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LIPID PROFILE AND CARDIAC MARKERS IN PATIENTS OF MYOCARDIAL INFARCTION: A DEMOGRAPHIC STUDY WITH BIOCHEMICAL PERSPECTIVE IN INDIAN POPULATION

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ABSTRACT The vascular disorders are leading cause of death, with increase in prevalence of physical deformity, cardiovascular and cerebrovascular disorders worldwide. The myocardial infarction also called as heart attack that affects mainly cardiac muscle functions. There are several risk factors including biochemical, genetic and environmental that causes myocardial infarction, Therefore even after an advancement of diagnostic science, diagnosis of acute myocardial infarction is challenging because of its multiple etiology. In the present study the focus was the biochemical factors causing myocardial infarction and the cardiac markers. The concentrations of triglycerides, total cholesterol and HDL cholesterol were estimated from fasting serum samples, followed by the calculations of LDL & VLDL cholesterol and ratios viz. TC/HDL-C, TG/HDL-C, and LDL-C/HDL-C. Also we have estimated the CK-MB, Troponin-I and Brain Natriuretic Peptide (BNP) levels. 150 acute myocardial infarction subjects along with 150 age and sex matched healthy controls were included in the present sutdy. Significantly higher levels of CK-MB, TnI and BNP were reported in AMI patients than controls. There was a significant increase in levels of triglyceride, total Cholesterol, LDL Cholesterol, VLDL Cholesterol, LDL-C/HDL-C, TC/HDL-C, TG/HDL ratios and decrease in HDL cholesterol was observed. Therefore it is suggestive to investigate the combination of lipid parameters with CK-MB, TnI and BNP levels, which will help in diagnosis of AMI with early signs.

KEYWORDS : Acute myocardial infarction, Brain natriuretic peptide, Troponin I and CK-MB, lipid Profile. Cardiovascular disease (CVD)

INTRODUCTION:

Worldwide the mortality and morbidity rate due to AMI is increased drastically. [1] Among various causes of AMI atherosclerotic coronary artery disease (CAD), with damage to plaque causing transitory, partial or complete blockage is one of the most common cause. The normal of heart is dependent of several factors, one of is the adequate blood flow, failing which lead to death. Numerous risk factors playing a crucial role in the development of coronary heart disease (CHD) are known for e.g. hypertension, diabetes, smoking, obesity etc. These factors however only partly attribute to cardiovascular diseases. [2, 3]. Globally, Cardiovascular disease (CVD) alone is responsible for about 17 million deaths. Almost 2 fold increase in deaths due to ischemic heart disease in India have been noted within last two decades. [4]

Myocardial infarction (MI) is one of important manifestation of CHD. [5] CVDs are multifactorial in origin including the role of genetic and environmental factors. Along with these, the traditional risk factors like age, alcohol, diabetes etc. leading to elevated CVD risk, leading to cardiac muscle injury raising the levels of different cardiac markers [6]. Estimation of lipid levels including triglycerides (TG), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C) help in assessment of risk of CVD, as elevated TG, TC, LDL-C and low HDL-C are independent risk factors to accelerate atherosclerotic plaque formation. [7–10]

Thus, the objective of this study was to estimate the creatine kinase-MB (CK-MB), troponin I and BNP level along with the estimation of lipid profile parameters viz. Total cholesterol, HDL cholesterol and triglycerides.

MATERIALS AND METHODS:

The present case-control study was performed in the Department of Biochemistry, B. J. Medical College Pune. The total 150 Myocardial infarction patients were recruited after clinical diagnosis was done by physician from department of medicine. Patients who had typical symptoms of MI like chest pain, sweating, breathlessness, etc. and specific abnormalities for MI on electrocardiogram and elevated cardiac markers were recruited in the present study. Patients were diagnosed for MI on the basis of relevant diagnostic modalities such as clinical examination and electrocardiogram along with routine biochemical investigations. The other group included 150 age and gender matched healthy individuals as controls.

After taking informed consent the 6 ml of blood samples were collected from all participants after overnight fasting in plain and EDTA vacutainers and used for analysis of biochemical and cardiac markers. For myocardial infarction subjects, sample was collected within three days of diagnosis of the disease. The approval of institutional ethics committee was obtained, and informed consent from all participants (or relatives) was taken.

Estimation of lipid profile was done using fully automated biochemistry analyzer. Total cholesterol was estimated by CHOD-PAP method [11], Triglycerides by GPO–Trinder's method [12], HDL-C by phophotungstic acid method [13], levels of LDL-C & VLDL-C were calculated by using Friedewald's formula [14]. CK-MB was measured by immunoinhibition method [15], Troponin I by immunoenzymometric assay [16], BNP was measured by flurescence immunoassays [17].

Statistical Analysis:

SPSS version 17.0 was used to analyse the data statistically. Continuous variables were presented as mean ± standard deviation (SD) and differences in means between MI subjects to that of controls were tested by student 't' test.

RESULTS:

In the present study, concentration of serum lipid profile, Troponin I, CK-MB, and BNP were studied in acute myocardial infarction (AMI) patients with age and sex matched healthy controls. Lipid profile viz. triglycerides, total cholesterol and HDL-C were estimated and the values of VLDL-C, LDL-C were calculated by using Friedwald's equation along with the ratios of TC/HDL-C, TG/HDL-C and LDL-C/HDL-C. The levels of Troponin I , BNP and CK-MB along with lipid profile values are depicted in table no. 2 & 3.

There was no significant difference in the means of age between study and control group as $p>0.05$. The mean ages of AMI patients and control group are shown in table no. 1.

Table 1 - Comparison of age in study and control group

Parameter	Study group (n=150)	Controls (n=150)	P Value
	Mean ± SD	Mean ± SD	
Age (Yrs)	59.96 ± 9.06	58.82 ± 8.78	0.27

Serum levels of TG, TC, LDL-C, VLDL-C along with LDL-C/HDL-C, TC/HDL-C, TG/HDL ratios were significantly (<0.0001) higher in MI patients than healthy controls . HDL_C was significantly (<0.0001) decreased in MI patients compared to healthy controls. Table no. 2 represents the comparison lipid profile, between myocardial infarction patients and controls.

Table 2 - Comparison of lipid profile in study and control group

Parameter	Study(n=150) Mean ± SD	Control(n=150) Mean ± SD	P Value
Triglyceride (mg/dl)	125.42 ± 13.73	98.53 ± 9.61	<0.0001
Total Cholesterol (mg/dl)	216.48 ± 33.69	173.74 ± 21.62	<0.0001
HDL Cholesterol (mg/dl)	38.90 ± 2.42	45.17 ± 11.59	<0.0001
LDL Cholesterol (mg/dl)	151.04 ± 12.47	103.17 ± 10.76	<0.0001
VLDL Cholesterol (mg/dl)	25.08 ± 2.75	19.71 ± 1.92	<0.0001
LDL-C/HDL-C	3.90 ± 0.44	2.46 ± 0.75	<0.0001
TC/HDL-C ratio	5.58 ± 0.91	4.13 ± 1.26	<0.0001
TG/HDL ratio	3.24 ± 0.42	2.34 ± 0.67	<0.0001

The mean level of serum Troponin I was significantly (P<0.0001) higher in MI patients (2.91 ± 1.31 ng/ml) as compare to controls (0.45 ± 0.37 ng/ml). CK-MB concentration (63.2 ± 46 IU/L) in MI patients was significantly (<0.0001) higher than controls (21.61 ± 3.48 IU/L). Mean BNP level in MI was 200.61 ± 48.33 pg/ml and in controls it was 57.90 ± 14.75 pg/ml and difference was statistically significant (P<0.0001). Table no. 3 represents the comparison of CK-MB, Troponin I and BNP between myocardial infarction patients and healthy controls.

Table 3 - Comparison of CK-MB, Troponin I and BNP in study and control group

Parameter	Study group (n=150) Mean ± SD	Controls (n=150) Mean ± SD	P Value
Troponin I (ng/ml)	2.91 ± 1.31	0.45 ± 0.37	<0.0001
Ck-MB (IU/L)	63.2 ± 46	21.61 ± 3.48	<0.0001
BNP(pg/ml)	200.61 ± 48.33	57.90 ± 14.75	<0.0001

DISCUSSION:

In India the leading cause of mortality and morbidity is the cardiovascular disease, [18] constitutes a burden of epidemic proportion, and understanding its determinants is essential to designing effective interventions. [19] CVDs were thought to be the disease of the elderly, but escalated to the younger age too with equal proportion in both gender in rural as well as urban population. [18] AMI is caused by multiple factors leading to its pathogenesis; however, numerous studies depicted the role of altered metabolism of lipid in development of disease as one of the crucial risk factor. [20]

Therefore we aimed to compare the levels of lipid profile and cardiac markers in MI patients to that of healthy population. Significantly higher concentration of TG, TC, LDL-C, and VLDL-C were found in MI patients when compared to healthy controls. The ratios of TC/HDL-C TG/HDL-C and LDL-C/HDL-C were also found to be raised in MI patients than healthy controls, while HDL cholesterol was significantly declined in MI patients than controls. The elevated flow of fatty acids along with impairment in clearing of VLDL from plasma, thought to be the mechanism to increase the triglyceride level after MI. [21]

The finding of present study are consistent with the present knowledge and similar results were reported by Rathor V. [22] with significantly higher levels of TC, LDL-C, TG, and VLDL-C and lower level of HDL-C in MI patients as compared to control subjects. Similar results were also observed by Sharma J. [23] in AMI patients. A study from Chhattisgarh by Agnihotri M.A. [24], and a study from Adilabad by Venkateshwarlu M. and Chelmakuri G. [25] depicted similar results in AMI patients when compared to healthy controls.

A study by Vijayalakshmy G. et.al [26] and in a different study by Santhosh K.N. et.al [27] from Tumkur Karnataka, reported significantly raised levels of serum cholesterol, triglyceride, LDL cholesterol &LDL-C/HDL-C ratio and decreased level of HDL cholesterol in patients of MI in comparison to controls. Achari V. et.al [28] reported significantly increased levels of TC/HDL-C and mean LDL-C/HDL-C ratios along with other lipid parameters in CAD patients than subjects without CAD.

Comparison of cardiac biomarkers in the present study between MI patients and control revealed myocardial infarction patients had significantly higher levels CK-MB, Troponin I, and BNP levels than controls. These are expected findings correlating with the present knowledge. The data of level of activity of enzyme creatine kinase, more specifically the MB isoenzyme still conflicts in defining myocardial infarction. In this study, CK-MB levels in patients with AMI were higher than healthy controls. The study supporting to our finding was carried out by Yilmaz A. et.al [29] showing higher CK-MB values, in some cases reach nine times more than the upper normal value, and were correlated with the clinical diagnosis.

Along with CK-MB, the present study evaluated the increase in the levels of Troponin I and BNP in AMI patients than controls. Kasap S. [2] also found the higher level of CK-MB in AMI patients than controls. Carlos A. [30] evaluated the use of troponin I and T as diagnostic markers in myocardial cell damage associated with unstable angina & MI. These contractile cardiac specific proteins were shown to be superior than the cardiac enzymes like CK-MB, and are known to predict adverse events validly in acute coronary syndrome (ACS) patients. Many studies have reported that Troponin-I is more specific and sensitive than CK-MB specifically in case of minor myocardial damage [31-33]. Similar to our findings Gupta S. et.al and Murray et.al also noted that Troponin-I is better indicator as a cardiac marker [34, 35]. Troponin is also predicted to help as early marker in cases of unstable angina [36]. Mehran et.al has also reported that CK-MB elevation correlates with coronary artery plaque burden and calcification which are considered to be the principal causes for myocardial ischemic lesions [37]. The level of BNP was found to be significantly (<0.001) increased in one of study, and stated brain natriuretic peptide (BNP) is released from ventricular myocites due to their stretching and volume overload. [38]

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