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Original Research Paper

Relation between Postprandial Triglycerides and Coronary Artery Disease severity

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ABSTRACT:
Background: The main cause of cardiovascular disease, atherosclerosis, is more likely to occur in those with elevated serum triglyceride concentrations. Compared to fasting triglycerides, postprandial triglyceride concentrations have been demonstrated to be a more reliable indicator of cardiovascular disease. **Objective**: The study was aimed to study the relation between postprandial triglyceride and coronary artery disease severity. Patients and methods: The current study included one hundred and twenty individuals presented to Cardiology Department, Al Bayda Medical Center, Omar Elmokhtar University within a time period of six months from December 2023 to May 2024, Inclusion criteria includes CAD based on coronary angiography (CA). The patients was subjected to complete history taking,clinical examination and investigation of fasting and postprandial lipids profile. **Results**: ischemic heart disease was statistically higher in group III versus group II and there was high statistically significant difference between the all studied groups regarding chest pain. There was a high statistical difference between all groups regarding fasting TG, fasting cholesterol, fasting LDL and fasting HDL. There high statistical difference between all groups in Post prandial TG, Post prandial cholesterol and Post prandial LDL. there was a high statistical significant difference between all studied groups in the severity of CA according to syntax score. **Conclusions**: There is statistically a significant correlation between postprandial Triglyceride and coronary artery disease even in patients having normal fasting triglyceride level. It means that patients having high postprandial triglyceride levels have a higher risk of coronary artery disease and severity could be a future target in managing CAD patients

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

INTRODUCTION:

Atherosclerosis is the primary cause of coronary artery disease (CAD), which is characterized by angina pectoris, myocardial infarction (MI), and silent myocardial ischemia (1). Even though the condition's mortality rate is on the decline, it still accounts for almost one-third of fatalities among adults over 35 years. Dyslipidemia is established, modifiable risk factor of atherosclerosis [(2)].

Low-density lipoprotein cholesterol (LDL-c) and fasting plasma total cholesterol are the most effective indicators for estimating the risk of cardiovascular disease (CVD) [(3)]. Still, no LDL-C elevation has been detected in atherosclerotic patients, and this approach is still unable to predict around one-third of cardiac events. Moreover, an enhanced postprandial lipemic response was linked to an elevated risk of CVD in fasting normolipidemic participants [(4)].

Triglyceride-rich lipoproteins, such as chylomicron remnants (CMRs) and remnant lipoproteins, increase after meals, which is indicative of postprandial dyslipidemia. Because of its connection to cardiovascular events, it has gained importance recently. It has been demonstrated that CMRs can enter an artery and stay inside the intima [(5)].

Triglyceridemia is an independent risk factor for coronary artery disease irrespective of total cholesterol and LDL cholesterol or low HDL cholesterol. Prior to assessing lipid profile, ATP III guidelines2 recommend fasting for at least nine hours. Numerous research refuted this method. This relationship does cast doubt on the need to collect fasting lipoprotein values, even though it is not totally conclusive. The primary cause of death and disability in the industrialized world is coronary artery disease (CAD), which is brought on by atherosclerosis. There are established risk factors for CAD, including diabetes, hypertension, obesity, and sedentary lifestyles. It is yet unknown how blood triglyceride levels relate to the independent prediction of CAD. In addition, it remains

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to be determined whether fasting or nonfasting levels are informative for CAD risk. In practice, triglycerides are routinely measured in the fasting state [6].

However, because postprandial triglyceride-rich remnant lipoproteins can pass through the endothelial cell layer, live in the subendothelial space, and aid in the formation of foam cells a telltale sign of early atherosclerosis—postprandial lipids may be crucial to the pathophysiology of CAD. Higher peak concentrations or delayed clearance of triglycerides after meals may also indicate an aberrant response to an oral lipid load, which may indicate insulin resistance and predispose a person to coronary artery disease (CAD). More fatalities and disabilities than any other killer illness affect the Western population, both male and female, and heart disease is fast becoming the major cause of death and disability among Indians as well [7].

Endothelial dysfunction is typically the first step in atherogenesis, and it plays a role in the pathophysiology of CAD. Endothelial dysfunction is the consequence of postprandial hyperlipidemia, also known as hypertriglyceridemia, which produces pro-inflammatory cytokines, draws neutrophils, and causes oxidative stress (8). Postprandial dyslipidemia is currently not a goal in the management of dyslipidemia, despite data linking it to CAD occurrences [9].

AIM OF THE WORK:

The aim of the study was to study the relation between postprandial triglyceride and coronary artery disease severity.

PATIENTS AND METHODS:

This case-control study was conducted on 120 CAD patients based on coronary angiography who were admitted to Cardiology Department, Al Bayda Medical Center, Omar Elmokhtar University within a time period of six months from December 2023 to May 2024.

Written informed consent was obtained from all participants and the study was approved by the research ethical committee of Al Bayda Medical Center. 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.

Inclusion Criteria:

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

Exclusion Criteria:

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

Patients were divided into 3 groups:

Group I (control group): included 40 patients with normal coronary angiography and normal postprandial triglycerides.

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

Group III: included 40 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).

All patients were subjected to the following:

- A thorough history that includes the patient's age, gender, and family history; also, any unique behaviours like alcohol or tobacco usage (current, past, or non-smoker) should be noted.
- Complete clinical and physical examination.

Serum triglyceride levels:

- Postprandial lipid and fasting (All subjects had their fasting triglycerides assessed after 8–12 hours). Each participant's postprandial triglycerides were also assessed four hours following a typical meal. A typical meal is 15% proteins, 30%–35% fats, and 50%–55% carbs. Triglyceride concentrations less than 150 mg/dl and 200 mg/dl were regarded as normal for both the postprandial and fasting periods.

Serum triglyceride levels and classifications are as follows [10].

Less than 100 mg/dL - Optimal
101-150 mg/dL - Normal
150-199 mg/dL - Borderline
200-499 mg/dL - High
500 mg/dL or higher - Very high

Coronary artery angiography:

- All patients underwent selective right and left coronary angiography (CA) through the femoral artery, or radial root.

Statistical Analysis:

The data were loaded into the statistical package for the social sciences (SPSS version 20.0) program in order to be analyzed. The following tests were used to assess differences for significance based on the type of data: qualitative data, which is represented as a number and percentage, and quantitative data, which is represented as a mean ± SD group. Find the difference and relationship of the qualitative variable using the Chi square test (X2) paired with Mc Nemar or sign. P values were set at less than 0.05 and less than 0.001 for outcomes that were considered highly significant. Quantitative independent group differences using the Mann

Whitney or t-test, combined with paired t-tests and the Kruskal Wallis test To compare between more than two investigated groups for quantitative variables that are not regularly distributed. A significance criterion of $P < 0.05$ was establish.

RESULTS:

Table (1): Sociodemographic characteristics of the studied groups

Item	Group I (control group) Normal CA Normal PP TG (N=40)		Group II Abnormal CA Normal PP TG (N=40)		GroupIII Abnormal CA Abnormal PP TG (N=40)		P-value
	No.	%	No.	%	No.	%	
Age (years)							
Mean ± SD	52± 6.8		57 ± 8.2		54 ±12.7		# P1=0.221 ##P2=0.027* ##P3=0.564
Median (Range)	52 (35 – 65)		57 (45 – 70)		54 (40-75)		
Sex							
Male	21	52.5	35	87.5	29	72.5	‡ P1=0.000* P2=0.000* P3= 0.002*
Female	19	47.5	5	12.5	11	27.5	

Kruskal Wallis test:P1; for comparison of all groups
##Mann Whitney test: P2;Normal CA Normal PP TG and Abnormal CA Normal PP TG
P3; Abnormal CA Normal PP TG and Abnormal CA Abnormal PP TG
‡Chi-square test
*P < 0.05 is significant.

Table (1), showed that the mean age of the patients in group I ranged from 35-65 years with a mean of 52± 6.8years, they were 21 males and 19 females. While group II ranged from 45-70 years with a mean age of 57 ± 8.2, they were 35 males and group III ranged from 40-75 years a mean age of 54 ±12.7 years, they were 29 males and 11 females. 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): Co-morbid diseases among the studied groups.

Item	GroupI Normal CA Normal PP TG (N=40)		Group I Abnormal CA Normal PP TG (N=40)		Group III Abnormal CA Abnormal PP TG (N=40)		P-value
	No.	%	No.	%	No.	%	
Diabetes Mellitus (DM)	4	10	2	5	1	2.5	P1=0.229 P2=0.842 P3=0.151
Hypertension (HTN)	10	25	8	20	8	20	P1=0.950 P2=0.780 P3=0.780
Ischemic heart disease (IHD)	7	17.5	12	30	22	55	P1=0.007* P2=0.255 P3=0.050
DM+HTN	4	10	3	7.5	0	0.0	P1=0.229 P2=0.842 P3=0.151
DM+HTN+IHD	0	0	3	7.5	4	10	P1=0.119 P2=0.151 P3=0.393
HTN+IHD	4	10	6	15	5	12.5	P1=0.754 P2=0.452 P3=0.721
No comorbid disease	11	27.5	6	15	0	0.0	P1=0.001* P2=0.163 P3=0.010*
Chest pain	6	15	15	37.5	25	62.5	P1=0.000* P2=0.000* P3=0.031*

Table (2), shows distribution of co-morbid diseases among the studied groups, where ischemic heart disease was statistically higher in group III versus group II (55% Vs 30 %) respectively, regarding chest pain there is high statistically significant difference between the all studied groups (62.5% , 37.5% & 15%) in group I , group II and group III respectively.

Table (3):Fasting lipid profile among the study groups

Item	Group I Normal CA Normal PP TG (N=40)	Group II Abnormal CA Normal PP TG (N=40)	Group III Abnormal CA Abnormal PP TG (N=40)	P-value
Fasting TG				
Mean ± SD	130.27±35.2	109.50±42.9	178.6±39.7	P1=0.000* P2=0.039* P3=0.000*
Median (Range)	136.3 (73.2-170)	101 (49-185)	195.3(106-212)	
Fasting cholesterol				
Mean ± SD	137.8±27.49	140.9±33.75	194.5±35.7	P1=0.000* P2=0.613 P3=0.000*
Median (Range)	131 (105-1480)	142(3.8-190)	185 (129-230)	
Fasting LDL				
Mean ± SD	86.24±12.3	83.3±22.8	110.35±29.89	P1=0.000* P2=0.814 P3=0.000*
Median (Range)	88 (67-1102)	83.9 (36-131)	107 (82.8-172)	
Fasting HDL				
Mean ± SD	45.35±6.35	40.7±8.56	33.24±5.38	P1=0.000* P2=0.000* P3=0.000*
Median (Range)	48 (32.6 - 52)	42(29- 71)	33 (27.9 - 42)	

Table (3), showed that mean fasting TG in group I ranged from (73.2-170) mg with a mean of 130.27±35.2 mg and in group II ranged from 49-185 mg with mean 109.50±42.9 mg while in group 3 ranged from (106-212) mg with a mean of 178.6±39.7 mg , with a high statistical difference between all groups regarding fasting TG, fasting cholesterol, fasting LDL and fasting HDL.

Table (4):Post prandial lipid profile among the study groups

Item	Group I Normal CA Normal PP TG (N=40)	Group II Abnormal CA Normal PP TG (N=40)	Group III bnormal CA Abnormal PP TG (N=40)	P-value
Post prandial TG				
Mean ± SD	137.89±32.7	133.36±51.87	228.64±18.67	P1=0.000* P2=0.601 P3=0.000*
Median (Range)	136(75-170)	145(49-195)	240 (205 - 250)	
Post prandial cholesterol				
Mean ± SD	143.94±23.84	151.85±31.28	207.6±35.4	P1=0.000* P2=0.338 P3=0.000*
Median (Range)	135(112-190)	151(106.3-205)	202 (135-240)	
Post prandial LDL				
Mean ± SD	85.63±12.57	78.85±27.15	114.82±24.35	P1=0.000* P2=0.544 P3=0.000*
Median (Range)	92(70-107)	84(41-120)	112(85.9-175)	

This table shows that Post prandial TG in the studied Normal CA ,Normal PP TG group ranging from (75-170) mg with mean 137.89±32.7mg and in the studied Abnormal CA ,Normal PP TG group it is ranging from 49-195 mg with mean 133.36±51.87 mg while in the Abnormal CA , Abnormal PP TG group fasting TG ranging from (205-250) mg with mean 228.64±18.67 mg , with high statistical difference between all groups in Post prandial TG, Post prandial cholesterol and Post prandial LDL.

Table (5): Severity of CA according to syntax score among the study groups

severity of CA	Group I Normal CA Normal PP TG (N=40)		Group II Abnormal CA Normal PP TG (N=40)		Group III Abnormal CA Abnormal PP TG (N=40)		P-value
	No.	%	No.	%	No.	%	
Normal	40	100.0	0	0.0	0	0.0	P1=0.000*
Mild	0	0.0	12	30	5	12.5	P2=0.000*
Moderate	0	0.0	10	25	2	5	P3=0.001
Severe	0	0.0	18	45	33	82.5	

Table (5), showed that in group II (45%) of patient with severe cardiac lesion on CA, while in Group III (82.5%) 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 (6): logistic regression of severity of CA among the studied groups.

variables	**B** coefficient	S.E.	Wald	P-value	Exp (B)
Age	1.178	713.886	.000	.999	3.589
sex	-15.487	13420.541	.000	.999	.000
Chest pain	-10.190	25437.253	.000	1.000	.000
Fasting TG	-.511	278.508	.000	.999	.600
Fasting cholesterol	.341	1344.557	.000	1.000	1.406
Fasting LDL	.592	662.253	.000	.999	1.807
Fasting HDL	.148	1309.512	.000	1.000	1.159
Post prandial TG	.505	224.424	.000	.998	1.657
Post prandial cholesterol	.955	1186.658	.000	.999	2.598
Post prandial LDL	-1.992	666.787	.000	.998	.136
Constant	-162.315	74301.368	.000	.998	.000

R^2= 0.98, Chi-square test for model coefficient =139.24, P-value=0.000*

The table represents the best fitting logistic regression model for severity of CA, A logistic regression was performed to ascertain the age, sex, chest pain, fasting triglyceride, fasting cholesterol, fasting LDL, fasting HDL, post prandial TG, post prandial cholesterol, post prandial LDL on the likelihood that participants have severe CA. The logistic regression model was statistically significant,. The model explained 97.5 % of the variance in severity of CA and correctly classified 97.8 % of cases. Patients with elevated post prandial TG were 1.6 times more likely to exhibit severe coronary disease.

DISCUSSION:

Atherosclerosis is the primary cause of coronary artery disease (CAD), which is characterized by angina pectoris, myocardial infarction (MI), and silent myocardial ischemia1. Even while the condition's mortality rate is on the decline, it still accounts for almost one-third of all fatalities among adults over 352. One known, adjustable risk factor for atherosclerosis is dyslipidemia. [(11)].

Low-density lipoprotein cholesterol (LDL-c) and fasting plasma total cholesterol are the best biomarkers for predicting the risk of cardiovascular disease (CVD) (12). Still, no LDL-C elevation has been detected in atherosclerotic patients, and this approach is still unable to predict around one-third of cardiac events. Moreover, an enhanced postprandial lipemic response was linked to an elevated risk of CVD in fasting normolipidemic participants. Triglyceride-rich lipoproteins, such as chylomicron remnants (CMRs) and remnant lipoproteins, increase after meals, which is indicative of postprandial dyslipidemia. Because of its connection to cardiovascular events, it has gained importance recently. It has been demonstrated that CMRs can enter an artery and stay inside the intima [(13)].

This case-control study was conducted on 120 CAD patients based on coronary angiography who were admitted to Cardiology Department, Al Bayda Medical Center, Omar Elmokhtar University within a time period of six months from December 2023 to May 2024 to evaluate the significance of postprandial triglyceride levels as a predictor of severity of coronary artery disease.

The current study showed that the patients in group I were 19 female and 21 male, with a mean age of 52± 6.8 years, ranging from 35 to 65 years. Group II comprised 35 male patients with an average age of 57 ± 8.2, while group I consisted of 29 male patients with an average age of 54 ± 12.7 years, and 11 female patients. Age differences between groups II and III were statistically significant, and the distribution of sexes showed a strong statistical difference across all group.

Similarly to the present study **Ariafar et al.** [14] examined 416 patients, 234 of whom were men and 192 of whom were women, who had suspected CAD. 69 individuals (22 males and 47 females, ages on average of 50.9 and 53.6 years, respectively) made up type 1 group; 99 patients (51 males and 48 females, ages on average of 56.9 and 59.6 years, respectively) made up type 3 group; 83 patients (40 men, ages on average of 59.3 years and 43 women, ages on average of 62.8 years) made up type 4 group; and 165 patients (111 males and 54 females, correspondingly) made up type 2 and type 3 groups. Regarding age and sex, there was a statistically significant difference between the groups under study.

Our result found that Group III had a statistically greater rate of ischemic heart disease (55% vs. 30%) than group II. Regarding chest discomfort, there is a substantial statistical difference (62.5%, 37.5%, and 15%) across all the groups examined in group I, group II, and group III. Within the same framework, Ariafar et al. [14] discovered a strong correlation between measured nonfasting triglyceride levels and a higher risk of myocardial infarction, ischemic heart disease, and mortality in both males and females. Both fasting and nonfasting triglycerides were recently thought to be significant in advance of an elevated risk of CAD.

In the same line Khilar, [6] discovered that, following a meal, 71 out of 100 patients had serum triglyceride levels greater than 160 mg%. These findings show that patients with ischemic heart disease may have poor postprandial lipid metabolism even if their fasting triglyceride levels are acceptable. The relative risk was 1.45 and the mean two hours postprandial (PP2TG) was 165.3 mg% (P Value < 0.05), suggesting that there is a correlation between coronary artery disease and PP2TG levels.

Also our study were in agreement with Miller et al. [10] who showed that the concentration of triglycerides during fasting had a negligible independent impact on the incidence of CAD. This phenomenon may be caused by the numerous daily fluctuations in plasma triglyceride concentrations as well as the robust inverse relationship between serum triglyceride and HDL concentrations.

The current study showed that there was a significant statistical difference between all groups in terms of fasting TG, fasting cholesterol, fasting LDL, and fasting HDL. The mean fasting TG in group I ranged from 73.2-170) mg with a mean of 130.27±35.2 mg, in group II from 49-185 mg with a mean of 109.50±42.9 mg, and in group III from 106-212 mg with a mean of 178.6±39.7 mg.. This results were in agreement with Manochehri and Moghadam [15] who discovered that the patient group's mean fasting triglyceride content was noticeably greater than the control group's (these individuals had CAD confirmed by an angiography test). Additionally, the patient group's difference in triglyceride levels during the fasting phase was noticeably greater. Compared to the control group, CAD patients had a considerably higher frequency of fasting TG abnormalities. The fasting TG test's sensitivity and specificity for diagnosing CAD were 65% and 83%, respectively.

Also, Razik et al., [16] revealed that compared to the fasting level, there was a significant decrease in the postprandial level of total cholesterol (162.37 ± 45.86 vs. 168.26 ± 45.96 mg/dl; P = 0.03) and a significant increase in postprandial TGS, either TGs (154.30 ± 73.23 vs. 128.07 ± 69.40 mg/dl; P < 0.001) or very-low-density lipoproteins (VLDL) (30.85 ± 14.65 vs. 25.60 ± 13.93 mg/dl; P < 0.001).

Our study showed that TG group with a mean of 137.89±32.7 mg and in the Abnormal CA, Normal PP TG group, it is ranging from 49-195 mg with a mean of 133.36±51.87 mg. In the Abnormal CA, Abnormal PP TG group, fasting TG ranges from (205-250) mg with a mean of 228.64±18.67 mg. There was a significant statistical difference between all groups in postprandial TG, postprandial cholesterol, and postprandial LDL, suggesting a role for PP TG in coronary artery disease. This was concordant with study done by Manochehri and Moghadam[15] who showed that the case groups' mean postprandial triglyceride concentration was considerably greater than that of the control group. Additionally, the postprandial TG test's sensitivity and specificity for diagnosing CAD were 88% and 75%, respectively..

In the same context of the present study Ariafar et al. [14] revealed that after a meal, the mean serum level of triglycerides in patients with severe coronary artery disease (group 4) was greater than in the other groups. The patients were classified according to the severity of their coronary artery disease (CAD) based on the results of their angiography. Patients with mild CAD (Group 2), moderate CAD (Group 3), severe CAD (Group 4) and normal patients (Group 1) were the four basic categories into which the angiography results based on the vascular score technique and the severity of CAD were separated.

Similarly, Bansal et al., [17] found that there was a strong independent connection between non-fasting TG levels and cardiovascular events. TGs levels measured two to four hours after the meal were strongly associated with cardiovascular events in their secondary analyses based on time since the last meal (fully adjusted hazard ratio [95% confidence interval] for highest vs. lowest tertiles of levels, 4.48 [1.98–10.15] [P < 0.001 for trend]).

In contrast to our results, Kats et al. [13], declared that, even in their subgroups of race, obesity, and carotid atherosclerotic severity, there was no significant correlation between meal alterations and unexpected CVD occurrences.

The current study revealed that there was a significant statistical difference in the severity of CA according to syntactic score across all study groups for patients in Group II (45%) and Group III (82.5%) of patients with severe CA. Similary, Ikeno, et al., [18] discovered that

patients at increased risk of significant adverse cardiovascular events and reflecting more complex disease are those with higher SYNTAX scores.

Razik et al., [16] revealed that there was a substantial positive moderate connection between the SYNTAX score and the 2-hour postprandial VLDL ($r = 0.50$; $P < 0.001$) and TGs ($r = 0.55$; $P < 0.001$). Older age OR: 1.23(1.11-3.47; P<0.001), post-prandial VLDL 1.76 (1.50–3.49), and post-prandial triglyceride 2.34 (1.89–5.66) were significant predictors of high Syntax score.

In the present study, In order to determine the impact of age, sex, chest pain, fasting triglycerides, fsating cholesterol, fsating LDL, fasting HDL, postprandial TG, postprandial cholesterol, and postprandial LDL on the probability that individuals had severe CA, a logistic regression model for severity of CA was run. There was statistical significance in the logistic regression model. The model successfully identified 98.1% of cases and explained 98% of the variance in CA severity. Severe coronary disease was 1.6 times more common in patients with increased postprandial TG.

This was concordant with study done by Borén et al. [19] discovered that alterations in postprandial lipoprotein metabolism are associated with an elevated family risk for coronary artery disease in young adult men. It was determined that young adult males in good health who had fathers with established coronary artery disease exhibit prolonged postprandial hypertriglyceridemia. A hereditary risk for coronary atherosclerosis appears to be connected to alterations in postprandial lipoprotein metabolism.

Similarly Staniak et al. [11] examined the connection between coronary computed tomographic angiography (CAD)-detected postprandial TG (CTA). They came to the conclusion that, in comparison to people without CAD, those with mild (<25% lumen obstruction) and moderate (25–50% lumen obstruction) CAD identified by coronary CTA had poorer postprandial metabolism and a delayed TG clearance. The decreased HDL-C helped to explain part of this discrepancy. Therefore, even while postprandial TG may have a role in the development of CAD, low HDL-C may also play a role in this connection.

This was concordant with study done by Manochehri and Moghadam[15] discovered that triglyceride fluctuations more than 80 mg/dl were considered abnormal (sensitivity and specificity 75%), and that the case group of patients with CAD had considerably higher triglyceride changes than the control group. They demonstrated that for patients with CAD, assessment of a high level of postprandial TG is more dependable than fasting TG.

our result was in disagreement with a cohort study which was performed on 80 patients, Atar et al. [20] who assessed the levels of fasting blood triglycerides 2, 4, 6, and 8 hours following a high-fat breakfast. They demonstrated that four hours after a meal would be when serum triglyceride would peak. In CAD patients, they demonstrated a substantial difference in postprandial lipid levels and fasting triglycerides. Additionally, they showed that while postprandial triglyceride has no discernible relationship with CAD in patients with normal fasting triglyceride, it may be elevated in patients with high fasting triglyceride and high postprandial triglyceride, which is linked to CAD.

Our result was disconcordance with study of Werner et al. [21] who examined how individuals with coronary artery disease (CAD) were predicted to be at risk based on postprandial blood triglycerides (TG) and fasting. They came to the conclusion that in patients with coronary artery disease using medication prescribed by guidelines, fasting serum triglycerides greater than 150 mg/dl predict cardiovascular events. In these patients, measuring postprandial TG does not enhance risk prediction in comparison to measuring TG during fasting..

However, technological difficulty and the absence of well-established clinical protocols for evaluating postprandial lipemia have impeded the detection of postprandial hypertriglyceridemia in the clinical environment. However, there is currently sufficient data to support the continued advancement of routine postprandial and nonfasting TG assessments for both clinical and research applications. Even though there is currently no globally accepted treatment for postprandial hypertriglyceridemia, a prior study by Kolovou et al. (22) has recommended a straightforward clinical protocol for examining postprandial TG measurements and has highlighted cutoffs for exaggerated and delayed response (TG concentration, 2.5 mmol/L (220 mg/dL) at any time after an oral-fat tolerance test (OFTT) meal should be regarded as a desirable postprandial TG response; higher levels would be undesirable).

Limitations:

Our study has some limitations; the number of patients enrolled in our study is relatively small, but our results are comparable to those of more extensive studies. Furthermore, the study was observational and single institutional, which may have restricted us from identifying and analyzing all the potential confounding factors.

CONCLUSIONS:

There is statistically a significant correlation between postprandial Triglyceride and coronary artery disease even in patients having normal fasting triglyceride level. It means that patients having high postprandial triglyceride levels have a higher risk of coronary artery disease and severity could be a future target in managing CAD patients

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