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Studying the Relation of Postprandial Triglyceride with Coronary Artery Disease (CAD)

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ABSTRACT

Background: Coronary artery disease (CAD) is the most common cause of mortality worldwide and determination of contributing factors is essential. **Aim:** This study was conducted to study the relation of postprandial triglyceride as a risk of coronary artery disease in patients with proven CAD by angiography, referred to 502 Hospital of Army in 2015. **Material and Methods:** This observational study conducted as a case-control and contained 80 male participants referred to 502 Hospital of Army. Half of these participants had proven CAD by angiography test and the other ones were healthy as a control group. Fasting serum triglyceride was evaluated in all participants and postprandial TG was checked 4 hours after a standard meal. Obtained data were analyzed by SPSS ver. 13. **Results:** The results indicated that fasting TG and postprandial TG level were significantly higher in CAD patients (P-value=0.001). It was also shown evaluation of postprandial TG is more sensitive test than fasting TG in case of CAD patients. **Conclusion:** Our obtained results shown, evaluation of high level of postprandial TG is more reliable than fasting TG for patients whom suffer from CAD.

Key words: Coronary artery disease (CAD), Postprandial triglyceride, Fasting triglyceride, Patient, SPSS.

1. INTRODUCTION

Cardiovascular disease (CAD) is the most common cause of death in the world. Before 1900 CAD infectious diseases and malnutrition were the most common cause of death and cardiovascular disease were included less than 10%. Currently, cardiovascular diseases cause 30% of deaths in the world and about 40% of these death occur in developed countries and about 28% of deaths in less developed countries (1). Current projections estimate that cardiovascular disease, especially atherosclerosis, will be the leading cause of all diseases in 2020 (2).

In 2013 CAD was the most common cause of death globally, resulting in 8.14 million deaths (16.8%) up from 5.74 million deaths (12%) in 1990 (3). The risk of death from CAD for a given age has decreased between 1980 and 2010 especially

in the developed world. The number of cases of CAD for a given age has also decreased between 1990 and 2010 (4). In the United States in 2010 about 20% of those over 65 had CAD, while it was present in 7% of those 45 to 64, and 1.3% of those 18 to 45. Rates are higher among men than women of a given age (5).

Cardiovascular risk factors divide into two groups. First, risk factors which are related to lifestyle and pharmaceutical modifiable ones such as, smoking cigarettes, blood pursuer (more than 140/90), HDL (lower than 40 mg.dL⁻¹), diabetes, obesity (BMI≥30kg.m²), and atherogenic diets. The second risk factors include unmodifiable ones such as sex, age (male≥45 and female≥55 years old), and cardiovascular disease familial history (1).

Coronary arteries clinical syndromes caused by chronic athero-

sclerosis of epicardial coronary arteries. This case is expected in all ages and both genders but its extent is related to genetic predisposition, risk factors, and local hemodynamic condition of individuals (6).

The endothelium vascular damage is an early event. Normal endothelium is a good mediator for vascular tone regulation. Endothelium produces several vasoactive substances such as prostacyclin and endothelium-derived relaxing factors and control local intravascular thrombosis in a complex way (7). High blood pressure, hypercholesterolemia, smoking cigarettes and local hemodynamic abnormalities can cause damage to the vascular endothelium. These factors disrupt endothelium-dependent vasodilatation and create favorable conditions for local thrombosis (8). Endothelial dysfunction is the earliest anomalies which is determined in atherosclerotic vascular. Endothelial damage leads to the accumulation of macrophages and lipids (often low-density lipoprotein) in the site of vessel injury (9).

Lipids play a major role in the development of atherosclerosis (10). Increasing of blood cholesterol (mainly LDL) promotes disease. Elevation of triglycerides may also be an independent risk factor for coronary artery disease (CAD), especially in women. On the other hand, high density lipoproteins (HDLs) seem to have a protective role and neutralize the effects of risk factors. Extensive investigations clearly have shown lipid lowering effect in primary and secondary prevention of CAD (11).

Triglycerides which contain a glycerol molecule and three fatty acids have insulation and energy saving role in fat tissues of human body. Total glyceride and/or LDL level more than 90% or HDL level lower than 10% indicates dyslipidemia (12). Triglycerides increase the risk of CAD by increasing the LDL level, decreasing HDL level, disrupting the function of artery walls, and activating the thrombogenic factors and plasminogen activators (13).

Recent studies indicated that fasting triglyceride concentration has low independent effect on risk of CAD. This phenomenon could be derived from lots of daily changes in plasma triglyceride concentrations and the presence of a strong reverse relation between the concentration of serum triglycerides and HDLs (14, 15). Changing the lifestyle and new therapeutic approaches and using old treatments can increase the life quality of individuals with CAD risk (16).

In a study which was conducted in Britain, fasting serum level of total cholesterol, ApoA-1, ApoB, triglyceride, LDL, and HDL were measured in 2508 healthy and young male persons (whiteout any heart disease) for 6 years. In 6 years follow up period, 163 of participants were stricken heart coronary diseases (include acute myocardial infarction, silent coronary artery bypass surgery and evidence of silent myocardial infarction in ECG). This study showed that the combination of increased ApoB and triglyceride as a well diagnostic criteria for coronary heart disease (17).

The postprandial triglyceride levels of proven CAD patients have been evaluated in this study to investigate the possibility of using postprandial triglyceride levels as a prognostic factor for CAD and also to find the relation of

these type of lipids with CAD. Determination of proper therapeutic strategy for lipids profile disorders is one of the benefits of this study.

2. MATERIAL AND METHODS

This observational study conducted as a case-control and contained 80 male participants referred to 502 Hospital of Army which were divided into two groups of case and control. 40 male participants of case group suffered from CAD which was proven by angiography. Control group also contained 40 male participants with normal angiography test result. Participants with risk factors, such as smoking cigarettes, high blood pressure (BP≥140/90), obesity (BMI≥30), diabetes, liver and kidney disorders and thyroid disease were control groups' exclusion criteria.

After coronary arteries angiography, fasting triglyceride were measured for all participants after 8-12 hours of fasting. Postprandial triglyceride also measured 4 hours after a standard meal for each participants (18, 19). A standard meal contains 50-55% carbohydrate, 30-35% lipid, and 15% protein. (15) Lower than 150 mg.dL⁻¹ and 200 mg.dL⁻¹ were considered as a normal concentration of triglyceride for fasting and postprandial period.

Demographic and other main variables entered in SPSS ver. 13 for statistical analysis. Independent T-test and chi-square were used to report the results. Quantitative and qualitative data were reported in the form of average and percentage of the distribution, respectively. 0.05 was considered as a significance level.

3. RESULTS

Obtained results of this study showed the mean age of control and case groups were 57±10.3 and 60.9±11.4, respectively. Statistical analysis did not show any significant differences between these two groups in their age and their age frequency were similar (P-value>0.05).

The mean concentration of fasting and postprandial triglyceride in case groups were significantly higher than control group (P-value=0.001). Furthermore, the amount of triglyceride concentration difference in fasting and postprandial period were significantly higher in case group (P-value=0.001) (Table 1).

Variable	Control group	Case group	P-value*
Fasting TG (mg/dL)	121.9±6.3	167±8.7	0.001
Postprandial TG (mg/dL)	197.3±7.4	284.5±1.5	0.001
Difference of fasting and postprandial TG	71.8±3.7	117.2±9.7	0.001

* Unpaired T-test was used

Table 1. The mean of fasting and postprandial TG in normal and CAD patients

The results indicated that the frequency of fasting TG abnormality in CAD patients were significantly higher than control group participants (P-value=0.001). The odd ratio was 8.8. Sensitivity and specificity of fasting TG test to diagnose CAD were 65% and 83%, respectively.

The frequency of postprandial TG in case group were also significantly higher than control group (P-value=0.001). The odd ratio was measured 28. Statistical

	Fasting TG			Postprandial TG		
	Control	Case	P-value*	Control	Case	P-value*
Normal TG	33 (83%)	14 (35%)	0.001	30 (75%)	5 (12%)	0.001
Abnormal TG	7 (17%)	26 (65%)		10 (25%)	35 (88%)	

* Chi-square was used for this results

Table 2- Frequency of fasting and postprandial TG in both groups

analysis of this parameter showed that Sensitivity and specificity of postprandial TG test to diagnose CAD were 88% and 75%, respectively (Table 2).

According to the ROC, changes in triglyceride levels greater than 80mg.dL⁻¹ was considered abnormal (sensitivity and specificity 75%) which it was significantly more in patients with CAD (Case group) than the control group. The odd ratio in CAD patients was 9 (Table 3).

	Control group	Case group	P-value
Normal	30 (75%)	10 (25%)	0.001
Abnormal	10 (25%)	30 (75%)	0.001

Table 3. Frequency of fasting and postprandial TG

4. DISCUSSION

In this study, the relation of postprandial triglyceride with the rate of coronary arteries disease was investigated in patients with proven CAD by angiography referred to 502 Hospital of Army in 2015. Moreover, these results show us a prevalence of serum triglycerides in CAD patients as well. In this study it was observed that the mean of fasting and postprandial triglycerides are significantly higher in patients with CAD than the control group. Furthermore, postprandial triglyceride abnormality was significantly more than fasting triglyceride in CAD patients. Our results illustrated that postprandial triglyceride has more association with CAD and this test had more sensitivity for CAD detection in comparison to fasting triglyceride measurement. These observations represent a higher value of postprandial triglyceride measurement rather than fasting triglyceride in association with CAD.

In a cohort study which was performed on 80 patients, fasting serum triglyceride levels were evaluated 2, 4, 6, and 8 hours after a fatty breakfast. This study showed the peak of serum triglyceride would be 4 hours after a meal. Same to our results, they showed a significant differences between fasting and postprandial triglyceride in CAD patients. This research also illustrated patients with high fasting triglyceride may have a high postprandial triglyceride which is associated with CAD, however postprandial triglyceride has no significant relation with CDA in patients with normal rate of fasting triglyceride (18). While in our study lack of association of high postprandial triglyceride was more than fasting one and its sensitivity were also higher.

In other study which concluded 61 CAD patients and 40 healthy people according to their angiography results, fasting triglyceride, low density lipoproteins (LDLs), high density lipoproteins (HDLs), apo-lipoprotein A and B and postprandial triglyceride after 2, 4, 6, and 8 hours after a meal were measured by standard tests. Their results showed more increase in triglyceride in CDA patients (15) which was same to our results.

In a study 2809 male patients were selected randomly to investigate the relation of fasting and postprandial triglyceride level with coronary heart disease (CHD) by multiple risk factor intervention trial. Proportional hazard regression models were used to study the association of fasting and postprandial triglyceride level with CHD. The mean level of fasting and postprandial triglyceride was 187 and 284 mg.dL⁻¹ respectively. The prevalence of hypertriglyceridemia (200 mg.dL⁻¹) was 31% and 61% for fasting and postprandial condition. Finally, they concluded that hypertriglyceridemia is more frequent in postprandial than fasting and evaluation of postprandial triglyceride level is more helpful (20) which is same to our results.

In a study performed in Iran, 60 male and female participants were divided into two equal groups. All participants were older than 35 years and without any history of diabetes, hypothyroidism, nephrotic syndrome, and lever disease. Triglyceride levels were determined in stress free condition in fasting period and 3 hours after a fatty breakfast. Fasting serum triglyceride in control and case group were 141±54 and 162±50 mg.dL⁻¹ respectively and statistical analysis did not show any significant difference between them. On the other hand, they showed significant difference between case and control group for postprandial triglyceride (21) which was same to our results.

5. CONCLUSION

According to our results and in contrast with other studies it could be concluded that evaluation of high level of postprandial TG would be a more reliable test than fasting TG to investigate the disease condition in patients whom suffer from CAD.

• Conflict of interest: none declared.

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