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# Comparision of Lipid Profile in Normal Subjects and Type – II Diabetes Mellitus.

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#### **Research Article**

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The prevalence of atherosclerosis and its clinical sequele like coronary artery disease (CAD) and peripheral vascular disease (PAD) is increasing among Indians and Diabetic population is more prone for atherosclerosis than normal population. Lipid profile is one of the basic and important parameters done to screen CAD, this study was under taken to compare lipid levels between diabetic and normal population. 30 normal individuals (mean age  $48.83 \pm 8.58$ ) and 30 T2DM (mean age 49.86 ± 7.21) were selected and were age and also BMI matched. Their blood samples were evaluated for FBS. PPBS, Total Cholesterol, Triglyceride, HDL cholesterol, LDL Cholesterol, VLDL Cholesterol. LDL was found to be significantly higher in T2DM than non diabetics (P = 0.035132). T - CHOL, TGL, VLDL were comparatively higher in T2DM although not significantly. HDL was comparatively lower though not significantly in DM than in normals. Despite similar BMI as that of normals,T2-DM are more prone for atherosclerosis and its clinical sequel.

ABSTRACT

## INTRODUCTION

A host of risk factors like lifestyle modification, increased body weight (BMI), elevated plasma insulin levels and insulin resistance are known to be associated with CAD and PAD in T2DM. Dyslipidemia is a major cause of coronary heart disease (CHD)—the most-common cause of death in the world—and is characterized by increased levels of lipids.

Biological molecules that are insoluble in aqueous solutions and soluble in organic solvents are classified as lipids. Plasma lipoproteins are separated by hydrated density; electrophoretic mobility; size; and their relative content of cholesterol, triglycerides, and protein into five major classes: chylomicrons, very-low-density lipoproteins (VLDL), intermediate-density lipoproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL) <sup>[1]</sup>.

Cholesterol, triglycerides, and high-density lipoproteins are important constituents of the lipid fraction of the human body. Cholesterol is an unsaturated alcohol of the steroid family of compounds; it is essential for the normal function of all animal cells and is a fundamental element of their cell membranes. It is also a precursor of various critical substances such as adrenal and gonadal steroid hormones and bile acids. Triglycerides are fatty acid esters of glycerol and represent the main lipid component of dietary fat and fat depots of animals. Cholesterol and triglycerides, being nonpolar lipid substances (insoluble in water), need to be transported in the plasma associated with various lipoprotein particles.

Dietary TGs are digested in the stomach and duodenum into monoglycerides (MGs) and FFAs by gastric lipase, emulsification from vigorous stomach peristalsis, and pancreatic lipase. Dietary cholesterol esters are de-esterified into free cholesterol by these same mechanisms. MGs, FFAs, and free cholesterol

are then solubilized in the intestine by bile acid micelles, which shuttle them to intestinal villi for absorption. Once absorbed into enterocytes, they are reassembled into TGs and packaged with cholesterol into chylomicrons, the largest lipoproteins. Cholesterol-rich chylomicron remnants then circulate back to the liver, where they are cleared in a process mediated by apoprotein E (apo E). Lipoproteins synthesized by the liver transport endogenous TGs and cholesterol. Lipoproteins circulate through the blood continuously until the TGs they contain are taken up by peripheral tissues or the lipoproteins themselves are cleared by the liver. TGs derived from plasma FFA and chylomicron remnants; VLDL synthesis increases with increases in intrahepatic FFA, such as occur with high-fat diets and when excess adipose tissue releases FFAs directly into the circulation (eg, in obesity, uncontrolled diabetes mellitus. Pathway defects in lipoprotein synthesis, processing, and clearance can lead to accumulation of atherogenic lipids in plasma and endothelium <sup>[2]</sup>.

A large body of epidemiological and pathological data documents that diabetes is an independent risk factor for CVD in both men and women both type 1 diabetes and type 2 diabetes are independent risk factors for CHD. Moreover, myocardial ischemia due to coronary atherosclerosis commonly occurs without symptoms in patients with diabetes. As a result, multivessel atherosclerosis often is present before ischemic symptoms occur and before treatment is instituted. A delayed recognition of various forms of CHD undoubtedly worsens the prognosis for survival for many diabetic patients <sup>[3]</sup>.

An elevated concentration of serum LDL cholesterol is a major risk factor for CHD. In fact, some elevation of LDL cholesterol appears to be necessary for the initiation and progression of atherosclerosis. In populations having very low LDL cholesterol levels, clinical CHD is relatively rare, even when other risk factors—hypertension, cigarette smoking, and diabetes—are common. In contrast, severe elevations in LDL cholesterol can produce full-blown atherosclerosis and premature CHD in the complete absence of other risk factors <sup>[3]</sup>.

#### Objectives

Lipid profile is one of the basic and important parameters done to screen CAD, this study was under taken to compare lipid levels between diabetic and normal population.

#### METHODS

#### Study population

The healthy controls and diabetic population, were recruited from the outpatient diabetic clinic at the Division of Endocrinology M.S Ramaiah Memorial Hospital Subjects were age and sex matched and were between 35-60 years.

#### Assays

Fasting blood samples were collected from controls and type 2 diabetics were evaluated for FBS and PPBS by glucose-oxidase method using glucose autoanalyser.

Total Cholesterol, Triglyceride, HDL Cholesterol, LDL Cholesterol, VLDL Cholesterol were evaluated by enzymatic method and calculated by Freidwald equation.

Ethical clearance was obtained from the M. S. Ramaiah Medical College ethical committee for human research to conduct the study. Pregnant women, patients on Statins for abnormal lipid treatment (both for Type 2 DM and Controls) were excluded. The study extended over a period of two years.

#### **Statistical Methods**

The Statistical software namely SPSS 11.0, Stata 8.0 and Systat 11.0 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc

#### **RESULTS AND ANALYSIS**

A case control study consisting of 30 T2DM patients and 30 healthy controls was under taken

#### Table 1: BMI of T2DM AND healthy controls

	T2DM	NORMAL
BMI	27.57 ± 5.04	25.57 ± 5.54

#### Table 2: Comparision of lipids T2DM AND healthy controls

	T2DM	Normal
T- CHOL	184.33 ± 35.6	178.97 ± 10.0
TDL	146.57 ± 56.2	145.4 ± 20.76
HDL	40.66 ± 7.98	42.39 ± 1.97
LDL	118.83 ± 26.13	106.03 ± 19.20
VLDL	33.11 ± 19.53	29.26 ± 5.534

#### DISCUSSION

Besides measuring blood pressure and glucose levels, assessing the lipid spectrum is the method most commonly used to identify individuals at high risk of cardiovascular disease (CVD), as well as those who are likely to benefit most from lipid-lowering therapy.

Type 2 diabetes is associated with a marked increased risk of cardiovascular disease (CVD). Individuals with diabetes have an absolute risk of major coronary events similar to that of nondiabetic individuals with established coronary heart disease (CHD) <sup>[1]</sup>. Furthermore, diabetic subjects develop congestive heart failure more frequently and have a higher mortality rate than nondiabetic individuals and dyslipidemia is one of the major cause and is a well-recognized and modifiable risk factor that should be identified early to institute aggressive cardiovascular preventive management <sup>[4]</sup>.

In our study we found LDL was found to be significantly higher in T2DM than non diabetics (P = 0.035132). This is similar to the study done by in the U.K. Prospective Diabetes Study who also stated despite a high frequency of modestly elevated baseline triglyceride levels (mean baseline 159 mg/dl), a multivariate analysis showed that triglyceride levels did not predict CHD events. LDL cholesterol was the strongest independent predictor of CHD followed by HDL cholesterol, supporting current national guidelines in which LDL lowering is the primary lipid target <sup>[5]</sup>.

A continuous, graded relationship exists between LDL cholesterol (LDL-C) levels and risk of cardiovascular disease (CVD). The size of LDL particles varies from large and buoyant to small and dense. Small, dense LDL is especially rich in cholesterol esters, is associated with metabolic disturbances such as hypertriglyceridemia and insulin resistance, and is especially atherogenic. The increased atherogenicity of small, dense LDL derives from less efficient hepatic LDL receptor binding, leading to prolonged circulation and exposure to endothelium and increased oxidation <sup>[3]</sup>.

LDL has a fairly long residence time with a plasma half-life of 2 to 5 days. Accordingly, the detection of elevated levels of fasting cholesterol usually reflects the presence of either increased numbers of LDL particles or increased cholesteryl ester in each LDL. LDL also exists in a range of sizes. Small dense LDL tends to occur in the setting of concomitant hypertriglyceridemia. This type of lipoprotein is thought to have greater atherogenic potential than larger LDL species, perhaps because of more facile access to the vascular wall and greater susceptibility to oxidative modification. Lipoprotein particle size and number can be quantified by nuclear magnetic resonance techniques, but it is not clear that these data provide diagnostic advantages beyond the determination of total cholesterol, triglycerides, LDL cholesterol, and HDL cholesterol <sup>[6,8]</sup>.

We also found in our study that T – CHOL, TGL, VLDL were comparatively higher in T2DM although not significantly. HDL was comparatively lower though not significantly in DM than in normal controls. As per the definition given by WHO(World health organisation) all these features are suggestive of dyslypidemia in T2DM which states that most typical lipoprotein pattern in diabetes, also known as diabetic dyslipidemia or atherogenic dyslipidemia, consists of moderate elevation in triglyceride levels, low HDL cholesterol values, and small dense LDL particles. Small dense LDL particles are highly atherogenic because of their enhanced susceptibility to oxidative modification and increased uptake by the arterial wall <sup>[5]</sup>.

We found that HDL was comparatively lower though not significantly in T2DM than in normals. The lipoprotein HDL has two important roles: first, it promotes reverse cholesterol transport, and second, it modulates inflammation. Epidemiological studies show that HDL-cholesterol levels are inversely correlated with the risk of cardiovascular events. However, many patients who experience a clinical event have normal, or even high, levels of HDL cholesterol. Measuring HDL-cholesterol levels provides information about the size of the HDL pool, but does not predict HDL composition or function <sup>[1,2]</sup>.

The American Diabetes Association (ADA) has set desirable LDL cholesterol, HDL cholesterol, and triglyceride levels as < 100, > 40 in men/> 50 in women, and < 150 mg/dl, respectively <sup>[7]</sup>.

The onset of hyperglycemia in patients with the metabolic syndrome appears to accelerate atherogenesis, possibly by enhanced formation of glycosylated proteins and advanced glycation products and/or by increasing endothelial dysfunction. These direct consequences of hyperglycemia probably contribute to the microvascular disease underlying nephropathy and retinopathy, and they may promote macrovascular disease as well <sup>[6]</sup>.

#### CONCLUSION

LDL was found to be significantly higher in T2DM than non diabetics (P = 0.035132) and is predictive of CVD risk. T – CHOL, TGL, VLDL were comparatively higher in T2DM although not significantly. Also HDL was comparatively lower though not significantly inT2DM than in normals. Despite similar BMI as that of normals,T2-DM are more prone for atherosclerosis and its clinical sequel.

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