Introduction
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Diabetes mellitus (DM) comprises a group of common metabolic disorder that share the phenotype of hyperglycemia. Diabetes mellitus is the most prevalent metabolic non communicable disorder in the world. The increasing prevalence of type II diabetes mellitus is a global problem, and it is unfortunately, a major one in developing countries such as India. The world presently has nearly 150 million diabetes of which one fifth that is approximately 33 million are in India. This number is predicted to double by 2025. In fact India has been dubbed as a diabetes capital of the world at the recent 2003 International diabetes federation (IDF) conference in Paris (1).

Diabetes mellitus has emerged a major public Health problem in our country and has assumed epidemic proportions. Prevalence of diabetes has increased form 2.1 % in 1972 to almost 20% in 2003. The vast majority of these are type 2 patients

Most of the diabetes develop micro and macrovascular complications of it, manifesting as cardiovascular disease, cerebro-vascular disease, renal disease, peripheral vascular
disease etc. Cardiovascular disease is presently the leading cause of death among persons with diabetes (2). Dyslipidemia is one of the major risk factors for cardiovascular disease and is also very common in both individuals with type I and type II.

Dyslipidemia is a state in which circulating levels of lipids or lipoprotein fractions are abnormal because of genetic and/or environmental conditions that alter the production, catabolism or clearance of plasma lipoproteins. An understanding of lipoprotein metabolism is of particular importance because of association of lipoproteins with coronary heart disease, one of the leading causes of mortality in today’s world (30% of total deaths).

Abnormalities in lipoproteins are very common in both individuals with type II and those with type I. Although lipoprotein alterations appear to be intrinsic part of these disorders such alterations also are induced by diabetes - associated complications such as obesity and renal disease.

**Dyslipidemia in Type I Diabetes:**

A spectrum of situations are possible in type I, from Insulin deficient ketoacidotic state with greatly elevated glucose, Free Fatty acids, ketones and lipolytic enzymes such as glucagon and...
epinephrine, to a state of continuous insulin infusion therapy with hyperinsulinemia, normal or close to normal glucose and fatty acids level. The lipid profile was near normal in well controlled type I diabetes.

In untreated type I diabetics the fractional catabolic rate for triglyceride decreased because the activity of lipoprotein lipase is dependent on insulin and leads to hypertriglyceridemia (3). Sometimes severe enough to cause lipoaemia retinalis, acute pancreatitis and eruptive xanthomas.

VLDL (C) levels are greatly elevated in individuals in diabetic ketoacidotic state (4). LDL (C) levels are increased in poorly controlled type I subjects. LDL fractional clearance is probably decreased in poorly controlled type I individuals because insulin potentiates LDL binding to its receptors.

The LDL particles of individuals with type I may exhibit an increase in the ratio of cholesterol to Apo - B (5). In addition glycation of the LDL also interferes with the clearance of it.

A number of studies have shown that HDL-C concentrations are low in poorly controlled type I individuals and it increased with the degree of glycemic control. But some
studies have shown that HDL-C concentration in type I individuals are higher or not lower than the control subjects. Low lipoprotein lipase activity may be an important for lowered HDL-C concentrations in type I subjects. Adequate control of plasma glucose in type I individuals leads to increase in the HDL-C levels (6,7).

**Type II diabetes mellitus causes A characteristic Dyslipidemia**

Triglycerides and very low density lipoprotein cholesterol (VLDL-C). The most common alteration of lipoprotein in type II is hypertriglyceridemia caused by an elevation in VLDL-C. The most important factor responsible for increased VLDL level is over production of VLDL triglyceride probably due to increase flow of substrates such as glucose and Free Fatty acids to the liver. In addition individuals with type II have defect in clearance of VLDL triglycerides. Due to decreased lipoprotein lipase activity which parallels to the insulin resistance, thus in turn to hyperglycemia.

**Low density lipoprotein-cholesterol (LDL-C):** studies examining plasma concentration of total cholesterol and LDL-C in type II have been contradictory with some showing higher and showing lower levels in type II than in control subjects. But the
recent study II of the National Health and Nutrition Examination Survey, USA, indicates that elevations of LDL-C concentrations are more common in individuals with type II DM that in the general population (8, 9,10).

**High Density Lipoprotein:** - Cholesterol (HDL-C) the individuals with type II DM have lower concentration of HDL-C as compared with control subjects because of increased clearance rate of HDL-C which is directly related with plasma glucose concentration. Since HDL-C concentration increases during lipolytic process, lipoprotein lipase activity has been shown to correlate significantly with the HDL-C concentrations in individuals with type II DM.

As with LDL and VLDL particles, an increased proportion of triglyceride in HDL particles have also been observed. An increase in the ratio of cholesterol to protein in HDL particles has been reported. These compositional changes appear to correlate with the degree of stimulation of adipose tissue lipoprotein lipase. In addition, glycation of the HDL particles appears to interfere with binding to receptors (11).

**Apo-B concentration:** - An elevated Apo-B concentration is
another common feature of the Dyslipidemia of type II diabetes.
 Elevated Apo-B levels are found in almost half of normocholesterolaemic patients with type II diabetes and are frequently associated with low HDL cholesterol levels and hypertriglyceridemia. Indeed, an increased in Apo-B levels may predict CHD events better than LDL-Cholesterol levels (12).

**Apo-Al Concentrations:** - Apo-Al is the crucial structural apoprotien for HDL. An contrast to athenogenic Apo-B lipoproteins, the Apo-Al containing HDL appear to be anti atherogenic. In fact in some studies, HDL cholesterol levels are as strong an indication of protection from CHD as LDL-cholesterol levels are an indicator of risk (13).

**Lipoprotein-a:**

Lipoprotein-a IS an LDL-like particle that carries the Lpa specific highly glucosylated protein Apo-a. Glycamic control and insulin therapy may influence Lp-a level in patient with diabetes. There is no clear evidence that Lp-a contributes significantly to the increased risk of atherosclerosis in diabetes, although diabetic nephropathy seem to be associated with high Lp-a levels. LDL cholesterol connected positively and
triglyceride negatively with Lp-a concentrations (14).

Lipoprotein level and coronary heart disease. In the past raised LDL-cholesterol levels were held largely responsible for the increased risk of CHD. But it is now clear that other lipid abnormalities, reduced HDL-cholesterol levels and increased triglyceride concentrations may be more important in diabetic patients.

Dyslipidemia and Hypertension:

Hypertension is an independent risk factor for the development of CHD as well as stroke, and does not significantly affect lipid levels. There is a synergistic risk enhancement effect of concurrent dyslipidemia and hypertension. So, every hypertensive patient should be screened for dyslipidemia.