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Original Article

Study of Postprandial Triglycerides as a Risk Factor for Ischemic Heart Disease

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Abstract

Introduction: Ischemic heart disease (IHD) is widely prevalent both in the developed and developing countries and continues to be a leading cause of mortality despite recent advances in diagnostic facilities and treatment modalities. There has been considerable controversy over whether elevated levels of triglycerides (TGs) or hypertriglyceridemia (HTG) are an independent risk factor in coronary heart disease.

Aim: This study aims to study the relationship between risk factors for atherosclerosis using postprandial TG levels in patients of unstable angina.

Materials and Methods: This prospective observational study was conducted in the Department of General Medicine at Ramanathapuram Headquarters Hospital from January 2019 to July 2019. The diagnosis was made based on typical chest pain history, electrocardiogram, and elevated levels of cardiac enzymes, creatinine kinase (CK), CK-MB, fasting serum TGs <150 mg%, and fasting serum cholesterol <180 mg%. Results were analyzed statistically and discussed below.

Results: A total of 50 patients were included in this study, 28 were male and 22 were female. In our research, the highest numbers of patients are in 46–55 years. About 40% of the patients in our study are overweight. About 76% of patients had high waist–hip ratio (WHR). About 64% of patients had elevated serum TG level; 58% of patients had low fasting high-density lipoproteins (HDL). About 64% of patients had diabetes, and 46% of patients had hypertension. About 69% of patients in high TGs had lower HDL level. About 82% of patients in the high WHR had increased TGs level. About 67% of overweight patients had increased TGs level. About 75% of diabetic patients had an increased TG level.

Conclusion: Postprandial HTG may be an independent risk factor for atherosclerosis in IHD patients. Evaluation of postprandial TG levels is essential during the assessment of IHD patients.

Key words: Ischemic heart diseases, Postprandial hypertriglyceridemia, Unstable angina

INTRODUCTION

Coronary heart disease (CHD) is widely prevalent both in developed and developing countries. It continues to be a leading cause of mortality despite recent advances in diagnostic facilities and treatment modalities. It is a multifactorial disease where atherosclerosis and dyslipidemia were the prominent causes involved.[1] Triglyceridemia is an independent risk factor for coronary artery disease irrespective of total cholesterol and low-density lipoprotein (LDL) cholesterol or low high-density lipoprotein (HDL) cholesterol.[2] Major risk factors for CHD are alterations in different cholesterol lipoprotein lipids, such as elevated total cholesterol, LDL cholesterol, very LDL (VLDL) cholesterol, triglycerides (TGs), and low HDL cholesterol. There is a clear pathophysiological association of elevated LDL cholesterol with the initiation and progression of coronary atherosclerosis. Robust data are available that show that lowering levels can minimize and stabilize the atherosclerotic vascular disease.

While the death rate of CHD decreases as a result of vital intervention initiatives and successful secondary prevention of risk factors, CHD is still the most common cause of death in the world, ranking the leading cause

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of death. Hypertension, diabetes, hypercholesterolemia, and smoking have been established as independent risk factors for CHD, and hypertriglyceridemia (HTG) has been controversial for a long time. However, several prospective studies conducted in recent years indicate that HTG is an independent risk factor for CHD. Recently, it has been proposed that postprandial lipoprotein may be a better indicator of deranged lipoproteins metabolism and hence of atherosclerosis and CHD.[3]

The most common dyslipidemia in India is borderline high LDL cholesterol, low HDL cholesterol, and high TG levels. Studies have recorded a rise in total cholesterol, LDL cholesterol, and TG levels in urban populations over 20 years. Case–control studies have identified a significant association of coronary events with elevated apolipoprotein B, total cholesterol, LDL cholesterol, and non-HDL cholesterol, and an inverse association with high apolipoprotein A and HDL cholesterol. The prevalence of suspected family hypercholesterolemia in urban subjects' ranges from 1:125 to 1:450. The association between atherosclerotic diseases and elevated fasting plasma LDL cholesterol (LDL-C) and reduced fasting plasma HDL-C is well established. However, many individuals without fasting lipid abnormalities develop atherosclerotic diseases, and several lines of evidence suggest that no fasting lipid measurements may be more relevant to atherogenesis.[4] Typical diets are associated with measurable postprandial lipidemia 18 h/day. A primary source of circulating TGs is dietary fat, which after hydrolysis into free fatty acids and glycerides, is transported through the intestinal villi and absorbed by enterocytes where these particles are synthesized into chylomicron-associated TGs for entry into the blood compartment and ultimately storage in adipose tissue.[5] Postprandial lipids and their associated partially hydrolyzed chylomicron remnants appear to promote early atherogenesis and adversely affect endothelial function, associate with atherogenic small LDL particles, and correlate with both pro-thrombotic and pro-inflammatory biomarkers, including factor VII, plasminogen activator inhibitor-1, and C-reactive protein.[6] Thus, measurement of postprandial TGs – mainly because they peak 3–4 h after ingestion of a fat-rich meal might well provide more relevant information on vascular risk than measures based on fasting concentrations. Diabetic patients have delayed clearance of TG from the blood. Most Type 2 diabetic patients have postprandial TGs [7] above optimal concentrations for several hours after meals. Moreover, optimal fasting concentrations are not always a good predictor of postprandial TGs. Hence, there should be some association of postprandial lipid metabolism and atherosclerosis, which remains to be proved.[3]

Aim

This study aims to study the relationship between risk factors for atherosclerosis using postprandial TG levels in patients of unstable angina.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of General Medicine at Ramanathapuram Headquarters Hospital from January 2019 to July 2019. Inclusion criteria include patient presented 1st time with ST-segment elevation myocardial infarction (STEMI), non-STEMI, or unstable angina and exclusion criteria are patients already on lipid-lowering drugs, Prinzmetal's angina rheumatic heart disease, and abnormal liver and renal function test.

The diagnosis was made based on typical chest pain history, electrocardiographic changes, and elevated levels of cardiac enzymes, creatinine kinase (CK), CK-MB, fasting serum TGs < 150 mg%, and fasting serum cholesterol < 180 mg%.

Body mass index (BMI) was calculated with the formula of BMI = wt (kg)/Ht (m2). Waist circumference was measured at the umbilical level. Waist–hip ratio cutoff points > 1.0 for male and > 0.85 for female were considered. Recording of electrocardiogram was done with 12 leads recording in standard fashion with BPL machine.

RESULTS

A total of 50 patients were included in this study, 28 were male and 22 were female. In our research, the highest numbers of patients are in 46–55 years. About 40% of the patients in our study are overweight. About 76% of patients had high waist–hip ratio (WHR). About 64% of patients had elevated serum TG level; 58% of patients had low fasting HDL. About 64% of patients had diabetes, and 46% of patients had hypertension. About 69% of patients in high TGs had lower HDL level. About 82% of patients in the high WHR had increased TGs level. About 67% of overweight patients had increased TGs level. About 75% of diabetic patients had an increased TG level [Figures 1-5].

DISCUSSION

A meta-analysis of 29 prospective studies in the Western population shows that TG is moderately or highly correlated with CHD.[8] Another meta-analysis involving 61 future studies further confirmed that high TG levels were associated with all-cause death of cardiovascular diseases.[9]

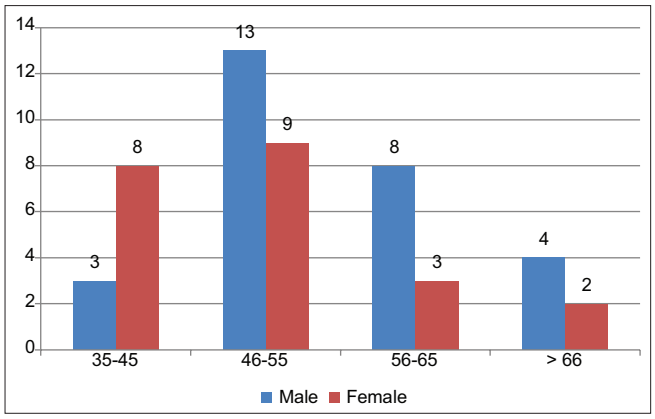

Figure 1: Age distribution according to gender

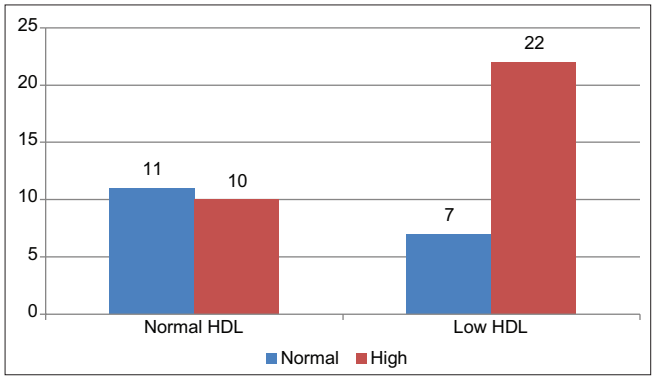

Figure 2: Distribution according to PP4TG and high-density lipoproteins

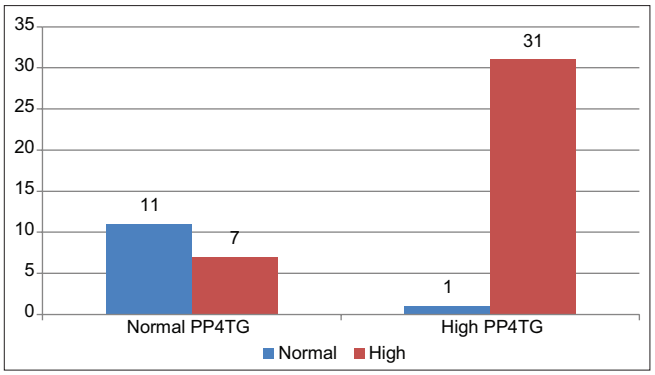

Figure 3: Distribution according to waist–hip ratio and PP4TG

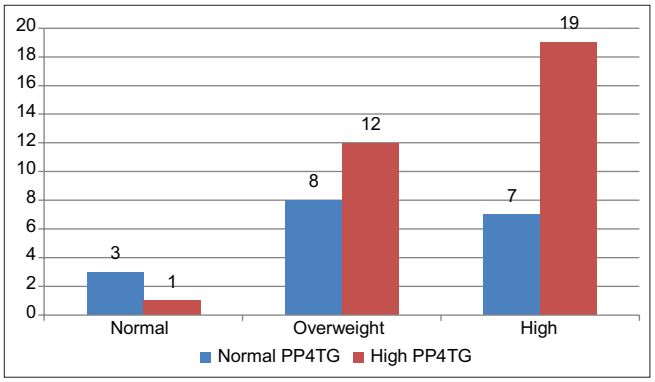

Figure 4: Distribution according to body mass index and PP4TG

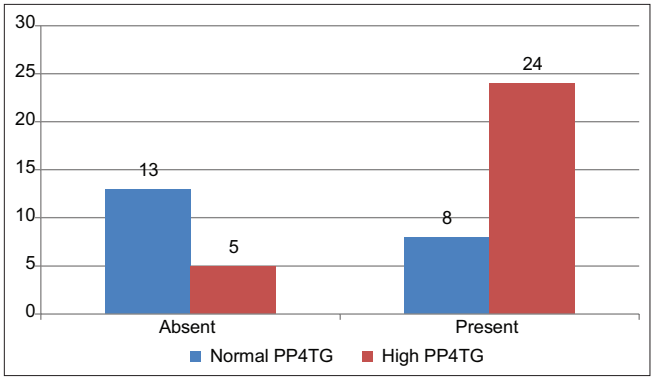

Figure 5: Distribution according to diabetes and PP4TG

Japan Diabetes Complications Study[10] found that TG is a risk factor of CHD equivalent to LDL-C as for Japanese patients with Type 2 diabetes. For every 1 mmol/L increase in TG and LDL-C levels, the risk of CHD increases by 63% and 64%, respectively. Bezafibrate Infarction Prevention study,[11] which analysis the death date of 15,355 patients with CHD, found that the level of TG was independently related to all-cause mortality of patients with CHD. A large sample cohort study with a follow-up of 15 years in many provinces and cities in China found that high TG was a predictor of CHD in low LDL-C population.[12] The 23-year follow-up results of Daqing study showed that the study evaluated the cardiovascular disease risk of 833 subjects, 34% of whom were HTG (baseline plasma TG level ≥ 1.7 mmol/L), and the cardiovascular disease risk in high TG group was 27% higher than that in non-high TG group. If the necessary level of TG increases by 1 mmol/L, the first cardiovascular disease risk increases by 8% in the following 20 years.[13]

In Hiroyasu *et al.* study, 55% were male, and 45% were female, the average age was 55.1 ± 6.3 years. In that study, also, the majority of patients were from the middle age group ($P < 0.05$).[14] In Hiroyasu *et al.* study, the mean BMI was 28.08. The mean WHR in normal PP4TG group was 0.968, and high PP4TG group was 1.028, so there is a strong correlation found between high WHR and high PP4TG. In Couillard *et al.* study, on postprandial TG response in visceral obesity showed that obesity and WHR are associated with impaired postprandial TG clearance.[2] In Hiroyasu *et al.* study, 52.1% of patients had diabetes.[15] A study was done by Axelsen *et al.*, on postprandial HTG and Type-2 diabetes showed postprandial lipid intolerance despite having normal fasting TG level and increased risk of macroangiopathy.[8] The mean PP4TG was 181.47 mg% ($P < 0.05$) which suggests that there is an association between coronary artery disease and PP4TG levels and

the relative risk was 1.75.[16] In Hiroyasu *et al.* study, 58% of male and 64% of female patients showed postprandial HTG ($P < 0.05$). In Nordestgaard *et al.* study on non-fasting TGs and risk of myocardial infarction, ischemic heart disease (IHD) and death in men and women showed that non-fasting TG levels independently predict myocardial infarction, IHD, and death.[17]

CONCLUSION

Postprandial HTG may be an independent risk factor for atherosclerosis in IHD patients. Evaluation of postprandial TG levels is essential during the assessment of IHD patients.

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