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ORIGINAL ARTICLE

Significance of Postprandial Triglycerides in Coronary Artery Disease

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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. Studies have demonstrated the association of severe anatomical coronary artery disease (CAD) with postprandial triglycerides (TG) concentrations. Nevertheless the relationship between less severe atherosclerosis plaque burden and postprandial TG is less established. The study was aimed to study the significance of postprandial triglyceride as predictor for the severity of coronary artery disease. **Methods:** The current study included one hundred and five individuals presented to Cardiology Department, Zagazig University Hospitals, within a time period of six months from July 2018 to December 2018, Inclusion criteria includes CAD based on coronary angiography. **Results:** In our study, ischemic heart disease is statistically higher in patients with abnormal CA and abnormal postprandial TG versus patients of abnormal CA with normal PP TG (51.9% versus 28.6%) respectively. **Conclusions:** According to our results, we concluded that a high level of postprandial TG would be a more reliable test than fasting TG in predicting the severity of CHD..

Keywords: Atherosclerosis; Triglycerides, Coronary artery disease (CAD), Postprandial triglyceride, Fasting triglyceride.

INTRODUCTION

Cardiovascular disease (CVD) causes death for 4 million subjects in Europe every year. It causes death for women [2.2 million (55%)] than men [1.8 million (45%)], and cardiovascular (CV) deaths under 65 years more prevalent in men (490 000 versus 193 000**)** [1]**.**

Endothelial dysfunction was the main cause of vascular atherosclerotic. The damage of Endothelial cause lipids and macrophages accumulation (mostly low-density lipoprotein) in vessel injury site [2] .

Lipids considered the major cause of atherosclerosis [3].

The increase of blood cholesterol (especially LDL) considered the main cause of the disease. High levels of triglycerides could be independent risk factor for coronary artery disease (CAD), particularly in women. Although, it was suggested that high level of density lipoproteins (HDLs) can prohibit these risk factors. Extensive examinations showed that lipid decrease the prevention of CAD in primary and secondary cases [4].

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The level > 90% of total glyceride and/or LDL and level > 10% of HDL confirm dyslipidemia [5].

High level of triglycerides increase the CAD risk due to LDL level increase, HDL level decrease, disturbance of the artery walls function, the activation of thrombogenic factors and plasminogen activators [6]

The postprandial triglyceride levels of confirmed CAD patients were evaluated in the current study to assess the use of postprandial triglyceride levels as a prognostic factor for CAD and the relation between lipids and CAD.

The aim of the study is to study the significance of postprandial triglyceride as predictor for the severity of coronary artery disease.

METHODS

This case-control study was conducted on 105 CAD patients based on coronary angiography who were admitted to Cardiology Department, Zagazige University, during the period from june 2018 to December 2018.

Written informed consent was obtained from all participants and the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Patients were divided into 3 groups:

Group I (control group): included 35 patients with normal coronary angiography and normal postprandial triglycerides.

Group II: included 35 patients with abnormal coronary angiography and high level of postprandial triglycerides more than 200mg/dl. They were divided into subgroups according to severity of coronary artery disease (mild, moderate and severe) and according to (Syntax score) .

Group III: included 35 patients with abnormal coronary angiography and normal postprandial triglycerides.

Inclusion criteria:

Inclusion criteria includes CAD based on coronary angiography,labratory and ECG evidence .

Exclusion criteria:

Patients with other co-morbidities diseases, such as liver and kidney disorders and thyroid diseases.

All patients were subjected to the following:

- Complete history taking (Age, gender and family history of the patient), special habits such alcohol intake, tobacco use (current, former or non-smoker).
- Complete clinical and physical examination.
- Fasting and postprandial lipid (Fasting triglycerides were measured for all participants after 8-12 hours of fasting. Postprandial triglycerides were also measured 4 hours after a standard meal for each participant. A standard meal contains 50-55% carbohydrates, 30-35% lipids, and 15% proteins. Lower than 150 mg/dl and 200 mg/dl were considered as a normal concentration of triglycerides for fasting and postprandial period.
- Serum triglyceride levels
- Coronary artery angiography (All patients underwent selective right and left coronary angiography through the femoral artery, or radial root).

Statistical analysis:

Data were collected, tabulated and analyzed by SPSS 20, software for Windows. The significance level was set at $P < 0.05$.

RESULTS

Table (1), showed that the age of the patients in group I ranged from 35-71 years with a mean of 55.43 ± 9.81years, they were 17 males and 18 females. While in group II the age of the studied patients ranged from 50-76 years with a mean of 60 ± 7.04 , all of them were males and age of the patients in group III ranged from 37-78 years with a mean of 57.8±13.79 years. There was a statistical significant difference between group II and group III regarding age and there was a high statistical difference between all groups according to sex distribution. **Table (2)**, shows distribution of co-morbid diseases among the

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studied groups, where ischemic heart disease was statistically higher in group III versus group II (51.9% Vs 28.6%) respectively, regarding chest pain there is high statistically significant difference between the all studied groups (65.7% , 40.0% & 14.3%) in group I , group II and group III respectively. **Table (3),** showed that there was no statistical significant difference between the 3 groups regarding smoking. **Table (4),** showed that fasting TG in group I ranged from (77.6-175) mg with a mean of 133.27±33.3 mg and in group II ranged from 51-190 mg with mean 112.47±45.16 mg while in group 3 ranged from (108-220) mg with a mean of 181.57±34.3 mg , with a high statistical difference between all groups regarding fasting TG, fasting cholesterol, fasting LDL and fasting HDL. **Table (5),** showed that there was a high statistical difference between all groups regarding Post prandial TG, Post prandial cholesterol and Post prandial LDL, but there was no statistical significant difference between group I and group II regarding Post prandial TG, post prandial cholesterol and Post prandial LDL. **Table (6),** showed that in group II (48.6%) of patient with severe cardiac lesion on CA, while in Group III (88.6%) of patients with severe CA, and there was a high statistical significant difference between all studied groups in the severity of CA according to syntax score.

Table 1. Sociodemographic characteristics of the studied groups

Item	Group I Normal CA Normal PP TG (N=35)		Group II Abnormal CA Normal PP TG (N=35)		Group III Abnormal CA Abnormal PP TG (N=35)		P-value
	No.	**%**	**No.**	**%**	**No.**	**%**	
Age (years)							
Mean ± SD	55.43 ± 9.81		60 ± 7.04		57.8±13.79		# P1=0.141
Median (Range)	55 (35 – 71)		57 (50 – 76)		55(37-78)		##P2=0.035* ##P3=0.728
Sex							
Male	17	48.6	35	100.0	26	74.3	P1=0.000*
Female	18	51.4	0	0.0	9	25.7	P2=0.000* P3= 0.002*

***P < 0.05 is significant.**

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Table 2. Co-morbid diseases among the studied groups.

Item	GroupI Normal CA Normal PP TG (N=35)		Group I Abnormal CA Normal PP TG (N=35)		Group III Abnormal CA Abnormal PP TG (N=35)		P-value
	No.	**%**	**No.**	**%**	**No.**	**%**	
Diabetes Mellitus	3	8.6	2	5.7	0	0.0	P1=0.229 P2=0.842 P3=0.151
Hypertension	9	25.7	8	22.9	9	25.7	P1=0.950 P2=0.780 P3=0.780
IHD	6	17.1	10	28.6	18	51.9	P1=0.007* P2=0.255 P3=0.050
DM+HTN	3	8.6	2	5.7	0	0.0	P1=0.229 P2=0.842 P3=0.151
DM+HTN+IHD	0	0.0	2	5.7	4	11.4	P1=0.119 P2=0.151 P3=0.393
HTN+IHD	3	8.6	5	14.3	4	11.4	P1=0.754 P2=0.452 P3=0.721
No co-morbid disease	11	31.4	6	17.1	0	0.0	P1=0.001* P2=0.163 P3=0.010*
Chest pain							
							P1=0.000*
	5	14.3	14	40.0	23	65.7	P2=0.000* P3=0.031*

***P < 0.05 is significant.**

Table 3. Smoking among the studied groups

Item	Group I Normal CA Normal PP TG (N=35)		Group II Abnormal CA Normal PP TG (N=35)		Group III Abnormal CA Abnormal PP TG (N=35)		P-value
	No.	**%**	**No.**	**%**	**No.**	**%**	
Smoking							
Not smoker	21	60.0	17	48.6	17	48.6	P1=0.210
Smoker	12	34.3	18	51.4	14	40.0	P2=0.164
Ex-smoker	2	5.7	0	0.0	4	11.4	P3=0.105

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Table 4. Fasting lipid profile among the study groups

Item	Group I Normal CA Normal PP TG (N=35)	Group II Abnormal CA Normal PP TG (N=35)	Group III Abnormal CA Abnormal PP TG (N=35)	P-value
Fasting TG				
Mean ± SD	133.27±33.3	112.47±45.16	181.57±34.3	P1=0.000* P2=0.049* P3=0.000*
Median (Range)	139.4(77.6-175)	104(51-190)	198.6(108-220)	
Fasting cholesterol				
Mean ± SD	140.9±25.49	143.86±31.26	197.5±28.9	P1=0.000* P2=0.613 P3=0.000*
Median (Range)	134(110-190)	145(86.3-200)	190(137-240)	
Fasting LDL				
Mean ± SD	89.07±11.09	86.8±23.69	113.19±22.9	P1=0.000* P2=0.814 P3=0.000*
Median (Range)	91(70-104)	87.4(39-134)	110(86.7-170)	
Fasting HDL				
Mean ± SD	48.26±5.92	43.2±9.61	36.17±4.69	P1=0.000* P2=0.000* P3=0.000*
Median (Range)	50(34.4-55)	44(31-73)	35(29.8-44)	

***P < 0.05 is significant.**

Table 5. Post prandial lipid profile among the study groups

Item	Group I Normal CA Normal PP TG (N=35)	Group II Abnormal CA Normal PP TG (N=35)	Group III Abnormal CA Abnormal PP TG (N=35)	P-value
Post prandial TG				
Mean ± SD	140.97±31.5	137.46±52.5	231.68±19.6	P1=0.000* P2=0.601 P3=0.000*
Median (Range)	140(79-177)	150(51-200)	240(207-260)	
Post prandial cholesterol				
Mean ± SD	146.86±24.35	154.4±30.51	210.7±33.4	P1=0.000* P2=0.338 P3=0.000*
Median (Range)	137(113-192)	154(107.5-210)	200(140.6-250)	
Post prandial LDL				
Mean ± SD	88.87±11.47	81.97±26.05	117.98±23.87	P1=0.000* P2=0.544 P3=0.000*
Median (Range)	95(75-106.5)	88(44-123)	115(87.9-178)	

***P < 0.05 is significant.**

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Table 6. Severity of CA according to syntax score among the study groups

severity of CA	Group I Normal CA Normal PP TG (N=35)		Group II Abnormal CA Normal PP TG (N=35)		Group III Abnormal CA Abnormal PP TG (N=35)		P-value
	No.	**%**	**No.**	**%**	**No.**	**%**	
Normal	35	100.0	0	0.0	0	0.0	P1=0.000*
Mild	0	0.0	9	25.7	4	11.4	P2=0.000*
Moderate	0	0.0	9	25.7	0	0.0	P3=0.001
Severe	0	0.0	17	48.6	31	88.6	

***P < 0.05 is significant.**

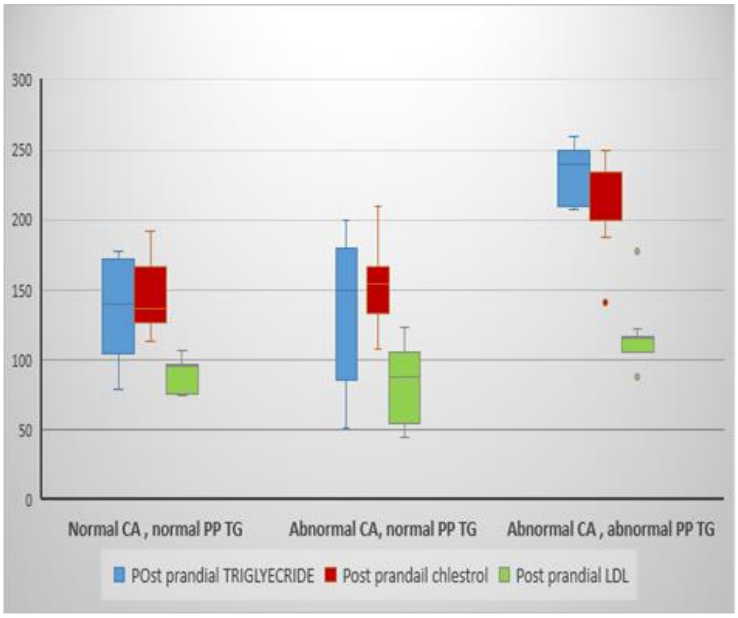

Figure 1. Box plot representing post prandial lipid profile among the study groups.

DISCUSSION

Lipids play a major role in the development of atherosclerosis. 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 **[3].**

Our result found , the ischemic heart disease was statistically higher in patients with abnormal postprandial TG versus patients with with normal PP TG (51.9% versus 28.6%) respectively. Regarding chest pain, there is a high statistically significant difference bewteen group I, group II and group III (65.7%, 40% and 14.3%, respectively). In the same context **Ariafar et al.** [7] found that measured nonfasting triglyceride level was significantly associated with an increased risk of myocardial infarction, ischemic heart disease, and mortality in the men and women. Recently, nonfasting triglyceride in anticipation of an increased risk

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of CAD as well as fasting triglyceride was considered important.

Also our study were in agreement with **Miller et al. [8]** indicated that fasting triglyceride concentration has small 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.

Our result showed that fasting TG in the studied normal CA, normal PP TG group ranged from 77.6 to 175 mg with mean of 133.4mg ± 33.3 mg and in the studied abnormal CA, normal PP TG group, it is ranged from 51 to 190 mg with a mean of 150mg. ± 45.16 mg; while in the abnormal CA, abnormal PP TG group, fasting TG ranged from 108 to 220 mg with a mean of 181.57±34.3 mg, with high statistical differences between all groups in fasting TG, fasting cholesterol, fasting LDL and fasting HDL. This results were in agreement with **Manochehri and Moghadam** [9] who found that the mean concentration of fasting triglyceride in patient group (these participants had proven CAD by angiography test) was significantly higher than control group. Furthermore, the amount of triglyceride level difference in fasting period was significantly higher in patient group. The frequency of fasting TG abnormality in CAD patients was significantly higher than control group. Sensitivity and specificity of fasting TG test to diagnose CAD were 65% and 83%, respectively.

Our study showed that postprandial TG in the studied normal CA, normal PP TG group ranged from 79 to 177 mg with mean of 140.97 ± 31.5 mg and in the studied abnormal CA, normal PP TG group, it is ranging from 51 to 200 mg with mean of 150 mg; while in the abnormal CA, abnormal PP TG group, fasting TG ranged from 207 to 260 mg with mean of 240 mg, with high statistical difference between all groups in postprandial TG, indicating that PP TG had a role in coronary artery disease. This was concordant with study done by **Manochehri and Moghadam** [9] indicated that the mean concentration of postprandial triglyceride in case groups was significantly higher than control group. Furthermore,Sensitivity and specificity of postprandial TG test to diagnose CAD were 88% and 75%, respectively.

In the same context **Ariafar et al. [7]** the results showed that the mean triglyceride serum level after a meal in patients with severe coronary artery disease (group 4) was higher than that in the other groups the patients were categorized based on the severity of their CAD according to the result of their angiography. The angiography results based on the vessel score method and the severity of CAD were divided into 4 general categories: normal patients (Group 1), patients with mild CAD (Group 2), patients with moderate CAD (Group 3), and patients with severe CAD (Group 4) .

In our result, about half of group II (48.6%) are moderate to severe (2 to 3 vesseles disease) cardiac lesions on CA, while most of group III patients with abnormal PP TG (88.6%) are with severe (3 or more vesseles disease) CA, there is high statistically significant difference between the 3 groups regarding severity of CA according to Syntax score. A logistic regression model for severity of CA was performed to ascertain the age, sex, chest pain, chronic illness, fasting triglycerides, fsating cholesterol, fsating LDL, fasting HDL, postprandial TG, postprandial cholesterol and postprandial LDL on the likelihood that participants have severe CA. The logistic regression model was statistically significant. The model explained 98% of the variance in severity of CA and correctly classified 98.1% of cases. Patients with elevated postprandial TG were 1.6 times more likely to exhibit severe coronary disease.

This was concordant with study done by **Uiterwaal et al.** [10]determined whether an increased familial risk for coronary artery disease in young adult men is related to changes in postprandial lipoprotein metabolism. They

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concluded that healthy young adult sons, whose fathers have established coronary artery disease, have prolonged postprandial hypertriglyceridemia. Changes in postprandial lipoprotein metabolism appear to be associated with familial risk for coronary atherosclerosis.

Similarly **Staniak et al.** [11]studied the relationship between postprandial TG and CAD detected by coronary computed tomographic angiography (CTA). They concluded that with mild (<25% lumen obstruction) and moderate CAD (25-50% lumen obstruction) detected by coronary CTA had an impaired postprandial metabolism, with a delayed TG clearance, when compared to individuals with no CAD. This difference was partially explained by the lower HDL-C. Thus, though postprandial TG may contribute to the development of CAD, this association is partially related to low HDL-C.

This was concordant with study done by **Manochehri and Moghadam** [9] who found that changes in triglyceride levels greater than 80 mg/dl was considered abnormal (sensitivity and specificity 75%) which it was significantly more in patients with CAD (case group) than the control group. They showed that evaluation of high level of postprandial TG is more reliable than fasting TG for patients whom suffer from CAD.

our result was in disagreement with a cohort study which was performed on 80 patients, **Atar et al.** [12] evaluated fasting serum triglyceride levels 2, 4, 6, and 8 hours after a fatty breakfast. They showed the peak of serum triglyceride would be 4 hours after a meal. They showed a significant differences between fasting and postprandial triglyceride in CAD patients. They 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 CAD in patients with normal fasting triglyceride.

Our result was disconcordance with study of **Werner et al.** [13] who compared risk prediction by fasting and postprandial serum triglycerides (TG) in patients with coronary artery disease (CAD). They concluded that fasting serum triglycerides >150 mg/dl predict cardiovascular events in patients with coronary artery disease on guideline-recommended medication. Assessment of postprandial TG does not improve risk prediction compared to fasting TG in these patients.

CONCLUSION

According to our results, we concluded that a high level of postprandial TG would be a more reliable test than fasting TG in predicting the severity of CHD.

Conflict of interest: Nothing to declare

Financial disclosure: Nothing to declare

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