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Original Article

A Study of Dyslipidemia Patterns and the Extent of Coronary Artery Disease

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ABSTRACT

Objective: This study aimed to identify dyslipidemia patterns and their correlation with the extent of coronary artery disease (CAD) in patients with a first ST-elevation myocardial infarction (STEMI).

Methods: This observational cross-sectional study was conducted from January 2023 through December 2023. A purposive, non-probability sampling technique was used to include 230 consecutive patients who presented to the emergency department of a tertiary care hospital with a primary diagnosis of acute STEMI. Demographics, symptoms, indicators, medical history, medication use, and diagnostic tests performed in the emergency department (e.g., ECG and chest X-rays) were recorded. Laboratory investigations, such as troponin-I and lipid profiles, were also extracted from the database.

Findings: The mean age of the study population was 60.39 ± 12.02 years. Of these, 164 (71.3%) patients were male. Multi-vessel disease (including double-vessel and triple-vessel CAD) predominated in the male population. Approximately 30 men in our study had combined hyperlipidemia with triple-vessel disease, followed by 23 men with isolated dyslipidemia and double-vessel disease. The SYNTAX score categories (low, intermediate, and high) demonstrated a statistically significant association with cholesterol levels (P <0.05).

Conclusions: We found that the presence of multi-vessel CAD coincided with various patterns of dyslipidemia, and mixed dyslipidemia was the most common pattern observed. ***(Iranian Heart Journal 2025; 26(4): 15-21)***

KEYWORDS: Dyslipidemia, Myocardial infarction, Coronary artery disease, Lipid profile

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Acute myocardial infarction (MI) is one of the most common causes of death worldwide and is more prevalent in the elderly population.[1] Despite significant advancements in recent years, the incidence and mortality of coronary artery disease (CAD) remain high.[1] Atherosclerosis, the primary pathophysiological cause of CAD, is closely linked to poorly managed lipid disorders.[2] The recommended treatment

for ST-segment elevation myocardial infarction (STEMI) is primary percutaneous coronary intervention. During index angiography, substantial stenosis is detected in one or more non-infarct-related arteries in up to 30% of these patients.[3]

Dyslipidemia constitutes a known risk factor for the onset of CAD in numerous clinical and epidemiological studies.[3] Low serum levels of high-density lipoprotein cholesterol (HDL-C) are a major risk factor for CAD.[4] Decreased HDL-C serum levels have been recognized as a significant risk factor for the emergence of CAD. Low-density lipoprotein cholesterol (LDL-C) and total cholesterol (TC) levels in the blood have also been linked to CAD development.[4]

The atherogenic process heavily depends on the subfractions of lipid particles. Small, dense LDL particles are more atherosclerotic than larger particles. HDL2 particles, which are larger and less dense, are considered protective, whereas HDL3 particles, which are smaller and denser, are thought to be atherogenic.[5] The former particles have an inverse correlation with blood triglyceride (TG) levels and small, dense LDL levels. [6]

Previous studies have reported that blood lipid indices, such as the TG/HDL-C ratio, apolipoprotein B/apolipoprotein A-I (Apo B/Apo A-I) ratio, TC/HDL-C ratio, or LDL-C/HDL-C ratio, have a better predictive value for CAD than a single parameter.[7] Recent research has also revealed a substantial correlation between blood lipid indices and the risk of metabolic syndrome (MetS) and cerebral atherosclerotic stenosis.[8] Combining lipid parameters allows for a comprehensive assessment of the balance between atherosclerosis and anti-atherosclerosis, as well as an accurate evaluation of lipid deposition.[9] Further, the Friesinger index derived from traditional coronary angiography has been shown to be one of the noninvasive indicators most significantly linked to the severity of CAD. [10]

Nevertheless, research on abnormal lipid levels as indicators of CAD severity in patients with a first STEMI is still scarce in the literature. Studies have shown, however, that young patients, in whom CAD is becoming more common, tend to have single-vessel disease (SVD) compared with older patients and present with more severe hypercholesterolemia.[11]

Accordingly, the purpose of this study was to evaluate the association between lipid profiles and the extent of coronary artery involvement (multi-vessel disease) in patients with a first STEMI.

Coronary heart disease (CHD) causes changes and disturbances in the function of other organs, such as the kidneys, blood vessels, and lungs.[5] Renal damage is one of the most important complications caused by CHD that can have long-term negative effects on these patients' lives. Renal disorders caused by CHD include a wide spectrum of conditions, such as glomerular and tubular dysfunction.[7] In patients with glomerular dysfunction, the glomerular filtration rate can decrease, and macro- or microalbuminuria may occur.[8] CHD can lead to glomerular enlargement, capillary dilation, mesangial cell proliferation, glomerular sclerosis, and dysfunction and loss of tubular integrity.[9] The duration of cyanosis and elevated hematocrit levels are considered the main contributors to nephropathy in patients with CHD.[10]

METHODS

This observational cross-sectional study was conducted from January 2023 through December 2023. A purposive, non-probability sampling technique was employed to enroll 230 consecutive patients who presented to the emergency department of Liaquat National Hospital, Karachi, with a primary diagnosis of STEMI. The study was approved by the institute's ethics and research committee. Patients were diagnosed

with STEMI based on clinical examinations and ECG. Serum troponin-I and lipid profiles were measured during index hospitalization.[12, 13] Fasting was not an exclusion criterion.

The four mutually exclusive dyslipidemias are defined in Table 1 by various HDL-C, LDL-C, and TG categories, with normolipidemia serving as a control group. Two independent cardiologists from the institute's catheterization laboratory conducted subjective analyses of all coronary angiograms via standard techniques.

The severity of CAD was assessed in multiple ways. First, the number of vessels affected was categorized as SVD (1 major coronary artery involved), double-vessel (2VD: 2 major coronary arteries involved), and triple-vessel (3VD: 3 major coronary vessels involved). Regarding luminal diameter narrowing, only vessels with a diameter of ≥1.5 mm and ≥50% stenosis in major coronary arteries were considered.[13] Additionally, angiograms were scored according to the SYNTAX score system. Calculation was performed using the software available (http://www.syntaxscore.com). Only coronary arteries with a diameter of 1.5 mm having lesions causing ≥50% stenosis were included in the calculation. Patients were divided into three SYNTAX score tertiles: a low-score group (≤22), an intermediate-score group (23–32), and a high-score group (≥33).[13]

Data were entered and analyzed using IBM SPSS, version 26.0, software. Means and standard deviations were calculated for numerical variables, and categorical variables were presented as proportions. The Kruskal-Wallis test was used to determine the association between the lipid profile and the number of stenotic vessels. The chi-square test was used to find the association between categorical variables, such as gender, smoking, hypertension, diabetes, and dyslipidemia. The *P*-value was considered significant at 0.05.

RESULTS

A total of 230 patients were enrolled in the study. The mean age of the study population was 60.39 ± 12.02 years. Of these, 164 (71.3%) patients were male. Approximately 52.2% of the participants were hypertensive, and 49.6% were diabetic. Only 24 (10.4%) patients in this study were smokers.

Regarding dyslipidemia, 83 (36.1%) patients had combined hyperlipidemia, followed by dyslipidemia compatible with MetS in 72 (31.3%) patients. Approximately 58 (25.2%) patients had SVD, 90 (39.1%) patients had 2VD, and 82 (35.7%)patients had 3VD. All baseline characteristics of the study population according to CAD are presented in Table 1.

A statistically significant difference was found in TG, HDL, and LDL levels among CAD categories (P <0.05) (Table 2). Notably, 3VD was most prevalent in patients with combined dyslipidemia (approximately 44 patients) and dyslipidemia compatible with MetS (approximately 26 patients). Nonetheless, 29 patients with isolated dyslipidemia had 2VD, and 14 patients with normolipidemia had SVD (Figure 1). A comparison between the lipid groups and the number of affected vessels with the severity of CAD showed significant between-group differences (P <0.001).

Table 1. Baseline characteristics of the study population

Characteristics	SVD (n=58)	2VD (n=90)	3VD (n=82)	*P*
Gender				
Male	44 (75.9%)	62 (68.9%)	58 (70.7%)	0.651
Female	14 (24.1%)	28 (31.1%)	24 (29.3%)	
Age (year)	60.86 ± 12.27	57.48± 12.11	63.24 ±11.13	0.006
Hypertension				
Yes	30 (51.7%)	48 (53.3%)	42 (51.2%)	0.959
No	28 (48.3%)	42 (46.7%)	40 (48.8%)	
Diabetes Mellitus				
Yes	24 (41.4%)	44 (48.9%)	46 (56.1%)	0.226
No	34 (58.6%)	46 (51.1%)	36 (43.9%)	
Smoking				
Yes	8 (13.8%)	12 (13.3%)	4 (4.9%)	0.121
No	50 (86.2%)	78 (86.7%)	78 (95.1%)	
Dyslipidemia				
Combined hyperlipidemia	10 (17.2%)	29 (32.2%)	44 (53.7%)	<0.001
Dyslipidemia compatible with MetS	22 (37.9%)	24 (26.7%)	26 (31.7%)	
Isolated dyslipidemia	12 (20.7%)	29 (32.2%)	10 (12.2%)	
Normolipidemia	14 (24.1%)	8 (8.9%)	2 (2.4%)	

SVD: single-vessel disease, 2VD: double-vessel disease, 3VD: triple-vessel disease, MI: myocardial infarction, LBBB: left bundle branch block, AWMI: anterior wall myocardial infarction, MetS: metabolic syndrome, VD: vessel disease

Table 2. Comparison of laboratory investigations with coronary artery disease severity in the study population

Investigation	SVD	2VD	3VD	*P*
Cholesterol (mmol/L)	187.22±86.93	195.32±76.73	197.67±78.39	0.06
TG (mg/dL)	77.29±45.58	159.60±57.27	191±77.29	0.01
HDL (mg/dL)	38.72±8.19	37.03±10.29	34.75±10.36	0.04
LDL (mg/dL)	155.85±62.71	170.05±37.84	175.35±41.05	0.009

SVD: single-vessel disease, 2VD: double-vessel disease, 3VD: triple-vessel disease, Trop-I: troponin, TG: triglyceride levels, HDL: high-density lipoprotein, LDL: low-density lipoprotein

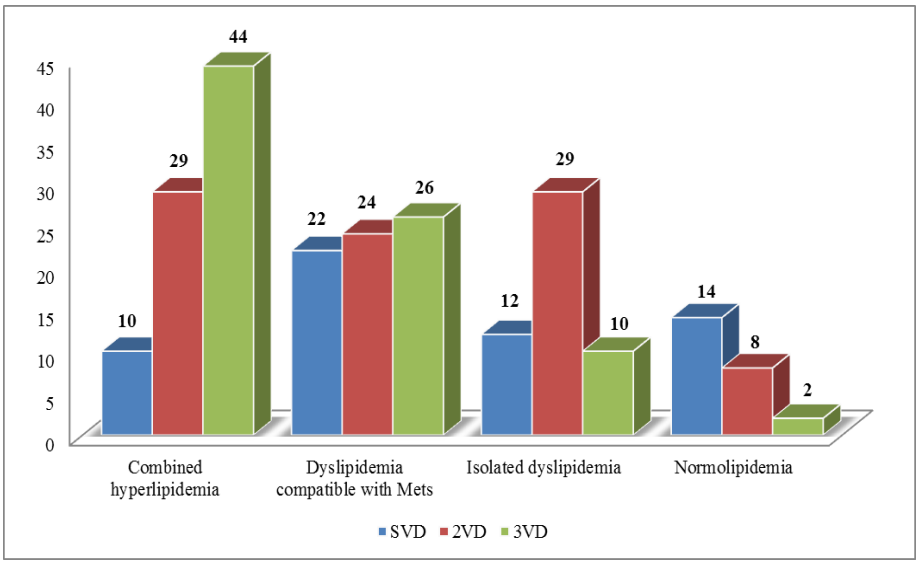

Figure 1. The image depicts the relationship between coronary artery disease severity and dyslipidemia types.

Mean cholesterol levels were 201.26 ± 82.27 mmol/L for the low SYNTAX score group, 179.29 ± 80.65 mmol/L for the intermediate group, and 205.58 ± 76.50 mmol/L for the high SYNTAX score group. A significant mean difference was observed in cholesterol levels across the SYNTAX score categories (*P* =0.006). Still, no significant mean difference was found for TG levels (*P* =0.160), HDL (*P* =0.260), or LDL (*P* =0.220) according to the SYNTAX score (Table 3).

Table 3. Association between laboratory investigations and the syntax score

Investigations	Low ≤22	Intermediate 23–32	High ≥33	*P*
Cholesterol (mmol/L)	201.26±82.27	179.29±80.65	205.58±76.50	0.006
TG levels(mg/dL)	169.58±74.39	189.04±61.38	182.30±44.25	0.16
HDL (mg/dL)	36.89±9.64	35.48±10.49	39.08±8.01	0.26
LDL (mg/dL)	163.52±60.21	168.58±43.17	173.24±33.51	0.22

TG: triglyceride levels, HDL: high-density lipoprotein, LDL: low-density lipoprotein

DISCUSSION

In our study, 30 men had combined hyperlipidemia with 3VD, followed by 23 patients with 2VD and isolated dyslipidemia. The mean SYNTAX score was 28.168 ± 6.727 in the combined hyperlipidemia group. A comparison between the lipid groups and the number of affected vessels concerning the severity of CAD showed significant between-group differences (*P* <0.001).

These results are consistent with recent studies that have focused on dyslipidemia as a risk factor for CAD and have investigated different components of the lipid profile. For instance, one study showed that, compared with older men, younger men who experienced MI had a high incidence of familial combined hyperlipidemia and increased levels of lipoprotein(a), whereas HDL-C levels were lower.[14] Similarly, some cross-sectional studies have reported significantly higher TG, LDL-C, and apolipoprotein B levels in younger compared with older MI patients, while HDL-C levels were lower.[15] Notably, these studies did not compare dyslipidemia with the severity and number of coronary arteries. The findings of our study suggest that individuals with low HDL and high TG are at greater risk of multi-vessel disease. This information may help identify specific groups of patients with STEMI who are at high risk of multi-vessel disease and could benefit from more extensive screening.

It is documented that South Asians have TC and LDL-C levels comparable to white individuals, but they do have higher TG and lower HDL-C levels.[16] A study conducted in Iran observed that patients with combined hyperlipidemia, simple hypercholesterolemia, and dyslipidemia of MetS had a statistically significant likelihood of leading to CAD than a normolipidemic reference group.[16] The present study shows similar results, with a higher proportion of patients with combined hyperlipidemia, dyslipidemia of MetS, and isolated dyslipidemia having CAD than patients with normolipidemia.

According to He et al.,[17] dyslipidemias cause CAD, mediated by inflammatory factors. Their observations suggested that the relationship between dyslipidemia and CAD appeared to be enhanced by increased high-sensitivity C-reactive protein levels, mediating approximately 8.27% of this effect. Tomizawa et al.[18] found that obstructive CAD was frequently increased among patients with dyslipidemia (15%; *P* <0.0001), and they reported a borderline positive correlation between

dyslipidemia and positive angiography (r =0.092; P =0.035).

There are, however, specific limitations to consider. First, because our study was cross-sectional, we succeeded in examining only lipid indicators and the stage of CAD as time-fixed factors. Another area of concern is the lack of information regarding predisposing and non-modifiable risk factors. Research suggests that combinations of many genes, including predisposing alleles, play a causal role in lipid level variations in populations regardless of participant lifestyle and age.[19] Future studies should investigate the prevalence and role of genetic factors in the association between dyslipidemia and CAD.

CONCLUSIONS

The findings from the present study demonstrated a significant relationship between lipid quartiles and the extent and severity of CAD, as assessed by the Friesinger index. Combined hyperlipidemias were most associated with multi-vessel disease involvement. Furthermore, according to age and gender, the severity of CAD was higher in men and more prevalent in the elderly population presenting with STEMI.

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