

Serial measurement of lipid profile in patients with acute myocardial infarction

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2. PGR CPEIC, **Abstract... Objective:** To investigate the effect of acute myocardial infarction (AMI) on the levels of lipid profile and inflammatory markers. **Study Design:** Prospective study. **Place of study:** Choudhary Pervaiz Ellahi Institute of Cardiology in one-year duration from May 2019 to May 2020. **Methodology:** Patients admitted within 24 hours of onset of symptoms were enrolled in study and their total cholesterol, triglycerides, high density and low density lipoprotein was assessed. SPSS version 23 was used for data analysis. Tests of significance were applied and p-value less than or equal to 0.05 was taken as significant.
3. PGR CPEIC, **Results:** The mean total cholesterol, triglyceride, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, TC / HDL-C, LDL-C / HDL-C, high sensitive C-reactive protein, interleukin-6 and interleukin-10 of the control patients was 169.02±6.69 mg/dl, 152.27±3.59 mg/dl, 108.18±4.22 mg/dl, 50.93±4.81 mg/dl, 3.97±0.49, 2.48±0.64, 0.96±0.48 mg/dl, 14.31±4.68pg/dl, and 13.98±2.53pg/dl, respectively. **Conclusion:** Early treatment of lipid changes provides advantages and treatment lines for clinical decisions and lipid lowering therapy. Therefore, within 24 hours of onset of symptoms serum lipid values should be assessed.
4. PGR CPEIC, **Keywords:** Cardiovascular diseases, chronic obstructive pulmonary disease, Risk factor
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INTRODUCTION

Coronary heart disease is a serious medical condition that may lead to morbidity or mortality in young age. Among its risk factors dyslipidemia is major risk factor. Epidemiologically low level of high density cholesterol, lipoprotein cholesterol of low density and raised total cholesterol are major contributing factors in incidence of coronary heart disease and its mortality rate.

It is documented that treatment of hyperlipidemia at earlier time after acute myocardial infarction gives good advantages and also reduce the risk of morbidity and mortality. During acute illness level of lipid and protein changes abruptly, which may complicate the management. Acute phasic changes during tissue necrosis change the lipid profile after coronary artery events.

After AMI serum lipid modifications may include decrease in HDL, LDL and TL in approximately 20% in values that reciprocally raised the triglycerides up to 30%. Number of mechanisms is responsible for this change like up regulation of LDL receptors which is associated with acute phase response. Many previous studies concluded hyperlipidemia in acute myocardial infarction and recommended lipid profile assessment within 24 hours of AMI. Early duration of hyperlipidemia ensures the medication for lipid lowering.

This study aims to investigate the serum lipid changes in acute myocardial infarction in different time periods.

Study was started after permission from hospital ethical board and completed at Choudhary Pervaiz Elahi institute of Cardiology, Multan in one year duration from May 2019 to May 2020. Informed written consent was obtained from patients. Non probability consecutive sampling technique was used. Suspected patients of acute myocardial infarction presented at emergency department were enrolled in study. Patient diagnosed as AMI on baseline characteristics. Sampling for lipid profile was done within 24 hours of onset of symptoms was included in the study. Patients with chronic liver disease, rheumatic heart disease, cancer, renal failure, critical illness from last one month, sepsis, stroke history of last months, surgical procedure in last three months and regularly taking anti inflammatory drugs were excluded from the study. Physical examination, clinical history, presence of CVS risk factors, chest pain was monitored and recorded for every patients. Continuous electrocardiogram and cardiac enzymes were recorded. A rise in cardiac biomarkers was labeled as MI and at least one alternation in electrocardiogram (ST segment change and bundle branch block). Loss of myocardium on echocardiography,

diabetic patients were labelled as fasting blood sugar more than 126mg/dl and history of taking anti diabetic. Systolic blood pressure more than 140 and diastolic above 90mmhg was labelled as hypertension. Patients of BMI more than was labelled as obese, total cholesterol above 200mg/dl labelled as hyperlipidemia. Lipid profile and inflammatory marker measured within 24 hours of symptoms and then at 2nd and 7th day after MI.

SPSS version 23 was used for data analysis. Mean and standard deviation was calculated for numerical data like age, BMI and hemoglobin. Frequency and percentages were calculated for categorical data like gender, hypertension, diabetes, socioeconomic status, obesity, dyslipidemia and ischemic heart disease. P value less than or equal to 0.05 was considered as significant.

Results

Three hundred patients were included in this study, both genders. The mean age of control patients was 54.77±5.89 years. There were n=97 (64.7%) males and n=53 (35.3%) females. The mean BMI and hemoglobin of control patients was 25.02±1.98 kg/m² and 12.48±2.34 g/mL, respectively. Hypertension and diabetes were noted in n=62 (41.3%) and n=42 (28%), respectively. n=48 (32%) patients were smokers. While, n=34.7 (52%) patients used alcohol. n=34 (22.7%) patients had family history. While, the mean age of AMI patients was 53.28±5.05 years. There was n=103 (68.7%) males and n=47 (31.3%) females. The mean BMI and hemoglobin of AMI patients was 24.89±2.03 kg/m² and 12.79±2.57 g/mL, respectively. Hypertension and diabetes was noted in n=72 (48%) and n=39 (26%), respectively. n=48 (32%) patients were smokers. While, n=47 (31.3%) patients used alcohol. n=34 (22.7%) patients had family history. The age difference was statistically significant, (p=0.020). (Table. I).

The mean total cholesterol, triglyceride, low density lipoprotein cholesterol, high density lipoprotein cholesterol, TC / HDL-C, LDL-C / HDL-C, high sensitive C-reactive protein, interleukin-6 and interleukin-10 of the control patients was 169.02±6.69 mg/dl, 152.27±3.59 mg/dl, 108.18±4.22 mg/dl, 50.93±4.81 mg/dl, 3.97±0.49, 2.48±0.64, 0.96±0.48 mg/dl, 14.31±4.68pg/dl and 13.98±2.53pg/dl, respectively. While, the mean total cholesterol, triglyceride, low density lipoprotein cholesterol, high density lipoprotein cholesterol, TC / HDL-C, LDL-C / HDL-C, high sensitive C-reactive protein, interleukin-6 and interleukin-10 of the AMI patients was 177.74±5.11 mg/dl, 160.31±6.54 mg/dl, 115.13±7.25 mg/dl, 45.57±3.47 mg/dl, 5.18±2.13, 3.03±1.02, 10.48±3.28 mg/dl, 54.01±4.11pg/dl and 11.74±3.19pg/dl, respectively. By using the independent sample t-test, the differences of lipid profile and inflammatory in baseline of both groups were statistically significant at (p<0.001)

Table. II). Comparison of inflammatory makers and serum lipid profile of post-acute myocardial infraction at one day, two days and seven days were shown in table. III. We set the hypothesis that the mean of serum lipid profile and inflammatory makers are equal verses at least two means are different. We test this hypothesis by using one way ANOVA. It can be concluded that serum lipid profile and inflammatory makers are not equal at day 1 and day 2, day 1 and day 7, day 2 and day 7, at (p<0.05). (Table. III).

Table-I: Demographic and clinical characteristics of both the groups

Variable	Control n=150 (50%)	AMI patients n=150 (50%)	P-value
Age (years)	54.77±5.89	53.28±5.05	0.020
Gender			
Male	n=97 (64.7%)	n=103 (68.7%)	0.462
Female	n=53 (35.3%)	n=47 (31.3%)	
BMI (kg/m ²)	25.02±1.98	24.89±2.03	0.566
Haemoglobin g/mL	12.48±2.34	12.79±2.57	0.271
Hypertension	n=62 (41.3%)	n=72 (48%)	0.246
Diabetes	n=42 (28%)	n=39 (26%)	0.696
Smoking	n=48 (32%)	n=48 (32%)	1.000
Alcohol	n=34.7 (52%)	n=47 (31.3%)	0.539
Family history	n=34 (22.7%)	n=34 (22.7%)	1.000

Table-II: Comparison of lipid profile and inflammatory in baseline between two groups

Variable	Control n=150 (50%)	AMI patients n=150 (50%)	P-value
TC (mg/dL)	169.02±6.69	177.74±5.11	0.000
TG (mg/dL)	152.27±3.59	160.31±6.54	0.000
LDL-C (mg/dL)	108.18±4.22	115.13±7.25	0.000
HDL-C (mg/dL)	50.93±4.81	45.57±3.47	0.000
TC / HDL-C	3.97±0.49	5.18±2.13	0.000
LDL-C / HDL-C	2.48±0.64	3.03±1.02	0.000
hs-CRP (mg/L)	0.96±0.48	10.48±3.28	0.000
IL-6 (pg/mL)	14.31±4.68	54.01±4.11	0.000
IL-10 (pg/mL)	13.98±2.53	11.74±3.19	0.000

Table-III: Comparison of lipid profile and inflammatory in baseline between two groups

Variable	Day 1	Day 2	Day 7
TC (mg/dL)	176.24±7.63	161.53±3.52	167.62±7.93
TG (mg/dL)	160.41±8.02	174.67±6.05	170.03±3.61
LDL-C (mg/dL)	115.23±6.59	103.09±3.68	104.09±3.52
HDL-C (mg/dL)	45.49±4.36	38.29±3.43	38.82±10.36
TC / HDL-C	4.72±1.29	5.19±1.36	4.79±1.21
LDL-C / HDL-C	2.82±0.99	3.01±1.08	2.93±1.36
hs-CRP (mg/L)	10.37±2.52	17.22±5.72	13.91±3.66
IL-6 (pg/mL)	52.21±3.39	67.96±5.89	54.20±6.35
IL-10 (pg/mL)	12.16±2.49	18.75±6.69	15.02±2.88

Discussion

In our study we observed a decrease in total cholesterol, high density lipids and low density lipids but an increase in plasma triglycerides was seen at acute phase. Similar findings were observed by Wattansuwan et al¹¹ in 2001 during acute phase. This change in lipid metabolism occurs during acute phase but some time later opposite response was observed gradually without time specification. Correia et al¹² reported in his study that a significant decrease in cholesterol levels was found during acute phase of MI. Treatment interruption should be cautionary because this change in lipid and lipoprotein may be because of inflammatory response of MI.

In 2013 Khan et al¹³ conducted a similar study and reported that a significant decrease was occur after acute myocardial infarction but this change may be temporary. Serum LDL-C changes in statistical way from day first to day seven (1-7). Balci et al¹⁴ also conducted a similar study and reported that LDL level increased at day 1 of hospital admission and decreases in later time. This decrease was reaction of up regulation of LDL R activity during acute myocardial infarction phase.

Pitt et al¹⁵ concluded in his study that decrease LDL C concentration altered that oral intake and shift the patient on intravenous hydration that need hospitalization of a person. This happened mostly at 48 hours of acute attack. Many review studies we're conducted on this topic that reflects the similar conclusions, Ko et al¹⁶ reported in his review that a decrease serum LDL level was seen in samples taken at 24 hours or after 24 hours of acute attack. Serum LDL was 120 mg/dl at 24 hours and 116 mg/dl after 24-hour

samples.

In our study we observed HDL-C started decreasing from day 2 and continue decreasing with passage of time. In a study by Nigam et al¹⁷ reported that HDL C decreased after 48 hours of acute attack and this fall in values remain at lower level after that. Kumar et al¹⁸ conducted a study on this topic in 2009 and reported that fall in high density lipids and cholesterol may be cause of metabolic alterations after acute myocardial infarction and enzyme changes. Both these studies coincide with our findings strengthens our conclusion.

Rosjika et al¹⁹ also reported that ideal time for determination of HDL level is first 24 hours after acute myocardial attack that reflects the real cause of actual cardiac event. Several quantitative and qualitative effects occur on serum HDL level and this event labeled as acute phase response. Endothelial lipase activity increases on this time that result in decrease of HDL in serum and also inflammatory response.

Conclusion

Early treatment of lipid changes provides advantages and treatment line for clinical decisions and lipid lowering therapy. Therefore, within 24 hours of onset of symptoms serum lipid values should be assessed.

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