Introduction
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Diabetes Mellitus

Prevalence of diabetes particularly type 2 diabetes mellitus is increasing rapidly all over the world\(^1\). International Diabetes Federation (IDF) - 2011 has reported that India have the second largest number of diabetic people (61.3 million) in the world and projections show that by 2030 this figure is likely to increase by 101.2 million (IDF Diabetes Atlas 2011). Also, studies from urban India show a rising trend in the prevalence of diabetes\(^2,3,4,5,6,7\). The prevalence of diabetes and impaired glucose tolerance (IGT) has reached epidemic proportions\(^7,8,9,10,11\) in urban India making it a major national public health problem.

There are major differences in prevalence of diabetes in rural (6.3%)\(^12\) and urban areas (12.1%)\(^9\). Recent studies from Chennai\(^13\) (14.3%) and Kochi\(^14\) (19.5%) have shown that the prevalence is rising even further.

The important risk factors reported for the high prevalence of diabetes in India are increasing age, ethnicity, familial aggregation and genetic factors, obesity particularly central obesity and life style changes associated with urbanization\(^9,15,16,17\) i.e., changes in dietary intake and physical activity.

Macrovascular Disease

Macrovascular disease is a common complication of diabetes mellitus and is the leading cause of morbidity and mortality in patients with type 2 diabetes mellitus. Mortality from cardiovascular disease is nearly twice as high in type 2 diabetes patients (both sexes) compared to those without T2DM\(^18,19\). Moreover, T2DM increases the risk of coronary and peripheral artery disease by 2 - 4 fold and the risk of stroke is increased by 10 fold if patients with type 2 diabetes are younger than 55 years\(^20,21,22\).
Approximately 75% of deaths among type 2 diabetic patients are accounted for by cardiovascular disease\(^{(23)}\). Age, family history, diabetes, hypertension, smoking, high total and LDL cholesterol, low HDL cholesterol, and obesity are established risk factors for atherosclerosis in the general population\(^{(24,25)}\). Recently, hypertriglyceridemia and homocystein have also been recognized as cardiovascular risk factors\(^{(25)}\). However, at any given number of risk factors, diabetes patients have an approximately 3-fold CV risk indicating a specific, diabetes-related effect. Data is also accumulating on detrimental postprandial lipid metabolism elevation of atherogenic lipoproteins in type 2 diabetic patients\(^{(26,27,28)}\).

### Post Prandial State

In post prandial state, there is an inevitable rise in blood sugar levels (hyperglycemia) and lipid levels (hyperlipidemia particularly hypertriglyceridemia), leading to the activation of metabolic pathways. Under healthy conditions, biosynthesis and oxidative metabolism of these absorbed food products viz glucose, proteins, lipids and other dietary components takes place and almost every major biological system, organ, tissue and cell responds with compensatory and corrective mechanisms to maintain body homoeostasis. This results in optimal levels of glucose, lipids etc in blood and the damage from transient hyperglycemia and hyperlipidemia is modest allowing the rapid recovery.

However, sometimes in post prandial state, body fails to maintain homeostasis and the metabolic defects leads to cellular dysfunction, diseases and ultimately death. Thus postprandial period is susceptible for biological systems disorders, particularly in view of the (a) most of the time, peoples live in post prandial state (b) type of meal people have these days\(^{(29)}\).

There is increasing evidence that when these repair mechanisms are not optimal, body homeostatis is disturbed and results in development of chronic diseases like type 2 diabetes, cardiovascular disease\(^{(30,31)}\).
Post Prandial Lipaemia

Thirty years ago it was BD Zilversmit (1979) who proposed the concept of post prandial lipaemia. He suggested that postprandial lipaemia (PPL) may play a role in atherogenesis (32).

Post prandial lipaemia is a disorder of lipid metabolism in post prandial state. The most affected and increased type of lipid in post prandial state is triglyceride, a condition known as post prandial hypertriglyceridemia (PPHTg). It comprises abnormally high concentrations of lipids particularly TGs in blood in the post prandial state. In last one decade or so role of postprandial lipaemia particularly PPHTg has emerged as an important risk factor in the development of macrovascular disease.

This has been shown to be an independent risk factor for atherosclerosis and correlates significantly with surrogate markers of atherosclerosis such as CIMT and endothelial function.

In India, studies on PPL are very few and apart from our own institute (33, 34, 35), only study on PP lipids is of Jamal Ahmad. He found significant association between post prandial hypertriglyceridemia and CIMT in north Indian T2DM patients (36).

Apart from its possible role in atherosclerosis, post prandial lipaemia particularly PPHTg is being recognized as a risk factor in insulin resistance. However, studies on the role of PPL in the pathogenesis of T2DM are few and preliminary. To the best of our knowledge, there is no study to date which clearly establishes the role of PPL in causation of IR and T2DM.

In recent years, some of the studies show that PPL is an early abnormality in the natural history of T2DM. Post prandial hypertriglyceridemia has been demonstrated in genetically predisposed first degree relatives of T2DM, in prediabetic subjects and in newly detected diabetes patients, all of which suggest that PPL occurs early and possibly much before the onset of T2DM. Experimental studies as well as case report in humans also suggest that post prandial hypertriglyceridemia could lead to IR and glucose intolerance. However, results of these studies have been inconclusive and require further investigation and substantiation. Despite the growing evidence of link of PPHTg with
insulin resistance and type 2 diabetes, the exact link/relationship of post prandial lipaemia with insulin resistance and or type 2 diabetes has not been understood so far. The present study was therefore carried out in an experimental setup to draw a conclusion that could help to understand the precise role of PPL particularly post prandial hypertriglyceridemia in the pathogenesis of IR, T2DM and diabetes related atherosclerosis.