: 331-336.

75. Steinberg D et al 1989: "Beyond cholesterol: modification of low density lipoprotein that increase its atherogenicity". *N. Engl. J Med.* 320: 915-924.
76. Howard BY et al 1983: "VLDL triglyceride metabolism in NIDDM. Relationship of plasma insulin and free fatty acids". *Diabetes,* 32: 271- 276.
77. *National Cholesterol Education Program:*ATP III Guidelines at a glance. Quick Desk Reference. U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES Public Health Service. National Institutes of Health. NIH Publication No. 01-3305.May 2001

ANNEXURE I

ETHICAL CLEARANCE

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-2012 at 3-30 pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "Study of lipid profile in patients with type 2 diabetes Mellitus"

—x—x—

Name of P.G. student Dr. Nagabushan Hesarur.
Medicine

Name of Guide/Co-investigator Dr R.C. Bidri
Professor of Medicine

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

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

ANNEXURE II

INFORMED CONSENT FORM

I ……. exercising free power of choice hereby give my written consent to be included as a subject in the study **"STUDY OF LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS''** conducted by **Dr NAGABUSHAN HESARUR** and undergo the necessary investigations required for this study and I fully consent for the same.

I am over 18 yrs of age and I have been explained to my satisfaction by the attending Physician, in the language I understand, about the purpose of the study .I have also understood that the investigator will maintain confidentiality regarding my identity.

SIGNATURE OF DOCTOR **SIGNATURE OF PATIENT/RELATIVE**

NAME OF THE DOCTOR **NAME OF PATIENT/RELATIVE**

Dr NAGABUSHAN HESARUR **RELATIONSHIP (IF RELATIVE)**

DATE:

ANNEXURE III

PROFORMA

BLDE'S SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR

"STUDY OF LIPID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS''

Name:

Age:

Sex:

Occupation:

Address:

Chief complaints:

Details of diabetes mellitus:

Age of onset:

Duration:

Complications:

Treatment history:

a) Only diet control

b) Oral hypoglycemic agents

c) Insulin Dose _____

Control of diabetes: Good/Fair/Controlled.

Past history: Any h/o HTN, MI, Stroke

Personal history:

Tobacco chewing: Yes/No

Smoking beedi : Yes/No

Smoking cigarette : Yes/No

Alcohol : Yes/No

Diet : Veg/Mixed

GENERAL PHYSICAL EXAMINATION:

Height:

Weight:

BMI:

Waist:

Hip:

Waist to Hip ratio:

Vitals

Pulse:

Peripheral pulses:

Blood pressure

Supine:

Standing:

Respiratory rate:

SYSTEMIC EXAMINATION

Respiratory system:

Cardiovascular system:

Per abdomen:

Central nervous system:

LABORATORY FINDINGS

Hb%: Total Count:

Differential count: ESR:

Urine Albumin

Sugar:

Microscopy:

Fasting blood sugar:

Post prandial blood sugar:

HbA1c:

Fasting Lipid profile: Cholesterol: HDL-C:

LDL-C: VLDL-C:

Triglycerides:

ANNEXURE IV

KEY TO MASTER CHART

FBS	–	Fasting blood sugar
PPBS	–	Post prandial blood sugar
HbA1C	–	Glycosylated haemoglobin
TC	–	Total cholesterol
TG	–	Triglyceride
LDL	–	Low density lipoprotein
HDL	–	High density lipoprotein
VLDL	–	Very low density lipoprotein
CVA	–	Cerebrovascular accident
IHD	–	Ischemic heart disease
RET	–	Retinopathy
NEP	–	Nephropathy
PVD	–	Peripheral vascular disease
NEU	-	Neuropathy

ANNEXURE IV

MASTER CHART

CASES

SI NO	NAME	AGE	SEX	DURATION	TREATMENT	FBS	PPBS	HbA1c	TC	TG	LDL	HDL	VLDL	TC/HDL	LDL/HDL	CVA	IHD	RET	NEP	PVD	NEU
1	JUMANNA	60	M	10YRS	OHA	260	253	11.2	191	50	141	40	10	4.77	3.52	N	Y	N	N	N	Y
2	MAHADEV	45	M	< 1YR	OHA	244	388	8.3	150	269	64.2	32	53.8	4.68	2	N	N	N	N	N	N
3	HANUMANT	58	M	5YRS	OHA	163	257	7.5	127	108	75.4	30	21.6	4.23	2.51	N	N	N	N	N	N
4	SAYED	65	M	17YRS	OHA	328	276	7.8	209	75	160	34	15	6.14	4.7	N	N	Y	Y	N	Y
5	SANJU	48	M	< 1YR	OHA	159	180	6.9	177	178	106.4	35	35.6	5.05	3.02	N	N	N	N	N	N
6	CHIDAMBAR	62	M	12YRS	OHA	75	151	8.4	148	68	86	48	13.6	3.08	1.79	N	Y	Y	N	N	Y
7	BHIMANGOUDA	52	M	< 1YR	OHA	71	158	6.3	155	162	91.6	31	32.1	5	2.95	N	N	N	N	N	N
8	HANUMANTH	56	M	5YRS	INSULIN	122	137	8.5	158	84	108.2	33	16.8	4.78	3.27	Y	Y	N	N	N	N
9	ABDUL BAGWAN	43	M	8YRS	OHA	331	260	8.2	231	219	232	84	43	2.75	2.76	N	N	N	N	N	Y
10	DADASAHEB	59	M	3YRS	OHA	322	239	13	284	120	224	36	24	7.88	6.22	N	N	Y	N	N	N
11	AMEER SHEIK	59	M	2YRS	OHA	153	256	8.8	176	500	220	31	33	5.67	7.09	N	N	N	Y	N	N
12	ABDUL MULLA	68	M	10YRS	OHA	195	234	10.6	269	231	76	40	30	6.72	1.9	N	N	Y	N	N	Y
13	BASAVARAJ	50	M	5YRS	OHA	224	241	10.1	145	69	103.2	28	13.8	5.17	3.67	N	N	Y	Y	N	N
14	RUDRAPPA	63	M	< 1YR	OHA	162	210	6.3	192	189	129.2	25	37.8	7.68	5.16	N	N	N	N	N	N
15	DASTGIR SAB	60	M	5YRS	OHA	108	140	6.2	199	115	112	54	23	3.68	2.07	N	Y	N	N	N	Y
16	SANGAMESH	42	M	< 1YR	OHA	182	198	6.8	187	330	90	31	66	6.03	2.9	N	Y	N	Y	N	N
17	MRITYUNJAY	48	M	8YRS	OHA	222	290	6.8	106	77	63.6	50	15.4	2.12	1.27	N	N	N	N	N	Y
18	SURYAKANT	57	M	< 1YR	OHA	134	210	7	167	83	115.4	35	16.6	4.77	3.28	N	N	N	N	N	N
19	SIDDRAMA	48	M	1YR	DIET	109	149	5.3	162	84	104.2	51	16.8	3.17	2.03	N	N	N	N	N	N
20	BHIMAPPA	75	M	10YRS	OHA	347	364	10.1	146	82	94.6	35	16.4	4.17	2.7	Y	N	N	Y	N	Y
21	MAHADEV M	53	M	15YRS	OHA+INS	65	274	7.2	139	97	72.6	47	19.4	2.95	1.54	N	N	Y	Y	N	Y
22	GOPAL	60	M	5YRS	OHA	209	250	10.3	138	73	95.4	28	14.6	4.92	3.4	N	N	N	Y	N	N
23	SHARNAPPA	58	M	< 1YR	INSULIN	268	280	10.3	199	417	87.6	28	83.4	7.1	3.12	Y	N	N	N	N	N
24	SIDDAPPA	65	M	< 1YR	INSULIN	377	322	8.3	225	201	120	32	28	7.03	3.75	N	N	N	N	N	N
25	YAMANAPPA	40	M	5YRS	OHA	230	236	7.8	184	185	117	30	37	6.13	3.9	N	Y	N	N	N	N
26	HUCCHANGOUDA	56	M	< 1YR	OHA	186	298	8.6	191	172	129.6	27	34.4	7.07	4.81	N	N	N	N	N	N
27	BASAVRAJ GOUDA	58	M	3YRS	OHA	108	130	6.9	159	189	87.2	28	37.8	4.2	3.11	N	N	N	N	N	N
28	SIDDAPPA M	64	M	4YRS	OHA	100	136	6.3	155	181	90.8	28	36.2	5.53	3.24	N	N	N	Y	N	N
29	HANNU CHAVAN	65	M	4YRS	INSULIN	281	327	12.9	260	206	90	29	38	8.96	3.1	N	N	N	N	N	N
30	SADANAND	65	M	5YRS	INSULIN	282	443	9.4	112	101	65.8	26	20	4.3	2.53	N	N	N	Y	N	N
31	PARSAPPA	60	M	< 1YR	INSULIN	196	215	8	185	152	115.6	39	30.4	4.74	2.97	N	N	Y	N	N	N
32	SURESH	45	M	4YRS	OHA	174	180	7.5	110	102	65.6	24	20.4	4.58	2.73	N	Y	Y	N	N	Y
33	RAJU KHARAT	50	M	< 1YR	INSULIN	221	285	10.3	240	230	92	40	25	6	2.3	N	Y	Y	Y	N	N
34	CHANDRASHEKAR	67	M	4YRS	OHA	261	308	8.2	137	164	76.2	28	32.8	4.89	2.72	Y	N	Y	Y	Y	Y

35	NARAYAN	58	M	1YR	OHA	138	200	7.4	150	140	94	28	28	5.35	3.35	N	Y	N	N	N	N
36	BHIMANNA	62	M	2YRS	OHA	201	230	7.7	313	191	115	53	38	5.9	2.16	N	Y	N	Y	N	N
37	HANMANTHAPPA	74	M	3YRS	OHA	368	329	6.6	157	82	110.6	30	16.4	5.23	3.7	N	N	N	N	N	N
38	BIRAPPA	65	M	5YRS	OHA	92	86	5.9	135	97	82.6	33	19.4	4.09	2.5	N	N	N	Y	N	Y
39	DEVENDRAPPA	45	M	5YRS	OHA	265	281	6.6	191	208	107.4	42	41.6	4.54	2.54	N	N	N	N	N	Y
40	SN BIRADAR	60	M	5YRS	OHA	212	276	7.4	165	212	51.8	52	10.4	3.17	0.99	N	N	Y	N	N	Y
41	UMARSAB	60	M	2YRS	INSULIN	216	262	8.6	235	228	136	57	45	4.12	2.38	N	N	Y	Y	N	N
42	SANGANGOUDA	60	M	5YRS	OHA	138	178	7.2	157	73	111.4	31	14.6	5.06	3.58	N	N	N	N	N	N
43	ABDUR REHMAN	57	M	< 1YR	DIET	131	123	6.9	178	126	116.8	36	25.2	4.94	3.25	N	Y	N	N	N	N
44	BASAVARAJ T	47	M	< 1YR	OHA+INS	298	459	10.3	107	65	72	22	13	4.86	3.27	N	N	N	Y	Y	N
45	LALASAHEB	65	M	5YRS	OHA	99	108	8	151	141	95.8	27	28.2	5.59	3.54	Y	N	N	N	N	N
46	IMAMUDDIN	60	M	8YRS	INSULIN	112	134	8	110	109	51.2	37	21.8	2.97	1.38	N	Y	Y	Y	N	Y
47	BASANGOUDA M	60	M	5YRS	OHA	172	238	7.6	153	128	25	63	58	2.42	0.39	N	N	N	Y	N	N
48	PRAKASH M	60	M	4YRS	OHA	128	146	8.8	136	123	71.4	40	24.6	3.4	1.78	N	N	N	Y	N	N
49	DEVENDRAPPA M	36	M	4YRS	INSULIN	298	459	12.8	288	138	114.4	47	26.6	6.12	2.42	N	N	N	N	N	N
50	RAMESH N	42	M	< 1YR	INSULIN	219	254	7.7	163	115	108	37	23	4.4	2.91	N	Y	N	N	N	N
51	BASAPPA KOLI	60	M	5YRS	OHA	218	260	8.2	236	112	155.6	58	22.4	4.06		N	Y	N	N	N	Y
52	ASHOK PUJAR	52	M	4YRS	OHA	188	176	8.3	199	225	118	45	23	4.42	2.62	N	Y	N	N	N	N
53	RAMU RATHOD	43	M	< 1YR	INSULIN	184	242	7	218	149	147.2	41	29.8	5.31	3.58	N	Y	N	N	N	N
54	SHIVAJI MASUTI	42	M	6YRS	OHA	224	390	8.8	122	205	51	30	41	4.06	1.7	N	Y	N	N	N	Y
55	HANAMANTRAYA	45	M	< 1YR	DIET	313	345	6.9	163	147	82	62	29	2.62	1.32	N	N	N	N	N	N
56	BASAVARAJ	54	M	6YRS	OHA	104	118	7	147	207	77.6	28	41.4	5.25	2.77	N	N	N	N	N	Y
57	SURESH SANTH	48	M	8YRS	OHA	176	226	9.9	167	188	88.4	41	37.6	4.07	2.15	N	N	Y	Y	N	Y
58	BASAGOND	63	M	10YRS	INSULIN	95	120	6.4	180	150	95	55	30	3.27	1.72	Y	N	Y	Y	N	Y
59	AYUB UPPARGAR	57	M	5YRS	INSULIN	112	170	10.7	226	159	160.2	34	31.8	6.64	4.7	N	Y	N	N	N	N
60	DUNDAPPA	60	M	< 1YR	INSULIN	268	332	9	248	210	178	30	42	8.26	5.93	Y	N	Y	N	N	N
61	SHREEKANT	55	M	2YRS	OHA	248	268	9.1	209	59	158.2	39	11.8	5.35	4.05	N	N	N	Y	N	N
62	AMMANNA	68	M	4YRS	OHA	108	138	6.5	222	210	146	34	42	5.28	4.29	N	N	N	N	N	N
63	YAMANAPPA A	60	M	5YRS	INSULIN	158	200	6.8	125	106	83.8	20	21.2	6.25	4.19	N	N	Y	N	N	N
64	SIDDANNA C	47	M	10YRS	OHA	303	350	10.8	192	117	128.6	40	23.4	4.8	3.22	N	N	N	Y	N	Y
65	PARASHURAM	35	M	< 1YR	OHA	140	209	7	245	258	158.4	35	51.6	7	4.51	N	N	N	N	N	N
66	TUKARAM	65	M	1YRS	OHA	132	180	6.9	110	126	54.8	30	25.2	3.66	1.82	N	N	N	Y	N	N
67	SALIM	39	M	10YRS	OHA	222	496	12.2	167	500	41	26	100	6.42	1.57	N	N	Y	Y	N	N
68	LAKSHMAN	65	M	4YRS	OHA	98	138	7.1	173	155	109	33	31	5.24	3.3	Y	N	N	Y	N	N
69	BAGAPPA	72	M	4YRS	OHA	380	423	10.8	280	210	178	60	42	4.66	2.96	N	N	N	Y	N	N
70	GOVIND MATH	50	M	< 1YR	OHA	120	228	7.2	180	169	122.2	24	33.8	7.5	5.08	N	Y	N	N	N	N
71	MALLIKARJUN	51	M	4YRS	OHA	180	228	10.8	180	73	120.4	45	14.5	4	2.66	N	Y	N	Y	N	N
72	BASAVARAJ A	56	M	< 1YR	INSULIN	214	225	8.6	113	154	85	23	30.8	4.91	3.69	N	N	N	N	Y	N
73	SIDDANAGOUDA	36	M	< 1YR	OHA	112	148	6.6	252	353	151.4	30	70.6	8.4	5.03	N	Y	N	N	N	N
74	BASANNA H	55	M	5YRS	INSULIN	380	233	9.7	176	121	103.8	48	24.2	3.66	2.16	N	N	Y	Y	N	Y
75	SANGANGOUDA P	65	M	10YRS	OHA	135	243	8.8	155	118	97.4	34	23.6	4.55	2.86	N	N	Y	Y	N	Y

76	HUSSEINSAB	70	M	< 1YR	INSULIN	233	285	11.9	215	210	143	30	42	7.16	4.76	Y	N	Y	N	N	N
77	NINGAPPA	39	M	< 1YR	OHA	208	279	10.2	262	499	113.2	49	99.8	5.34	2.3	N	N	N	N	N	N
78	ADIVEPPA	61	M	10YRS	INSULIN	163	212	7.3	179	137	78	69	27	2.59	1.13	N	N	Y	Y	N	Y
79	SANGAPPA	60	M	1YR	INSULIN	275	320	10.5	127	83	71.4	39	16.6	1.84	1.83	Y	N	Y	N	N	N
80	KASHIBAI	75	F	< 1YR	INSULIN	154	162	11	302	180	211	55	36	5.49	3.83	Y	N	Y	N	N	N
81	LAKKAWWA	65	F	< 1YR	INSULIN	171	168	10.5	251	86	194.8	34	17.2	7.38	5.73	Y	N	Y	N	N	N
82	NEELAWWA	75	F	20YRS	INSULIN	138	376	7.3	205	173	129.4	41	34.6	5	3.14	N	N	Y	Y	N	Y
83	SHAMSHAD	40	F	< 1YR	OHA	112	188	7.9	129	213	58.4	28	42.6	4.6	2.08	N	N	N	N	N	N
84	PARATEWWA	62	F	4YRS	OHA	76	156	10.5	244	202	166.6	37	40.4	6.59	4.51	N	N	Y	Y	N	N
85	GURUSHANTAWWA	45	F	2YRS	OHA	250	260	8.7	198	132	140.6	31	26.4	6.38	4.54	Y	N	Y	Y	N	N
86	SHANKRAMMA	50	F	< 1YR	INSULIN	310	392	8.4	215	192	145	31	38.4	6.93	4.67	N	N	N	N	Y	N
87	ADAMMA	65	F	1YR	OHA	190	262	6.3	352	420	21	57	84	6.17	0.36	N	Y	N	N	N	N
88	KALAVATHI	56	F	10YRS	INSULIN	318	386	11	740	1000	250	70	55	10.57	3.57	N	N	Y	N	N	Y
89	CHAANNAMMA	74	F	15YRS	OHA	366	380	9.6	166	217	94.6	28	43.4	5.92	3.37	Y	N	Y	Y	N	N
90	SHREEDEVI	46	F	1YR	OHA	171	219	7.1	257	190	181	38	38	6.76	4.76	N	Y	N	N	N	N
91	KALAMMA	55	F	3YRS	OHA	221	280	6.8	163	412	48.6	32	82.4	5.09	1.51	N	Y	N	N	N	N
92	MEENAKSHI PATIL	53	F	10YRS	OHA	176	329	8.6	169	186	97.8	34	37.2	4.97	2.87	N	N	Y	N	N	N
93	TULUSABAI	65	F	9YRS	OHA	148	193	8.3	200	192	118.6	43	38.4	4.65	2.76	N	N	Y	Y	N	N
94	UMABAI	70	F	< 1YR	INSULIN	105	226	10.3	304	94	223.2	62	18.8	4.9	3.59	Y	N	N	N	N	N
95	MAHADEVI	50	F	4YRS	INSULIN	275	334	10.5	152	137	85	48	27	3.16	1.77	N	N	N	N	N	N
96	VASANTI	56	F	6YRS	OHA+INS	99	350	8.8	185	525	134	28	50.4	6.6	4.78	N	N	Y	Y	N	N
97	SHANTA	42	F	< 1YR	OHA	109	221	7.4	390	246	290.8	50	42.2	7.8	5.82	N	Y	N	N	N	N
98	KASTURIBAI	50	F	2YRS	OHA	274	378	9.9	187	208	41.6	28	117.4	6.67	1.48	N	N	N	N	N	N
99	BASAMMA	62	F	5YRS	OHA	107	227	7.2	203	113	137.4	43	22.6	4.72	3.18	N	N	Y	N	N	N
100	CHINNAKKA	60	F	2YRS	OHA	430	288	9	215	80	149	50	16	4.3	2.98	N	N	N	N	N	N
101	SHANTABAI	50	F	2YRS	OHA	286	312	7.8	171	108	119.4	30	21.6	5.7	3.96	N	Y	N	N	N	N
102	RAHMATBI	62	F	2YRS	DIET	125	168	6.7	187	126	112.8	49	25.2	3.81	2.3	N	N	N	N	N	N
103	YAMANAVVA	70	F	2YRS	OHA	152	172	7	208	122	128.6	55	24.4	3.78	2.34	N	Y	N	N	N	N
104	GOURABAI	60	F	6YRS	OHA+INS	221	264	11	194	101	142.8	31	20.2	6.25	4.61	N	Y	Y	N	Y	Y
105	SUMITRABAI	55	F	< 1YR	OHA	134	180	7.2	253	187	178	37	37.4	6.83	4.81	N	Y	N	N	N	N
106	KASHIBAI D	55	F	6YRS	OHA	221	200	9.9	132	189	54	30	48	4.4	1.8	N	Y	Y	N	N	Y
107	SHANKREWWA	60	F	< 1YR	INSULIN	212	380	10.8	254	104	172	61	20.8	4.16	2.81	Y	N	N	N	N	N
108	GUNAVANTAVVA	60	F	< 1YR	INSULIN	317	228	8.1	278	500	152	26	100	10.69	5.84	N	N	N	N	N	N
109	BOURAMMA	43	F	< 1YR	DIET	149	202	7.6	324	321	183	88	64	3.68	2.07	N	N	N	N	N	N
110	SIDDAMMA	70	F	1YR	DIET	106	138	6.6	179	201	103.2	35	40	5.11	2.94	N	N	N	N	N	N
111	RATNABAI	53	F	8YRS	OHA	68	143	8.6	193	123	135	33	24.6	5.84	4.09	N	N	Y	N	N	Y
112	SUSHILABAI	65	F	5YRS	OHA	186	253	10.8	216	211	141	32	42	6.75	4.4	N	N	N	N	N	Y
113	GURULINGAWWA	55	F	< 1YR	OHA+INS	232	208	7.6	351	120	291	35	24	10.02	8.31	N	Y	N	N	N	N
114	BHARATHI	35	F	1YR	OHA	97	130	7.7	166	100	119	27	20	6.14	4.4	N	N	N	N	N	N
115	LEELA PUROHIT	68	F	< 1YR	DIET	153	230	7	256	160	181	43	32	5.95	4.2	N	N	N	N	N	N
116	KAMALABAI	54	F	3YRS	OHA	138	167	7.1	187	145	112	54	12	3.46	2.07	N	N	N	N	N	N

117	VIJAYALAXMI	38	F	< 1YR	INSULIN	200	254	10.6	135	117	84.6	27	23.4	5	3.13	N	N	N	N	N	N
118	BASAMMA H	65	F	15YRS	OHA	286	423	10	154	602	120.4	23	10.6	6.69	5.21	N	Y	Y	Y	N	N
119	NEELAMMA	55	F	< 1YR	INSULIN	181	250	10.1	150	291	204.8	28	58.2	5.35	7.32	N	N	N	N	N	N
120	KASHIBAI P	70	F	< 1YR	DIET	100	125	6.6	175	78	109.4	50	15.6	3.5	2.18	N	N	N	N	N	N
121	MAMTAZ BEGUM	40	F	< 1YR	OHA	97	262	8.7	189	200	109	40	40	4.72	2.72	N	N	N	N	N	N
122	AMEERBEE	65	F	8YRS	OHA	181	257	7.1	213	179	109	52	35	4.09	2.09	N	N	Y	Y	Y	Y
123	RUDRAMMA	68	F	25YRS	INSULIN	180	223	7.8	202	249	112.2	40	49.8	5.05	2.8	N	N	Y	Y	Y	N
124	GANGUBAI	55	F	6YRS	OHA	225	265	9.2	170	226	86.8	38	45.2	4.47	2.28	N	Y	Y	N	N	N
125	LAXMIBAI	75	F	10YRS	OHA	219	248	9.5	162.6	150	108	24.6	30.6	6.62	4.39	N	Y	Y	N	N	N

CONTROLS

SI NO	NAME	AGE	SEX	FBS	PPBS	HbA1c	TC	TG	LDL	HDL	VLDL	TC/HDL	LDL/HDL
1	HANUMANTH	65	M	76	85	6.8	115	50	79	26	10	4.42	3.03
2	BHIMGOND	65	M	93	123	5.9	176	91	107.8	50	18.2	3.52	2.16
3	KASHIM M	45	M	89	99	6	168	185	96	35	37	4.8	2.74
4	MALLAPPA	45	M	114	138	6	155	56	95.8	48	11.2	3.22	2
5	RAJESAB	65	M	93	102	5.9	127	78	74.4	37	15.6	3.96	2
6	RAJU P	45	M	102	128	5	192	142	131	32	28.4	6	4.09
7	RAMLING	65	M	112	148	5.6	206	92	120	63	18.4	3.26	1.9
8	NAZEER AHMED	60	M	113	128	5.6	138	350	41.8	25	71.2	5.52	1.68
9	RAGHVENDRA	60	M	100	120	5	239	141	175	35	28.2	6.82	5
10	SHIVALINGAPPA	55	M	110	130	5.3	142	165	77	32	33	4.43	2.4
11	BASAVARAJ	68	M	83	100	5.6	227	119	97	83	23	2.73	1.16
12	SIDDAPPA	55	M	80	99	4.9	154	142	100	23	30.4	6.69	4.34
13	GIRIMALLA	65	M	112	139	5.2	157	81	100	40	16.2	3.92	2.5
14	GURUSIDDAYA	60	M	112	134	4.7	124	105	72	31	21	4	2.32
15	VITTAL JADHAV	65	M	73	85	5.7	131	55	76	44	11	2.97	1.72
16	SIDDANGOUDA	38	M	85	97	5	207	137	112	59	33	3.5	1.89
17	BASAPPA	75	M	72	85	4.8	139	59	76.2	51	14	2.72	1.72
18	RAMAKANTA	70	M	112	140	5.4	142	186	73.8	31	37.2	4.58	2.38
19	SIDDANNA J	65	M	138	112	6.1	158	98	94.4	44	13.6	3.59	2.13
20	AYYANGOUDA	59	M	88	120	5.1	141	93	85.4	37	18.6	3.81	2.29
21	BOJU LAMANI	65	M	95	110	5.8	120	95	73	28	19	4.28	2.6
22	BASAPPA	62	M	88	100	5.7	224	86	150	52	17.2	4.3	2.88
23	BHIMAPPA	55	M	78	110	4.9	151	94	92	40	18.8	3.77	2.3
24	YANKAPPA	65	M	88	132	5.3	147	87	92.6	47	17.4	3.12	1.97
25	CHANDRASHEKAR	70	M	96	125	5.6	143	90	87	48	18	2.97	1.81

26	SANGAYYA	65	M	80	120	5.7	138	91	91.8	28	18.2	4.92	3.28
27	BASAVARAJ B	30	M	100	143	4.6	130	85	89	24	17	5.41	3.7
28	KALLAPPA	50	M	94	106	4.5	136	61	77.8	46	12.2	2.95	1.69
29	SHARNAGOUDA	65	M	90	122	5.1	150	85	76	52	22	2.88	1.46
30	MALLAPPA B	45	M	116	145	6.1	169	154	58	62	30	2.72	0.93
31	SURESH SINDHE	47	M	108	140	6	235	206	129.8	64	41	3.67	2.03
32	GURAPPA	75	M	80	96	5	149	109	87	50	21	2.98	1.74
33	NIJEPPA	70	M	109	132	6	182	132	89	51	26	3.56	1.74
34	NAGAPPA	65	M	103	118	4.9	155	80	89	50	16	3.1	1.78
35	MADIVALAPPA	65	M	90	120	6.1	169	144	104.2	36	28	4.69	2.88
36	GUNDAPPA	68	M	78	99	5.6	137	61	69.8	55	12	2.49	1.27
37	SHIVAKANT	58	M	112	130	5.9	189	147	110.6	49	29.4	3.85	2.26
38	SHIVAPPA	60	M	90	120	5.6	145	80	90	40	16	3.62	2.25
39	ZAMPU	75	M	96	110	5.1	116	90	75	43	18	2.69	1.74
40	MD ISMILLA	57	M	81	96	5.7	199	198	134	25	39.6	7.96	5.36
41	SHIVAJI	50	M	108	130	5.9	139	75	87	37	15	3.75	2.35
42	ARAVIND	40	M	111	120	6	203	108.8	112	46	48	4.41	2.43
43	RAYAGOND	70	M	93	118	5.7	166	132	103.6	36	26.4	4.61	2.88
44	MAHADEV	50	M	111	138	5.3	198	137	136.6	34	27.4	5.82	4.02
45	ANNAPPA	39	M	99	110	4.8	206	139	120	43	27	4.79	2.79
46	GANGAPPA	60	M	105	128	5.9	191	232	108.6	36	46.4	5.3	3.02
47	MALLAPPA	75	M	76	93	5.8	198	149	98	53	29	3.73	1.84
48	LAKKAPPA	35	M	120	148	4.9	203	108.8	112	46	48	4.41	2.43
49	KHAJASAB	38	M	96	118	5.1	230	128	170.4	44	25.6	5.22	3.86
50	VITTAL GOVIND	68	M	96	110	4.9	221	137	159.6	34	27.4	6.5	4.7
51	MURIGEPPA	50	M	120	130	6.5	205	148	61	58	33	3.53	1.05
52	UMESH	38	M	123	140	5.8	205	201	127.8	37	40.3	5.54	3.45
53	BASAPPA D	65	M	96	110	5.1	159	96	93.8	46	19.2	3.45	2.04

54	BASAVARAJ	52	M	81	96	5.7	82	126	110.6	36	16.4	2.27	3.08
55	YAMANAPPA	45	M	96	128	5.9	146	160	83	29	33.8	5.03	2.86
56	APPASAHEB	58	M	80	120	5.1	100	84	53	40	16.8	2.5	1.32
57	SHARNAPPA	38	M	122	154	6	143	69	91	43	13.8	3.32	2.11
58	SHIVREDDY	30	M	80	120	4.7	227	104	118	85	20	2.67	1.38
59	KAMU RATHOD	70	M	76	93	5.8	133	56	77.8	44	11	3.02	1.77
60	BHIMARAYA	50	M	96	110	4.9	110	55	61	48	11	2.29	1.27
61	KALLAPPA K	46	M	88	120	5.1	153	127	83.6	44	25	3.47	1.9
62	SHIVANGOUDA	70	M	80	120	5.1	176	93	94	41	18	4.29	2.29
63	MALKAPPA C	56	M	80	110	5.9	171	139	100	48	28	3.56	2.08
64	ASHOK	39	M	78	102	6	143	88	94	41	18	3.48	2.29
65	SHIVAPPA G	55	M	97	112	5.2	208	186	106	55	37	3.78	1.92
66	SHEVU DEVU	60	M	82	106	4.9	148	157	97	51	31	2.9	1.9
67	RAMU RATHOD	65	M	113	145	6	138	65	97	38	13	3.63	2.55
68	SHIVANANDA	48	M	88	120	5.1	137	108	75.4	40	21.6	3.42	1.87
69	SAHEBLAL	65	M	110	138	5.9	227	127	150.6	51	25	4.45	2.96
70	ASHOK MAITY	52	M	120	140	5.9	209	129	133	50	25.8	4.18	2.66
71	RANGAPPA	75	M	92	140	6.1	250	179.4	118	47	23.6	5.31	2.51
72	THAKURSINGH	65	M	91	124	6.1	203	144	97.2	69	36	2.94	1.4
73	NINGONDAPPA	46	M	112	148	6.1	160	212	87.2	30	42	5.33	2.9
74	MALASIDDA	40	M	97	112	5.4	111	59	55	44	11.8	2.52	1.25
75	CHANDU	60	M	112	145	5.4	218	88	147.4	53	17	4.11	2.77
76	KEERAPPA	70	M	123	158	6.1	191	124	134	32	24.8	5.96	4.18
77	IRAPPA	62	M	96	120	4.9	150	70	90	54	14	2.77	1.66
78	SIDDANGOUDA	72	M	90	110	5.3	202	148	125	48	29.6	4.2	2.6
79	SHANKARGOUDA	62	M	91	130	5.1	213	139	99	50	27	4.26	1.98
80	KALLAPPA	70	M	90	138	5.6	137	71	87.8	35	14.2	3.91	2.51
81	NINGAPPA	65	M	92	130	5.6	137	61	86	40	12	3.42	2.15

82	RAHUTAPPA	52	M	120	160	7	98	76	111	42	18.6	2.33	2.64
83	TUKARAM	51	M	99	108	5.1	164	65	93	56	13	2.92	1.66
84	KANTAPPA	54	M	94	120	5.6	172	71	116.8	41	14.2	4.19	2.85
85	SADASHIV	50	M	92	130	5.8	174	120	112	48	24	3.62	2.33
86	SHIVAMMA	65	F	118	134	6	70	70	98	45	14	1.55	2.17
87	GANGABAI	58	F	116	150	5.4	168	212	95	30	42.4	5.6	3.16
88	GANGABAI Y	60	F	94	82	5.6	240	199	151	49	39.8	4.89	3.08
89	CHANDRAWWA	55	F	97	82	4.9	246	173	165.4	46	34.6	5.34	3.58
90	BHIMABHAI	70	F	106	133	5	201	264	85	58	52	3.46	1.46
91	KAMALABAI	40	F	86	103	5.1	183	100	85	42	18	4.35	2.02
92	BHARATI	36	F	106	130	5.1	174	120	95	40	24	4.35	2.37
93	POORNIMA	53	F	99	123	5.3	138	67	89	39	13	3.53	2.28
94	KAMABAI R	56	F	87	98	5	148	109	97	29	21.8	5.1	3.34
95	GOMALABAI	50	F	95	100	4.9	250	94	192.2	39	18.8	6.41	4.92
96	SHANTABAI	65	F	71	99	4.9	157	56	87	58	11	2.7	1.5
97	LAXMIBAI	45	F	103	110	5.2	145	220	64	37	44	3.91	1.72
98	ASHA T	47	F	90	110	5.6	192	133	93	42	34	4.57	2.21
99	KASHIBAI	66	F	98	61	6	196	115	96	59	19	3.32	1.62
100	NEELAWWA	75	F	80	99	5.2	168	78	100	44	15.4	3.81	2.27
101	LATHA	43	F	98	108	5.9	177	98	106	31	32	5.7	3.41
102	SAVITRI	46	F	90	110	5.4	116	93	65	32	65	3.62	2.03
103	SAVITRI T	44	F	112	120	5.6	182	160	109	41	32	4.43	2.65
104	BHAGIRATHI	68	F	100	121	5.7	155	112	88	44	22	3.52	2
105	GOURAWWA	55	F	96	120	6	227	98	147	60	19	3.78	2.45
106	RATNABAI	58	F	99	110	5.6	220	137	76	62	27	3.54	1.22
107	MAHADEVI	66	F	111	130	5.9	169	94	118	32	19	5.28	3.68
108	KAVITA	32	F	80	96	5.6	158	112	79	47	22	3.36	1.68
109	KAMALABAI P	50	F	86	102	5.1	209	85	98	50	17	4.18	1.96

110	BANGAREWWA	50	F	80	96	5.9	214	138	100	75	27	2.85	1.33
111	NEELAWWA B	50	F	83	100	5.2	204	122	96	67	24	3.04	1.43
112	SATTYAWWA	68	F	86	102	4.9	135	67	82	40	13	3.37	2.05
113	SHANTAMMA	70	F	96	120	6	195	106	98	31	21	6.29	3.16
114	KASHIBAI	60	F	88	112	5.6	174	74	112	47	14	3.7	2.38
115	MAHADEVI	60	F	110	128	6.1	229	109	166	41	21	5.58	4.04
116	SANGAMMA	45	F	118	138	4.9	169	139	108.9	29	31	5.82	3.75
117	RUKMAWWA	75	F	98	124	3.8	163	135	99	47	26	3.46	2.1
118	NEELAMMA	38	F	89	103	5.4	177	118	111	42	23	4.21	2.64
119	SHIVAMMA H	50	F	81	112	5.9	187	106	79	55	21	3.4	1.43
120	DRAKSYANI	58	F	118	139	5.4	188	122	75	51	15	3.68	1.47
121	SONUBAI	65	F	90	130	5.3	186	126	87	45	15	4.13	1.93
122	GANGUBAI C	65	F	100	140	6.3	212	133	98	44	27	4.81	2.22
123	SULTANBEE	40	F	121	145	5.6	174	74	112	47	14	3.7	2.38
124	VENKAMMA	40	F	64	108	5.1	184	119	94.2	46	23.8	4	2.04
125	GIRIJABAI	64	F	78	122	4.9	157	56	97	58	11	2.7	1.67