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Relationship of LDL and HDL Among Patients Suffering Acute Myocardial Infarct

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ABSTRACT: ***Background:*** Regardless of underlying hyperlipidaemia or dyslipidemia, the serum lipid profile exhibits phasic fluctuations immediately following a catastrophic cardiovascular event. The clinical management of dyslipidemias in patients experiencing an acute myocardial infarction (MI) has evolved significantly over the past decade. ***Objective:*** To examine the connection between LDL and HDL among patients suffering from sudden myocardial infarction. ***Materials and Methods:*** The cross-sectional study conducted in the department of cardiology, TMSS Medical College Hospital, from May 2024 to April 2025. All the patients admitted with ST-elevation MI (STEMI) were included after receiving informed consent. STEMI was diagnosed by pertinent history, electrocardiogram (ECG), and cardiac biomarkers. Patients those already taking lipid- lowering drugs, who presented after 24 hours of MI, and who had previously been diagnosed with hyperthyroidism were excluded in this study. ***Results:*** The lipid profiles of the patients were compared at 2 different time instances (within 24 h vs. after 48 h). The frequency of isolated deranged TGs, and deranged combined TGs and HDL-C, was statistically significant after 48 h of acute MI. The mean levels of serum TC (213.75 ± 29.15 vs 191.64 ± 37.13), serum LDL- C (146.52 ± 36.41 vs 135.24 ± 34.7), and HDL-C (45.96 ± 8.96 vs 41.73 ± 10.78) decreased significantly, whereas serum TGs (157.48 ± 15.12 vs 173.8 ± 16.23) increased significantly. ***Conclusion:*** Patients with acute coronary syndrome must have their serum lipid profiles evaluated during the first 24 hours of admission. Although there may be a brief period of phasic fluctuation, the pattern that follows is one of lower TC, LDL, and HDL and greater TGs.

Keywords: Serum Triglyceride, LDL, Lipid Profile, Acute Myocardial Infarction, Serum Cholesterol.

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INTRODUCTION

Cardiovascular disease is one of the leading causes of death worldwide, and hypercholesterolemia is a standalone risk factor for numerous cardiovascular illnesses (CVDs) and disorders, including myocardial infarction, peripheral vascular diseases, and coronary heart diseases.[1] Acute myocardial infarction (AMI) is by far the most important form of ischemic heart disease (IHD), and it alone is the leading cause of death in the

United States (US). About 1.5 million individuals in the US suffer from acute MI annually and approximately one-third of them die.[2] The severity of IHD and its resultant mortality can be reduced by controlling modifiable risk factors. Among the modifiable risk factors of IHD--hypertension (HTN), diabetes mellitus (DM), cigarette smoking, dyslipidemia, and severe obesity--dyslipidemia (hyperlipidemia and hypercholesterolemia) has been given the greatest attention recently.[3] Epidemiological surveys have shown that atherosclerosis due to dyslipidemia is directly correlated with a risk of IHD. Coronary artery disease (CAD) has been directly linked to hypercholesterolemia, particularly elevated plasma levels of cholesterol in low-density lipoproteins (LDL-C).[4,5] Increased risk of AMI has been seen in patients with low plasma levels of high-density lipoprotein (HDL-C) cholesterol.[6]

The clinical management of dyslipidemias in patients experiencing an acute myocardial infarction (MI) has evolved significantly over the past decade.[7] Hypercholesterolemia is one of the major cardiovascular risk factors and plays a key pathophysiological role in the development of acute ST-elevation myocardial infarction (STEMI).[8] Inflammation, endothelial dysfunction, and increased thrombogenicity as well as plaque vulnerability are crucial underlying mechanisms explaining the complex interplay between cholesterol metabolism and STEMI.[8] The disease is characterized by hypertension, high LDL (bad) cholesterol, low HDL (good) cholesterol, or diabetes.[9] The main causes of coronary artery disease are smoking, high-fat diet, diabetes, high cholesterol, hypertension, and obesity. Drugs that block newly synthesized cholesterol are used to rapidly regulate cholesterol levels and reduce dietary cholesterol intake.[9] Current study evaluated the impact on cardiovascular outcome of the systematic introduction in our institution of a personalized strike early and strong (SES) approach for lipid-lowering therapy (LLT) in patients admitted for acute myocardial infarction (MI).

MATERIALS AND METHODS

It was a prospective, cross-sectional study conducted simultaneously in the department of cardiology, TMSS Medical College Hospital, Bagura, Bangladesh, from May 2024 to April 2025. All the patients admitted with ST-elevation MI (STEMI) were included after receiving informed consent. STEMI was diagnosed by pertinent history, electrocardiogram (ECG), and cardiac biomarkers. Those patients already taking lipid-lowering drugs, who presented after 24 hours of MI, and who had previously been diagnosed with hyperthyroidism were excluded in this study. The reason for excluding patients with these comorbidities was to reduce the bias in the results. Along with all other forms of biochemical testing, their lipid profile (in mg/dl) was also routinely evaluated as a hospital protocol. This study did not make any additional interventions and did not cause any extra burden on the patient or hospital resources.

The patient's age, gender, smoking history, co-morbidities including diabetes mellitus and hypertension, previous history of major cardiovascular events (MACE including MI and stroke), and body weight and height were noted for all patients. The body mass index (BMI) was calculated and BMI >30 kg/m2 were termed as 'obese'. Lipid profile within the first 24 hours of the event and after 48 hours was recorded. There were five patients who died before 48 hours of admission and two were shifted to another hospital. All seven patients were replaced with new patients. Statistical analysis was done using SPSS v. 23.0 (IBM Corporation, Armonk, New York, United States). Continuous variables including age and lipid profile were analyzed via descriptive statistics and were presented as mean and standard deviation (SD) while categorical variables including gender, cardiovascular history, smoking history, and the type of lipid abnormality were presented by percentages and frequencies.

RESULTS

Table 1: Demographic Characteristics of the Patients (n=130)

Patient Characteristics	Frequency (%)	Percentage
Age in years		
25-40	12	9.23
41-50	35	26.92
51-60	66	50.77

61-70	17	13.08
Mean ±SD	54.17±11.62	Range 25-70 years
Gender		
Male	92	70.77
Female	38	29.23
Co-morbidities		
Hypertension	39	30.00
Diabetes mellitus	35	26.92
Diabetes + hypertension	42	32.31
Obesity (BMI >30 kg/m2)	60	46.15
Previous history of MACE	22	16.92

The mean age of the patients was 54.17±11.62 years. There were more men than women (70.77% vs. 29.23%). The details of patient characteristics including co-morbidities, the presence of obesity 60(46.15%), Hypertension 39(30%), Diabetes mellitus 35(26.92%), Diabetes + hypertension 42(32.31%) and previous history of MACE are shown 22(16.92%).

Table 2: Biochemical Lipid Profile (mg/dl) of the Patients Within the First 24 hours Compared with 48 hrs of STEMI

Parameters of Lipid Profile	**Two-time intervals**		**p value**
	Within 24 h	After 48 h	
Serum total cholesterol (mg/dl)			
Elevated	43 (33.08)	48 (36.92)	0.51
Non-elevated	87 (66.92)	82 (63.08)	
Serum triglyceride (mg/dl)			
Elevated	86 (66.15)	102 (78.46)	0.02s
Non-elevated	44 (33.85)	15 (11.54)	
Serum LDL-C (mg/dl)			
Elevated	40 (30.77)	47 (36.15)	0.35
Non-elevated	90 (69.23)	83 (63.85)	
Serum HDL-C (mg/dl)			
Decreased	44 (33.85)	43 (33.08)	0.89
Non-decreased	86 (66.15)	87 (66.92)	
Combine deranged TGs and HDL (mg/dl)			
Elevated	25 (19.23)	40 (30.77)	0.03 s
Non-elevated	105 (80.77)	90 (69.23)	

The biochemical lipid profile of the patient was compared over the first 24 hours and 48 hours following the myocardial infarction. On an isolated level, the most commonly deranged parameter was serum TGs (66.15% had elevated TGs within 24 hours and 78.46% had elevated TGs after 48 hours of the myocardial infarction), followed by 33.08% vs 36.92% patients who had elevated TC, 30.77% vs 36.15% who had elevated LDL-C, and decreased HDL-C 33.85% vs 33.08% within 24 hours and after 48 hours, respectively. Combine disturbed TGs and HDL (mg/dl): 19.23% vs 30.77% ($p<0.05$). Similar observational was found Kumar *et al*.[2] they also reported the patients were compared at both time instances (within 24 h vs. after 48 h). The frequency of isolated deranged TGs, and deranged combined TGs and HDL-C, was statistically significant after 48 h of acute MI. The mean serum levels of serum TC, serum LDL- C, and HDL-C decreased significantly, whereas serum TGs increased (Table 2).

Table 3: Comparison of mean of biochemical lipid profile (mg/dl) of the patients within the first 24 h and after 48 h

Parameters of Lipid Profile	Two-time intervals		p value
	Within 24 h	After 48 h	
Serum TC	213.75 ± 29.15	191.64 ± 37.13	<0.0001
Serum TGs	157.48 ± 15.12	173.8 ± 16.23	<0.0001
Serum LDL-C	146.52 ± 36.41	135.24 ± 34.7	0.0111
Serum HDL-C	45.96 ± 8.96	41.73 ± 10.78	0.0007

The lipid profiles of the patients were compared at both time instances (within 24 h vs. after 48 h). The frequency of isolated deranged TGs, and deranged combined TGs and HDL-C, was statistically significant after 48 h of acute MI. The mean serum levels of serum TC (213.75 ± 29.15 vs 191.64 ± 37.13), serum LDL- C (146.52 ± 36.41 vs 135.24 ± 34.7), and HDL-C (45.96 ± 8.96 vs 41.73 ± 10.78) decreased significantly, whereas serum TGs (157.48 ± 15.12 vs 173.8 ± 16.23) increased respectively as shown in Table 3.

DISCUSSION

In this study, it was observed that the mean age of the patients was 54.17±11.62 years. There were more men than women (70.77% vs. 29.23%). The details of patient characteristics including co-morbidities, the presence of obesity 60(46.15%), Hypertension 39(30%), Diabetes mellitus 35(26.92%), Diabetes + hypertension 42(32.31%) and previous history of MACE are shown 22(16.92%). Similar observation was found in Kumar *et al*. they reported there were more men than women (75.6% vs. 24.4%).[2] The mean age of the patients was 53.8 ±10.2 years. The details of patient characteristics including their ages, co-morbidities, the presence of obesity, and previous history of MACE. The biochemical lipid profile of the patient was compared over the first 24 hours and 48 hours following the myocardial infarction. On an isolated level, the most commonly deranged parameter was serum TGs (66.15% had elevated TGs within 24 hours and 78.46% had elevated TGs after 48 hours of the myocardial infarction), followed by 33.08% vs 36.92% patients who had elevated TC, 30.77% vs 36.15% who had elevated LDL-C, and decreased HDL-C 33.85% vs 33.08% within 24 hours and after 48 hours, respectively. Combine disturbed TGs and HDL (mg/dl): 19.23% vs 30.77% ($p<0.05$). Similar observational was found in Kumar *et al*.[2] They also reported the patients were compared at both time instances (within 24 h vs. after 48 h).

The frequency of isolated deranged TGs, and deranged combined TGs and HDL-C, was statistically significant after 48 h of acute MI. The mean serum levels of serum TC, serum LDL- C, and HDL-C decreased significantly, whereas serum TGs increased. The common, isolated forms of lipid irregularity were low HDL-C (17.3%) and high triglyceride (TG) (11.2%).[10] Both of these parameters have been regarded as linear risk factors for CAD and stroke.[11,12] In a study with young patients of AMI (within 24 hours), it was seen that 60.83% were dyslipidemic; the most common isolated deranged lipid fraction was TG (45%) whereas low HDL was least common (10.83%).[13] In this study observed that the mean serum levels of serum TC (213.75 ± 29.15 vs 191.64 ± 37.13), serum LDL- C (146.52 ± 36.41 vs 135.24 ± 34.7), and HDL-C (45.96 ± 8.96 vs 41.73 ± 10.78) decreased significantly, whereas serum TGs (157.48 ± 15.12 vs 173.8 ± 16.23) increased respectively. The lipid profile must be assessed in every patient admitted with acute coronary syndrome within the first 24 hours and then periodically until a steady healthy state is achieved. The change within the first 24 hours is minimal, and then phasic changes follow. Hence, the first measurement can serve as a relatively reliable source to inform the selection of the lipid-lowering therapy. However, correctly recognizing the baseline is still difficult. Since a decreasing trend in TC, LDL, and HDL is seen periodically after MI, lipid-lowering therapy must be initiated even if the results are within the physiological range in the first few days.[14]

CONCLUSIONS

Serum lipid profile evaluation must be required within the first 24 hours of admission for patients with acute coronary syndrome. A brief period of phasic fluctuation may be seen, but the trend that follows consists of higher TGs and decreased TC, LDL, and HDL. Predicting the choice of cholesterol-lowering medicine, comprehending the changing trend, and starting lifestyle

changes to obtain target lipid levels are all made easier with regular evaluation of the lipid profile.

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