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Postprandial Hypertriglyceridemia among Type-2 Diabetes Mellitus Patients of Chittagong, Bangladesh

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ABSTRACT

Diabetes mellitus (DM) is a metabolic disorder of multiple aetiology characterized by chronic hyperglycemia with disturbances of carbohydrate,fat and protein metabolism resulting from defects in insulin secretion, insulin action,or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels. A case-control study was carried out in Chittagong Medical College, Chittagong, Bangladesh during the period of January 2012 to December 2012. The samples were collected from the department of Medicine, Chittagong Medical College Hospital. The age limits of the patients were from 30-70 years. The data were collected by a structured questionnaire including age, sex, blood pressure, total cholesterol. Total numbers of patients were 90 of which 50 were considered as cases (All type-II diabetic patients having (i) age 30-70 years (ii) Fasting blood sugar ≥ 7 mmol /L. (iii) Random blood sugar ≥11.1 mmol/L. (iv) 2 hrs. after oral glucose load is ≥ 11.1mmol/L and 40 controls (non-diabetic, age 30-70 years, absence of renal, liver and cardiovascular diseases). The study was designed to observe the postprandial triglyceride level in type-II diabetic patients. The mean fasting triglyceride level of cases was 210.70 (±19.5) and the 2hrs, 4hrs and 6hrs after test meals were 238.9 (±22.75), 260.5 (±15.36), and 260.32 (±5.94) respectively. At the same time the mean fasting triglyceride of the control was 173.75 (±19.86) and the corresponding mean of the control were 189.75 (±15.23), 174.38 (±16.49) and 173.88 (±15.79) mg/dl respectively. The fasting and postprandial (2hrs, 4hrs and 6hrs) triglyceride levels were significantly higher than that of corresponding control. There are also significant differences of triglyceride level in fasting and 2hrs, 4hrs and 6hrs after test meal among the cases which indicated that the triglyceride levels remained elevated for longer postprandial duration. It is also observed that there is a tendency of raised triglyceride level with time after test meal in cases compared with that of controls; it is because insulin resistance is a feature of type-II diabetes mellitus (T2DM), which is responsible for triglyceride overproduction.

Keywords: Type 2 diabetes mellitus, Triglyceride (TG), Cholesterol

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INTRODUCTION

Diabetes mellitus is a metabolic disorder of multiple aetiology characterized by chronic hyperglycemia with disturbances of carbohydrate, protein and fat metabolism resulting from defects in insulin secretion, insulin action,or both[1] . There are two main types of diabetes mellitus named as type-1 and type-2[2]. Type 1 diabetes mellitus is caused by an immune mediated destruction of the β cells resulting in an absolute insulin deficiency. The body does not produce insulin in type 1 diabetes and only 5% of people with diabetes have this form of the disease[2]. In individuals with type-2 diabetes mellitus, hyperglycemia is caused by a combination of an impairment of the β- cells to release sufficient insulin (β-cell dysfunction) and an impaired metabolic response of peripheral tissues to circulating insulin (insulin resistance)[3]. Deficient action of insulin on targeted tissues either due to insulin resistance or insulin deficiency, not only leads to hyperglycemia but also to other metabolic abnormalities including abnormalities in fasting and postprandial lipid metabolism (dyslipidemia)[4] . Dyslipidemia associated with type-2 diabetes mellitus is characterized by hypertriglyceridemia, decrease high-density lipoprotein (HDL)-cholesterol, and increased small and dense low-density lipoprotein (LDL) particles, which are more atherogenic[5].

The worldwide prevalence of T_2DM is growing rapidly. One of the major reasons of the increased prevalence in developing countries is the adoption of the so-called western lifestyle, i.e. a high intake of energy dense food and a low physical activity pattern. These life-style changes lead to one of the key abnormalities underlying T_2DM, i.e. insulin resistance[6] . In 1988, the insulin resistance syndrome or syndrome X was described[7], now called the metabolic syndrome. It was originally defined by the presence of hyperinsulinemia (compensatory for the underlying insulin resistance), varying degrees of glucose intolerance, hypertriglyceridemia and low plasma HDL cholesterol concentration.

The most common working definition, as proposed by the Adult Treatment Panel III [ATP3] of the National Cholesterol Education Program (NCEP), the metabolic syndrome is characterized by central obesity, hypertriglyceridemia, low plasma HDL-cholesterol, hypertension and impaired fasting glucose[8]. Postprandial lipemia, characterized by a rise in triglyceride-rich lipoproteins after eating, is a dynamic, non-steady state condition in which humans spend the majority of time. There are several lines of evidence suggesting that postprandial lipemia increases risk of atherogenesis. Clinical data show a correlation between postprandial lipoproteins and the presence or progression of coronary artery disease and carotid intimal thickness[9]. Mechanistic studies demonstrate that triglyceride-rich lipoprotein remnants may have adverse effects on endothelium and can penetrate into the sub-endothelial space[9].

It is well recognized that type-2 diabetic patients have an excess risk of developing atherosclerosis, resulting in high cardiovascular disease (CVD) morbidity and mortality[10] Therefore, with the rise of the prevalence of diabetes, it may be expected that the global burden of CVD will also increase[11]. Among individuals with T_2DM, the cardiovascular disease morbidity and mortality is two to four times higher compared to individuals with normal glucose metabolism[12]. The CVD mortality in individuals with T_2DM accounts for as much as 75% of all deaths[12, 13]. In the Reykjavik Study, a population-based study with over 18000 elderly participants, the relative impact of elevated glucose, triglycerides and systolic blood pressure with regard to fatal and non-fatal coronary heart disease (CHD) was higher in women than in men[13]. Over 200 years ago, William Heberden made the first observation regarding a postprandial effect on the circulation of blood[14]. Zilversmit postulated that atherosclerosis is a postprandial phenomenon[15]. Ever since, a large body of evidence has accumulated indicating a relationship between postprandial dysmetabolism, especially hyperglycemia and hypertriglyceridemia, and the risk of CVD[15]. After a fatty meal, the dietary fats leading to triglyceride-rich lipoproteins (TRL) and endogenous TRL production[16] . In the insulin-resistant state the production of very-low density lipoprotein (VLDL) by the liver is inappropriately high. Together with a reduced lipoprotein lipase (LPL) activity this results in high triglyceride (TG) concentrations, especially in the postprandial state. These abnormalities may explain the characteristic of diabetic dyslipidemia, which is now recognized to be significantly atherogenic[16]. In the past few years several clinical studies have suggested that high postprandial TRL may be related to coronary heart and/or carotid artery disease in non-diabetic and diabetic subjects[17].

The Physicians' Health Study, a prospective nested case control study was conducted in 14,916 men aged 40 to 84 years. 85% of them had baseline blood samples taken under non-fasting conditions[18]. The primary outcome was occurrence of myocardial infarction (MI) during 7 years of follow-up. A key finding was that cases (n = 266) had higher median non-fasting TG levels compared to controls (n = 308). After simultaneous adjustment for age, smoking status, HDL- and total cholesterol levels, LDL and a variety of coronary risk factors, this study suggested that random or postprandial TG concentrations are an important indicator of CVD risk[18].

MATERIALS AND METHOD

An oral fat challenge was given to type II diabetic patients (n=50) and age, sex & BMI matched healthy controls (n=40) those had no family history of diabetes mellitus. Diabetes was diagnosed as per revised American Diabetic Association (ADA) criteria. Patients with nephropathy, hepatic disease, hypothyroidism, Cushing's disease, inherited disorders of lipid

metabolism, clinical or ECG evidence of coronary artery disease (CAD), alcoholism, smoking or use of medication affecting lipids were excluded. All (cases and controls) were subjected to preliminary clinical & laboratory assessment which included fasting plasma glucose (FPG), 2 hrs postprandial plasma glucose (PPPG), lipid profile in fasting and 2hrs, 4hrs, and 6hrs after test meal. All the above set of investigations was also performed for controls. After a 14 hour overnight fast, a standardized meal was given to all subjects providing regular amounts of energy (about 700–1000 kcal) and fat (about 40–50 g/test meal). Dubois et al. (1998) advised a test meal of following composition: [bread (30 g), cooked pasta (100 g), tomato sauce (130 g), one non-fat yogurt (125 g), a cup of tea (100 ml) and the tested fat (sunflower oil). Protein (22.1g), carbohydrate (125.1g), Fat (50 g)]. Blood was drawn at 0, 2, 4, and 6 hrs for lipids estimation. Serum was separated and stored at - 20^{o}c for various estimations. All type-2 diabetic patients (cases) having following criteria: age within 30-70 years, fasting blood sugar ≥7 mmol/L., random blood sugar ≥11.1mmol/L.& 2 hrs after oral glucose load ≥11.1mmol/L. Healthy, non-diabetic (controls) were within age 30-70, absence of renal and liver disease and, absence of cardiovascular diseases.

Data analysis

Data were analyzed by using SPSS 18 statistical software. Values were expressed as mean ± SEM. Confidence level was fixed at 95% level and 'p' value of 0.005 or less was considered significant. Student's t' test for quantitative or continuous variable and Chi-square test for categorical variable were done.

RESULTS AND DISCUSSION

Total 90 individuals have been included in this study where 50 diabetic patients (cases) and 40 normal healthy individuals (controls) (Table 1). Mean fasting triglyceride of the cases was 210.70 (±19.51) mg/dl. The 2 hrs, 4 hrs and 6 hrs mean triglyceride level after test meals were 238.9 (±22.75), 260.5 (±15.36), and 260.32 (±5.94) mg/dl respectively. At the same time the mean fasting triglyceride of the control was 173.75 (±19.86) mg/dl and the corresponding mean of the control were 189.75 (±15.23), 170.2 (±12.3) and 167.5 (±10.3) mg/dl respectively. The fasting and postprandial (2 hrs, 4 hrs and 6 hrs) of study groups were significantly higher than that of corresponding controls. There is an also significant difference among the triglyceride level in fasting and 2hrs, 4hrs & 6hrs after test meal among the study group.

Table–1: Distribution of serum triglyceride status among the study groups (with X^2 test significance) 2 hrs after test meal

Serum Triglyceride Status	Study Groups				Total	
	Group A		Group B			
	n	%	N	%	N	%
Increased	45	90.0	14	35.0	59	65.6
Normal	05	10.0	26	65.0	31	34.4
Total	50	100.0	40	100.0	90	100.0
X^2 value = 29.770. P = 0.000. Highly Significant.						

Table–2: Distribution of serum (fasting & post-prandial) triglyceride among the study groups (with t - test significance) 2 hrs, 4hrs and 6hrs after meal

		N	Mean	± SD	Median	Range	Sign.
Serum Triglyceride (Fasting)(mg/dl)	Group A	50	210.70	19.51	210.00	160 – 250	P = 0.000
	Group B	40	173.75	19.86	172.50	140 – 210	Highly
	TOTAL	90	194.28	26.89	197.50	140 – 250	Significant
Serum TG (Post-prandial- After 2 hours) (mg/dl)	Group A	50	238.90	22.75	237.50	200 – 300	P = 0.000
	Group B	40	189.75	15.23	187.50	170 – 220	Highly
	TOTAL	90	217.06	31.46	217.50	170 – 300	Significant
Serum TG (Post-prandial- After 4 hours) (mg/dl)	Group A	50	260.50	15.36	260.00	230 – 290	P = 0.000
	Group B	40	174.38	16.49	175.00	150 – 200	Highly
	TOTAL	90	222.22	45.84	242.50	150 – 290	Significant
Serum TG (Post-prandial- After 6 hours) (mg/dl)	Group A	50	260.32	5.94	260.00	250 – 275	P = 0.000
	Group B	40	173.88	15.79	175.00	150 – 200	Highly
	TOTAL	90	221.90	44.66	255.00	150 – 275	Significant

This was a case control study done over a period of 12 months from January 2012 to December 2012. Study included 50 cases and 40 controls. All cases were type-II diabetic mellitus patients. Age and sex matched healthy individuals included as control; all of them were non diabetic. The study was designed to observe the postprandial triglyceride level in type-II diabetic patients. The fasting mean triglyceride level of cases was 210.70 (±19.51) mg/dl and 2hrs, 4hrs, and 6hrs mean triglyceride level after test meal were 238.9 (±22.75), 260.5 (±15.36), and 260.32 (±5.94) mg/dl respectively. At the same time the mean fasting triglyceride of the control was 173.75 (±19.86) and corresponding control were 189.75 (±15.23), 174.38 (±16.49) and 173.88 (±15.79) mg/dl.

The fasting and postprandial (2hrs, 4hrs and 6hrs.) of study groups were significantly higher than that of corresponding control. There is also significant differences among the triglyceride level in fasting and 2hrs, 4hrs 6hrs after test meal among the study group of time. A trend was seen in study group that TG level increases and remain elevated even in 6hrs after test meal which was also confirmed by t-test.

A prediction may be done that in study cases TG level remained elevated for longer postprandial duration. It is because of the food habits (mainly rice) of our study group

individuals might have longer duration of lipemic status in blood in other times. At the same time it may be added the metabolic tolerance of ingested TG of the test meal with T_2DM might be altered in such cases as because of insulin resistance.

CONCLUSION

There are multiple changes in lipoprotein metabolism in both insulin-dependent and non-insulin-dependent diabetes mellitus. In this study, there is a positive correlation of rising triglyceride level with time after test meal in cases compared with that of control. As because insulin resistance is a feature of type-II diabetes mellitus, which is responsible for triglyceride overproduction. It must be determined whether there are alterations in content or composition of triglyceride rich lipoproteins in diabetes, because of the relative importance of these in the atherogenic process. Much of the pathophysiology linking diabetes and dyslipidemia has been elucidated. Although undoubtedly of importance, diabetic dyslipidemia is likely to be but one of many reasons for the accelerated macrovascular disease in diabetic patients. Nonetheless, treatment of lipid abnormalities has the potential to reduce cardiovascular events more than 50%, to rates that are seen in countries with lower cholesterol and less atherosclerotic burden. This leads to the expectation that treatment of elevated lipid levels will allow patients with diabetes to lead longer healthier lives.

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