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The relationship between lipid profile after fat loading and coronary artery disease severity assessed by SYNTAX score

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OriginalArticle

Abstract

BACKGROUND: Dyslipidemia is an established risk factor for coronary artery disease (CAD). Despite this, only half of CAD patients present with fasting dyslipidemia. Some reports have linked postprandial lipemia to atherosclerosis.

We aimed to test the relationship between postprandial lipid profile (after fat loading) and CAD severity assessed by the SYNTAX score.

METHODS: We included 53 patients with documented CADs. We checked both fasting and postprandial (2 hours) lipograms after fat loading with 17 g/body surface area (m2). Then we assessed CAD severity via coronary angiography using the SYNTAX score. Our study is registered in clinicaltrials.gov (NCT03175393).

RESULTS: *53 patients with age 57.92 ± 7.82 were recuirted. 36 (68%) of them were male .We observed a significant increase in postprandial triglycerides (TGs); TGs (154.30 ±73.23 vs. 128.07 ± 69.40 mg/dl; P < 0.001), very-low-density lipoproteins (VLDL) (30.85 ± 14.65 vs. 25.60 ± 13.93 mg/dl; P < 0.001) as well as a significant decrease in the postprandial level of total cholesterol (162.37 ± 45.86 vs. 168.26 ± 45.96 mg/dl; P = 0.03) in comparison to their fasting level.

We found that the SYNTAX score had a significant positive moderate correlation with 2-hour postprandial TGs (r = 0.55; P < 0.001) and 2-hour postprandial VLDL (r = 0.50; P < 0.001).

Based on the current study, predictors of high Syntax score were older age OR: 1.23(1.11-3.47); P< 0.001, post-prandial triglyceride OR: 2.34 (1.89-5.66); P< 0.001 and post-prandial VLDL OR: 1.76(1.50-3.49); P< 0.001

CONCLUSION: Postprandial lipograms, especially TGs, are significantly and positively related to CAD severity.

Keywords: Total cholesterol; Lipogram; Postprandial lipogram, SYNTAX score; Triglycerides

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Abbreviations

BMI: body mass index
CAD: coronary artery disease
CMRs: chylomicron remnants
CVD: cardiovascular disease
EF: ejection fraction
HDL-C: high-density lipoprotein cholesterol
LDL-C: low-density lipoprotein cholesterol
MI: myocardial infarction
TC: total cholesterol
TGs: triglycerides
TTE: transthoracic echocardiography
VLDL: very-low-density lipoproteins

Introduction

Coronary artery disease (CAD) includes the diagnoses of angina pectoris, myocardial infarction (MI), and silent myocardial ischemia and is related mainly to atherosclerosis(1). Despite the declining mortality for this condition, it still causes about one-third of all deaths in people older than 35 years(2). Dyslipidemia is an established, modifiable risk factor of atherosclerosis(3).

Fasting plasma total cholesterol and low-density lipoprotein cholesterol (LDL-c) cholesterol are the best biomarkers for predicting cardiovascular disease (CVD) risk (4). However, no LDL-C elevation has been found in patients with atherosclerosis, and about one-third of cardiac events remain unpredicted using this method. Furthermore, in fasting normolipidemic subjects, increased CVD risk was found to be associated with an exaggerated postprandial lipemic response (5).

Postprandial dyslipidemia is characterized by a rise in triglycerides (TGs)–rich lipoproteins, including chylomicron remnants (CMRs) and remnant lipoproteins after eating. Recently, it has become an important subject because of its association with cardiovascular events. CMRs have been shown to penetrate the artery wall and be retained within the intima(6).

Atherogenesis usually starts with endothelial dysfunction, which contributes to the pathogenesis of CAD. Postprandial hyperlipidemia (or hypertriglyceridemia) is responsible for producing pro-inflammatory cytokines, recruiting neutrophils, and generating oxidative stress, resulting in endothelial dysfunction(7). Despite evidence of its association with CAD events, postprandial dyslipidemia is still not a target in dyslipidemia management (8). This study aimed to test the relationship between lipid parameters before and after fat loading and SYNTAX score.

Methods

Study population

We performed this prospective observational study at a University Hospital between May 2018 and May 2019. The study included 53 patients with documented CAD. We included patients with documented CAD who were stable for three months and excluded those with recent acute coronary syndrome or familial dyslipidemia. Our study is registered in clinicaltrials.gov (NCT03175393).

We did the following for our study patients:

- Full history taking, including cardiovascular risk factors such as hypertension, diabetes mellitus, smoking, medications and any family history of dyslipidemia.
- Complete clinical examination, including body mass index (BMI) and waist circumference.

Echocardiography

Standard resting transthoracic echocardiography (TTE) was performed on all the patients using a General Electric VIVID S5 echo machine (GE, Horton, Norway). We calculated the ejection fraction (EF) using Simpson's rule as the difference between end-diastolic volume and end-systolic volume divided by end-diastolic volume. Resting wall motion abnormalities were detected using two-dimensional echocardiographic views.

Coronary angiography

Diagnostic coronary artery catheterization was performed on the patients based on positive noninvasive testing through either femoral or radial artery access. For those with significant disease progression, revascularization was done according to the usual practice. At least two independent cardiologists evaluated all the coronary angiographies. We calculated the SYNTAX score using the web-based calculator (http://www.syntaxscore.com/calculator/start.htm).

Fasting and non-fasting lipid profile

We checked the fasting (14 hours) and postprandial (2 hours) lipograms after fat loading with 17 g/

body surface area (m^2) (9). The samples were collected using traditional methods of antecubital venipuncture under aseptic conditions. The fasting and postprandial lipograms were done under standard clinical laboratory methods using the BT1500 fully automated clinical chemistry analyzer from Biotencica Indonesia. In addition, we assessed total cholesterol, HDL-C, and TGs via the direct method, and LDL-C was calculated using the Friedewald formula: LDL-C = total cholesterol (TC) − high-density lipoprotein (HDL-C) cholesterol − TGs/5 in mg/dl (10).

Ethical consideration

All the patients gave their informed consent after being briefed about the study's aims and process. The study procedures were free from any harmful effects on the participants as well as the service provided. The principal investigators have safely kept each individual's data private. Approval was obtained from our local ethical committee (approval number 170230).

Statistical analysis

Data was collected and analyzed by using SPSS (Statistical Package for the Social Science, version 20, IBM, and Armonk, New York). Quantitative data were expressed as mean ± standard deviation (SD) and compared with Student t test. Nominal data are given as number (n) and percentage (%). Chi2 test was implemented on such data.

Fasting and 2h-postprandial lipid profile were compared by Paired t test. Correlation of SYNTAX score with fasting and 2h-postprandial lipid profile were determined by Pearson correlation. Significant data between those with low and those with high SYNTAX score in univariate analysis were collectively used for further analysis by logistic regression to determine independent factors for high SYNTAX score. Level of confidence was kept at 95% and hence, P value was considered significant if < 0.05. The inter-observer agreement for SYNTAX score was calculated with weighted Kappa statistics.

Results

Baseline data

53 patients with age 57.92 ± 7.82 were recuirted and 36 (68%) of them were male.The baseline data of our patients is summarized in Table 1. We divided our patients into high SYNTAX (includs 19 patients) and low SYNTAX (includs 34 patients) groups, and our

Table 1. Baseline data of studied patients

	N= 53
Age (mean [SD], (range)	57.92 ± 7.82 (34-72)
Male gender	36 (68%)
Smoking	26 (49 %)
Diabetes mellitus	33 (62.3%)
Hypertension	33 (62.3%)
Family history of CAD	18 (34%)
Body mass index (kg/m^2) (mean [SD])	33.08 ± 3.88
Waist circumference (mean [SD])	100.90 ± 13.46
Body surface area (m^2) (mean [SD])	2.07 ± 0.15
Fasting blood sugar (mg/dl) (mean [SD])	97.88 ± 9.39
Therapeutic history	
Beta-blockers	53 (100%)
Acetyl salicylic acid	53 (100%)
Nitrates	53 (100%)
ACEI/ARBs	43 (81.1%)
Calcium channel blockers	11 (20.8%)
Clopidogrel	14 (26.4%)
Diuretics	3 (5.7%)
Ezetimibe	3 (5.7%)
Statins	38 (71.7%)
Fibrates	5 (9.4%)
Trimetazidine	5 (9.4%)

Data expressed as frequency (percentage), mean (SD), range. ACE: Angiotensin-converting enzyme, ARBS: Angiotensin receptor blockers, CAD: Coronary artery disease, ACS: Acute coronary syndrome.

Table 2. comparison between low and high SYNTAX groups

	SYNTAX score		*P* value
	Low (n= 34)	High (n= 19)	
Age (years)	57.74 ± 7.83	58.26 ± 8.06	0.81
Smoking	16 (47.1%)	10 (52.6%)	0.77
Hypertension	21 (61.8%)	12 (63.2%)	0.92
Diabetes mellitus	19 (55.9%)	14 (73.7%)	0.20
Statins	10 (29.4%)	5 (26.3%)	0.81
Fibrates	4 (11.8%)	1 (5.3%)	0.43
Weight (kg)	95.97 ± 13.08	96.37 ± 12.16	0.40
FBG (mg/dl)	96.50 ± 10.68	100.37 ± 6.17	0.91
Fasting total cholesterol (mg/dl)	167.50 ± 47.24	153.216 ± 41.29	0.09
Fasting triglycerides (mg/dl)	122.58 ± 62.59	137.89 ± 81.08	0.25
Fasting HDL-c (mg/dl)	108.94 ± 43.70	90.61 ± 34.33	0.12
Fasting VLDL-c (mg/dl)	24.52 ± 12.61	27.56 ± 16.22	0.09
PP total cholesterol (mg/dl)	175.03 ± 47.85	156.16 ± 40.79	0.48
PP triglycerides (mg/dl)	150.16 ± 71.29	161.7 ± 77.99	0.13
PP-HDL-c (mg/dl)	34.376 ± 6.06	31.716 ± 4.67	0.10
PP-LDL-c (mg/dl)	110.61 ± 40.78	87.91 ± 33.78	**0.04**
PP-VLDL-c	30.01 ± 14.27	2.35 ± 15.59	0.58

- Nominal data expressed as frequency (percentage) and compared by *Chi*^{2}-test while continuous data expressed as mean (SD) and compared by Student t test. *P* value was significant if < 0.05.
- FBG: fasting blood glucose; HDL-c: high density lipoprotein- cholesterol; LDL-c: low density lipoproteins-cholesterol; VLDL-c: very low density lipoproteins; PP: post-prandial

Table 3. Fasting and 2-h postprandial laboratory lipid profile.

	Fasting (n= 53)	2h-postprandial (n= 53)	*P* value
Total cholesterol (mg/dl)	168.26 ± 45.96	162.37 ± 45.86	0.03
Triglycerides (mg/dl)	128.07 ± 69.40	154.30 ± 73.23	< 0.001
LDL-c (mg/dl)	112.45 ± 41.21	101.45 ± 39.56	0.04
HDL-c (mg/dl)	34.26 ± 6.29	33.42 ± 5.70	0.88
VLDL-c (mg/dl)	25.60 ± 13.93	30.85 ± 14.65	< 0.001

Data expressed as mean (SD) and compared by Paired t test. *P* value was significant if < 0.05.
HDL-c: high density lipoprotein- cholesterol; LDL-c: low density lipoproteins-cholesterol; VLDL-c: very low density lipoproteins

threshold was 22(11, 12). The comparison between the two groups listed in Table 2. No significant differences between the two groups except post-prandial LDL was observed.

Fasting and postprandial lipograms

We noticed 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) in comparison to fasting level as well as a significant decrease in the postprandial level of total cholesterol (162.37 ± 45.86 vs. 168.26 ± 45.96 mg/dl; P = 0.03) in comparison to fasting level (Table 2).

SYNTAX score

Average SYNTAX score was 14.69 ± 9.78. Most of our patients were in the low SYNTAX (<22) category(11) (77% of patients). The SYNTAX score was found to have a positive significant moderate correlation with 2-hour postprandial TGs, either TGs (r = 0.55; P < 0.001) or 2-hour postprandial VLDL (r = 0.50; P < 0.001) (Table 3, Figure 1 and 2). The inter-observer agreement was calculated with weighted Kappa statistics and showed a good agreement (k = 0.93, P = 0.001).

Correlation of SYNTAX score with BMI and waist circumference

The SYNTAX score had no significant correlation with BMI (r = −0.07, P = 0.58) and waist circumference (r = −0.01, p = 0.91).

Correlation between triglycerides (TGs) and waist circumference

Waist circumference had no significant correlation

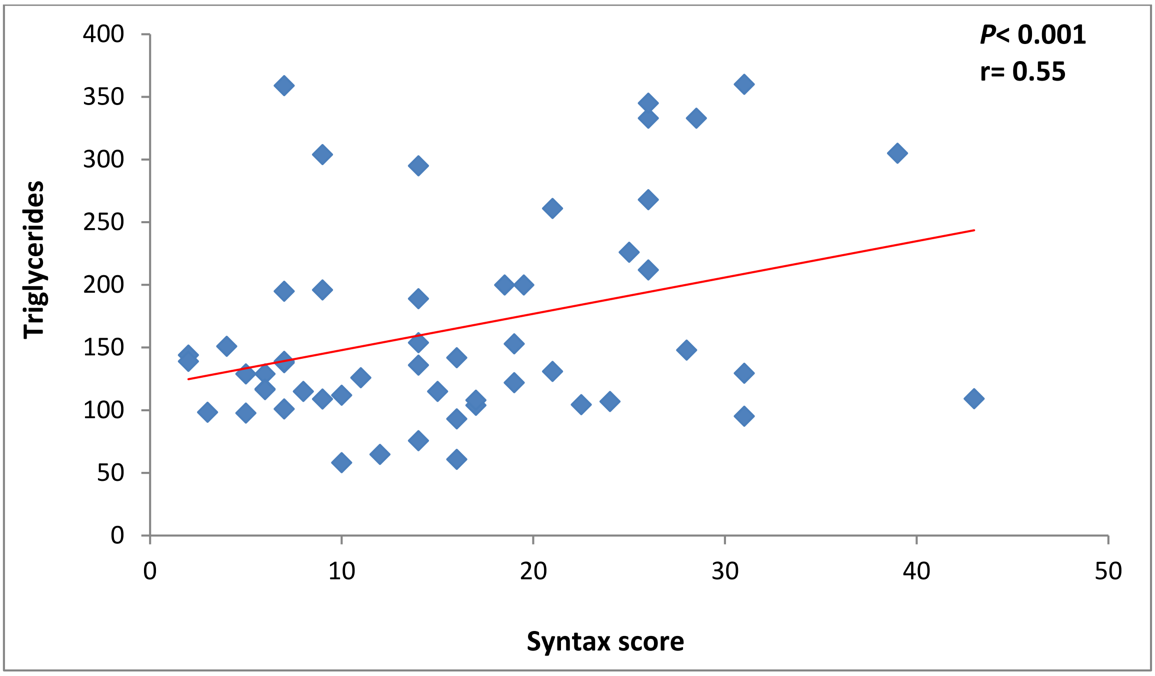

Figure 1. Correlation between SYNTAX score and postprandial triglycerides

with the TGs, either fasting (r = 0.05, P = 0.71) or postprandial (r = 0.03, P = 0.78).

Predictors of high SYNTAX scores

Using multiple regression analysis, age (OR: 1.23 (1.11-3.47); P< 0.001), post-prandial triglyceride (OR: 2.34 (1.89-5.66); P< 0.001) and post-prandial VLDL (OR: 1.76 (1.50-3.49); P< 0.001) were associated with syntax score in male .

Discussion

We tested the relationship between postprandial lipid profile (TGs, total cholesterol, VLDL, LDL-C, and HDL-C) and CAD severity assessed via SYNTAX score. Our results showed a significant increase in postprandial TGs compared with fasting level and a significant decrease in the postprandial level of total cholesterol and LDL-C compared with fasting level.

This finding was in agreement with Lund et al. regarding the postprandial decrement in LDL-C level; they showed that the LDL-C levels were lower in the non-fasting than in the fasting status 7.35 mg/dl using the direct method measurement of LDL-C cholesterol (13). Similarly, Nordestgaard et al. observed a transient drop in LDL-C concentration of 0.6 mmol/L (23 mg/dl) 1–3 hours after meals in diabetic patients. They favored the use of non-fasting rather than fasting lipid measurements. They found no significant clinical difference between fasting and non-fasting total cholesterol, HDL-C, and LDL-C levels. Moreover, they recommended the use of the non-fasting test in the follow-up of dyslipidemic patients (14).

On the other hand, Langsted et al. reported a minor increase in plasma TGs and a minor decrease in TC and LDL-C concentrations, with no change in HDL-C concentrations. They stated that these minor and transient changes in lipid concentrations appear to be clinically insignificant (15). Langsted et al. also explained that the postprandial increase of TGs is a response to regular food intake rather than fluid intake after a correction for albumin levels and hemodilution related to fluid intake (16). Lipid profiles change minimally in response to regular food intake. Therefore, the use of non-fasting lipid measurements in Copenhagen and Denmark as a standard was suggested with repeat fasting TGs measurement only if non-fasting concentrations exceed 4 mmol/l (352 mg/dl) (17).

Our results contradicted Sidhu and Naugler's findings, which included more than 2,000 individuals and showed minimal total cholesterol and HDL-C changes in the postprandial status compared with fasting level. However, they showed a more significant

Table 4. Correlation between lipid profile and SYNTAX score

	SYNTAX score with			
	Fasting		2h-postprandial	
	r value	*P* value	r value	*P* value
Cholesterol	0.01	0.06	0.11	0.07
Triglyceride	0.23	0.34	**0.55**	**< 0.001**
HDL	-0.23	0.22	-0.12	0.07
LDL	0.09	0.54	0.22	0.76
VLDL	0.13	0.12	**0.50**	**< 0.001**

*HDL: High-density lipoproteins, LDL: Low-density lipoproteins, VLDL: Very low-density lipoproteins.
*Significant correlations are labeled in bold (P<0.05).
*SYNTAX score is a web-based grading system that evaluates the complexity of coronary lesions.

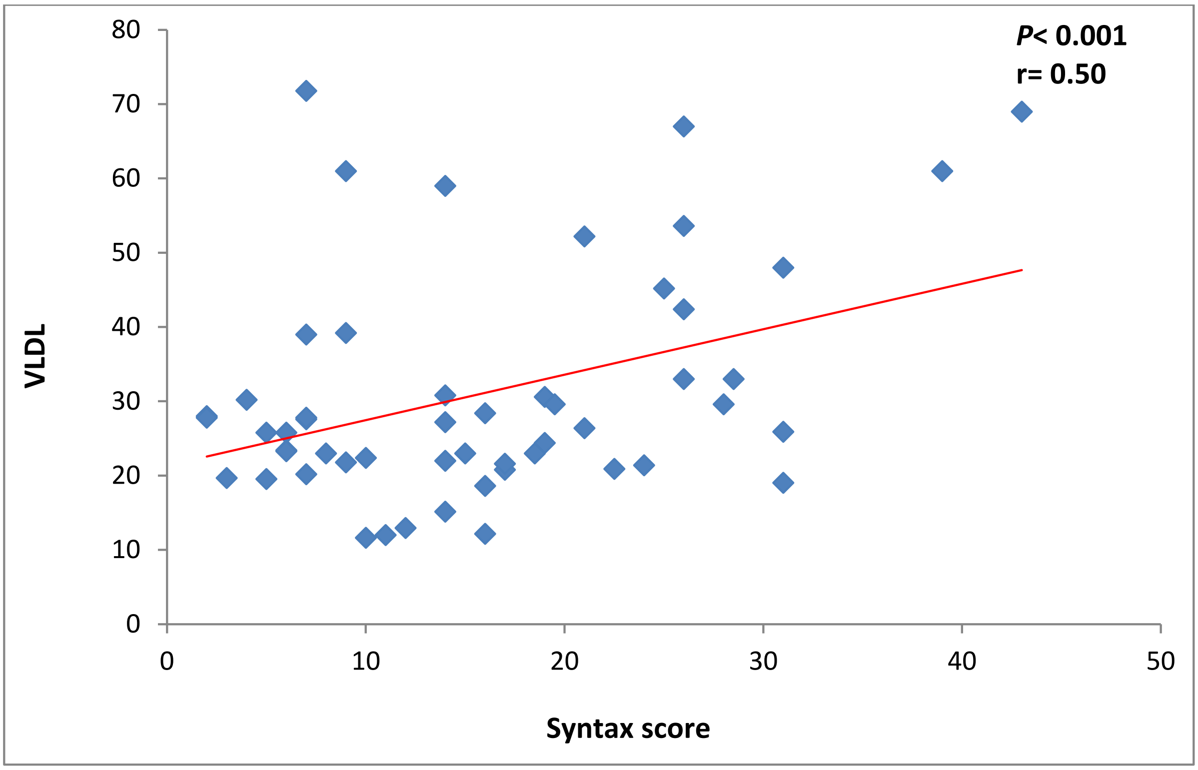

Figure 2. Correlation between SYNTAX score and postprandial VLDL

LDL-C variation by 10% in the general population and 20% in TGs levels. These results may be due to their larger sample size. Despite these results, they concluded that the fasting state showed little association with lipid subclass levels in a community-based population, suggesting that fasting for routine lipid levels was unnecessary (18).

In our study, the SYNTAX score had a positive significant moderate correlation with 2-hour postprandial TGs. In a recent study, Chatuverdi et al. found a positive association between TGs, either fasting or postprandial, and SYNTAX score categories. At the same time, they did not observe any changes in this LDL-C level, either fasting or postprandial (19).

According to our results, the SYNTAX score had an insignificant correlation with BMI and waist circumference (P > 0.05). In contrast, Ibrahim et al. showed that weight and BMI had a highly significant positive correlation with the SYNTAX score, with a highly significant statistical difference (20).
El Kersh et al. reported a weak positive correlation

between BMI and SYNTAX score (r [50] = 0.182, p = 0.091 (21)). This relation seems to depend on sample size as we recruited a similar sample size as El Kersh et al., but Ibrahim et al. recruited nearly 1,000 CAD patients.

The current study showed that the age, postprandial triglycerides, and postprandial VLDL-C were the significant predictors of high SYNTAX scores. In Copenhagen City Heart Study, they found that for men with non-fasting TGs < 1 mmol/l (89 mg/dl), their hazard ratios for MI and ischemic stroke increased for each 1 mmol/l increase in TGs, with the highest hazard ratios for the TGs level of 5 mmol/l (489 mg/dl) of 4.6 for MI and 3.2 for ischemic stroke. A similar increase in risk was observed in women with increasing non-fasting TGs, with the highest hazard ratios for TGs of 5 mmol/l (489mg/dl) of 16.8 for MI and 5.1 for ischemic stroke (22).

Similarly, the Women's Health Study demonstrated that non-fasting TGs levels kept a significant independent relationship with cardiovascular events. In their secondary analyses according to time since the last meal, TGs levels measured 2 to 4 hours after the meal were strongly associated with cardiovascular events (fully adjusted hazard ratio [95% confidence interval] for highest vs. lowest tertiles of levels, 4.48 [1.98–10.15] [P < .001 for trend]) (23).

Lindman et al. at the Norwegian Counties Study demonstrated that after an adjustment for CVD risk factors other than HDL-C, the hazard ratios per 1 mmol/ increase in non-fasting TGs were 1.16 (1.13–1.20) for all-cause mortality, 1.20 (1.14–1.27) for CVD, 1.26 (1.19–1.34) for IHD, and 1.09 (0.96–1.23) for stroke mortality in women. The corresponding figures in men were lower than those in women. In a subsample where HDL-C was measured (n = 40,144), the association between CVD mortality and TGs observed in women disappeared after HDL-C adjustment (24).

In contrast to our results, Dmitry Kats et al. stated no significant association between postprandial changes and unanticipated CVD events, even in their subgroups of race, obesity and carotid atherosclerotic severity (all p > 0.3) (25). Manochehri et al., in agreement with our study, found that fasting TGs and postprandial TGs levels were significantly higher in CAD patients (P = 0.001). They showed that postprandial TGs evaluation is a more sensitive test than fasting TGs in CAD patients. They also stated that evaluating a high level of postprandial TGs is more reliable than fasting TGs for CAD patients (26).

We noticed that waist circumference and BMI had an insignificant correlation with TGs, either fasting (P = 0.71, P = 0.36) or postprandial (P = 0.78, P = 0.45). As in our study, Sahade et al. stated that lipoproteins' behavior in the postprandial state is similar in eutrophic and overweight adolescents. Thus, weight excess does not induce postprandial lipemic alterations. The total increase in TGs, corresponding to the difference between the maximum and the basal TGs levels, was similar in both groups (overweight and eutrophic) (29.8 [21.5 mg/dl] vs. 28.2 [24.5 mg/dl], p = 0.762). TC, HDL-C, and LDL-C did not change significantly throughout the test (27). Contrary to our study, Schauren et al. stated that the BMI z-score was positively correlated with LDL-C and TGs and inversely correlated with HDL-C (28).

Our study noticed that patients on statins had significantly higher HDL-C and lower total cholesterol, LDL-C, TGs, and VLDL, either fasting or postprandial, than those who did not receive statins (P < 0.001). These results matched those of Collins et al. (2002), who showed the blood lipid differences between patients administered simvastatin and those administered placeboes with lower total cholesterol, LDL-C, and TGs levels fasting or postprandial. In contrast, the HDL-C level rose (29). Iso et al. (2001) reported that CAD incidence was more remarkable in a dose-response manner across increasing quartiles of non-fasting TGs levels (30).

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. Most of our patients were in the low SYNTAX score category. Finally, we did not demonstrate the effect of treating different modifiable risk factors on CAD lesion complexity.

Conclusion

We found a significantly positive correlation between postprandial TGs and VLDL and SYNTAX scores. Age, postprandial TGs and postprandial VLDL-C were the most significant predictors for high SYNTAX scores. At the same time, in the female subgroup, fasting VLDL was the only significant predictor.

Postprandial dyslipidemia, especially TGs, could be a future target in managing CAD patients.

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Confilict of interest

None-declared

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