Original Research Article

Lipid profile and its correlation with C-reactive protein in patients of acute myocardial infarction

Vedika Rathore[1]*, Neelima Singh[1], Puneet Rastogi[2], Roshan Kumar Mahat[1], Mritunjay Kumar Mishra[3], Reetika Shrivastava[1]

[1]Department of Biochemistry, Gajra Raja Medical College and J.A. Group of Hospitals, Gwalior Madhya Pradesh, India
[2]Department of Cardiology, Gajra Raja Medical College and J. A. Group of Hospitals, Gwalior Madhya Pradesh, India
[3]Department of Biochemistry, MGM's Medical College, Navi Mumbai, Maharashtra, India

*Correspondence:
Dr. Vedika Rathore,
E-mail: ved_sin26@rediffmail.com

ABSTRACT

Background: Acute myocardial infarction (AMI) is a significant cause of morbidity and mortality worldwide, which results from occlusion of coronary artery. Dyslipidemia is a major risk factor of AMI. C-reactive protein (CRP) is an acute phase protein, synthesized by hepatocytes in response to cytokines released into circulation by activated leukocytes and has been found to increase after AMI. The objective of the present study is to investigate lipid profile in AMI patients and correlate it with inflammatory marker i.e. CRP.
Methods: The present study includes 150 AMI patients and 100 normal healthy individuals as controls. In all the cases and controls, serum lipid profile and inflammatory marker were measured by diagnostic kits supplied by ERBA.
Results: The levels of lipid profile and inflammatory marker were significantly altered in the AMI cases compared to controls. We found significantly higher levels of total cholesterol, TG, LDL, VLDL, CRP and lower level of HDL in AMI compared to that of control subjects. We also found strong positive correlation of CRP with total cholesterol, triglyceride, LDL-C and VLDL-C and significant negative correlation with HDL-C in AMI patients.
Conclusions: We found alterations in the lipid profile and inflammatory marker in AMI cases; hence, all the people should undergo regular check up including lipid profile evaluation and inflammatory marker such as CRP to decrease the incidence, morbidity and mortality from the disease.

Keywords: Acute myocardial infarction, CRP, Dyslipidemia

INTRODUCTION

Acute myocardial infarction (AMI), commonly known as a heart attack, results from the interruption of blood supply to a part of the heart, causing heart cells to die. This is most commonly due to occlusion of a coronary artery following the rupture of a vulnerable atherosclerotic plaque which is an unstable collection of lipids (cholesterol and fatty acids) and white blood cells (especially macrophages) in the wall of an artery. The resulting ischemia (restriction in blood supply) and ensuing oxygen shortage, if left untreated for a sufficient period of time, can cause damage or death (infarction) of heart muscle tissue (i.e. myocardium).[1,2] Acute myocardial infarction (AMI) continues to be a major cause of morbidity and mortality worldwide.[3,4] It remains a leading cause of death in India and represents an enormous cost to health care system.[5] The mortality rate of MI is approximately 30% and for every 1 in 25 patients who survive the initial hospitalization, dies in the first year after AMI. Indians are four time more prone to AMI as compared to the people of other countries due to a combination of the genetic and lifestyle factors that promote metabolic dysfunction. The risk of

cardiovascular disease is predicted by various factors such as age, sex, smoking, hypertension and dyslipidemia.[6]

AMI is a multifactorial disease and impaired lipid metabolism is one of the crucial factors in the development of this disease.[7] During tissue necrosis, as seen in AMI, alteration in lipid profile is encountered.[8]

Kumar et al observed significantly higher total cholesterol (TC) and triglyceride (TG) levels and lower high-density lipoprotein cholesterol (HDL) levels in AMI patients.[9] Woo et al observed higher mean TC, LDL, and TG as well as lower mean HDL in AMI patients; high HDL was among the protective factors.[10] It is well known fact that early treatment of hyperlipidemia following acute myocardial infarction (AMI) provides potential benefits and reduces the morbidity and mortality of coronary heart disease.[11]

AMI is associated with profound systemic inflammatory response including elevated levels of circulating inflammatory mediators and activation of peripheral leukocytes and platelets. The excessive inflammatory response in AMI could be caused by a deregulated immune system.[12]

C-reactive protein (CRP) is a phylogenetically highly conserved plasma protein that participates in the systemic response to inflammation and its plasma concentration increases during inflammatory states, a characteristic that has long been employed for clinical purposes.[13] Tissue necrosis is a potent acute phase stimulus, and following AMI, there is a major CRP response, the magnitude of which reflects the extent of myocardial necrosis.[14] The present study was undertaken with aim to investigate lipid profile in AMI patients and correlate it with inflammatory marker i.e. CRP.

METHODS

The present study was carried out in the Department of Biochemistry and Department of Cardiology, Gajra Raja Medical College and J. A. Group of Hospitals, Gwalior. The study included 250 subjects (both sex) of age group 35-75 years.

Out of them 100 were normal healthy individuals and they formed the normal control group. 150 of them were patients of AMI admitted to the Cardiology Department of J.A. Group of Hospitals formed the study group.

Each patient undergone an initial clinical and laboratory evaluation, which included the detailed clinical history, clinical examination, standard 12 lead ECG, routine blood investigations and cardiac biomarkers [CK-MB and cardiac troponin T (Card test)] as a part of routine assessment and diagnosis of AMI was made after critical review of all these information by a cardiologist. Patients with diabetes mellitus, chronic muscle disease, renal disease, recent surgery, implanted pacemaker, autoimmune disease, arthritis, any inflammatory disease and any other disease except AMI were excluded from the study.

5ml of fasting venous blood sample was taken from AMI patients and controls under all aseptic precautions. Blood sample was collected in plain vial and incubated at 37°C for 30 minutes. After incubation, clot was removed and remaining sample was taken in centrifuge test tube. Samples were centrifuged at 3000rpm for 10 to 20 minutes. Supernatant was collected in clean and dry test tube for analysis of lipid profile and inflammatory marker.

Lipid parameters and inflammatory marker i.e CRP were estimated by standard biochemical kits supplied by ERBA using Mindray-BS 400, a fully autoanalyzer. This study was approved by institutional ethical committee and written consent was also obtained from the patients prior to study.

Statistical analysis

Data are presented as mean±SD values. The statistical differences between cases and control were determined by student independent sample t-test. Data analyses were performed with the Statistical Package for the Social Sciences, version 21.0 (SPSS, Chicago, Illinois, USA).

RESULTS

A total of 250 subjects were included in the present study. Of these, 150 were cases of AMI and rest 100 were controls. Table 1 and figure 1 shows the mean levels of TC, TG, HDL-C, LDL-C, VLDL-C and CRP in AMI cases and controls. The mean levels of TC, TG, LDL-C, VLDL-C and CRP were increased in AMI cases as compared to controls and were statistically highly significant (p<0.001) whereas mean level of HDL-C was highly significantly decreased in AMI cases as compared to controls (p<0.001).

Table 1: Mean levels of lipid parameters and CRP in AMI cases and controls.

| Parameters	Controls (n=100) (mean±SD)	AMI cases(n=150) (mean±SD)
TC (mg/dl)	150.64±8.98	223.96±35.08**
TG (mg/dl)	105.62±21.79	202.97±36.15**
HDL-C (mg/dl)	42.02±6.66	34.70±2.17**
LDL-C (mg/dl)	87.50±12.11	148.66±29.31**
VLDL-C (mg/dl)	21.12±4.36	40.59±7.23**
CRP(mg/L)	4.31±1.00	8.14±1.84**

**Highly Significant (p<0.001), TC=Total Cholesterol, TG=Triglyceride, HDL-C=High density lipoprotein cholesterol, LDL-C=Low density lipoprotein cholesterol, VLDL-C=very low density lipoprotein cholesterol, CRP= C-reactive protein.

Table 2: Correlation of CRP with lipid profile in patients of AMI with CRP.

Markers	Pearson correlation, r value	p-value
TC	0.240	0.003**
TG	0.282	0.000**
HDL-C	-0.190	0.020*
LDL-C	0.231	0.004**
VLDL-C	0.282	0.000**

*Significant at the 0.05 level, **Highly Significant at the 0.01 level, TC=Total Cholesterol, TG=Triglyceride, HDL-C=High density lipoprotein cholesterol, LDL-C=Low density lipoprotein cholesterol, VLDL-C=very low density lipoprotein cholesterol, CRP= C-reactive protein.

Table 2 shows correlations of CRP with lipid profile. CRP was positively correlated with total cholesterol, triglyceride, LDL and VLDL in AMI patients and was statistically highly significant whereas there was significant negative correlation between CRP and HDL cholesterol.
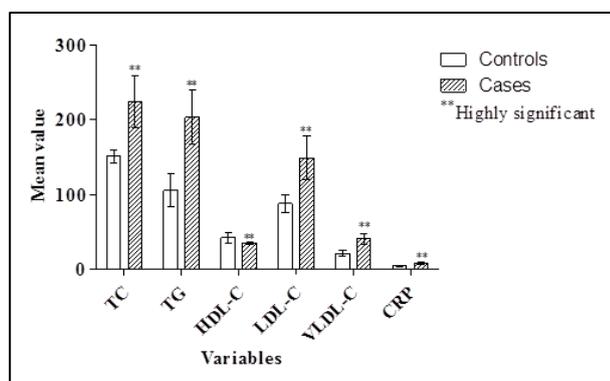
Figure 1: The status of lipid profile and CRP in AMI cases and controls.

DISCUSSION

In this study, lipid parameters in AMI subjects were studied and correlated with inflammatory marker. In our study, we found significant increased levels of TC, TG, LDL, VLDL and significant decreased level of HDL in AMI cases as compared to controls (p<0.001). Kumar et al in their study observed significantly higher total cholesterol (TC) and triglyceride (TG) levels and lower high-density lipoprotein cholesterol (HDL) levels in AMI patients, which are in agreement with our study.[9] In agreement with the present study, Woo et al also observed higher mean TC, LDL and TG as well as lower mean HDL in AMI patients.[10]

Similar results have been also observed by Shirafkan et al who also found higher levels of TC, LDL, VLDL and TG as well as lower levels of HDL in AMI patients.[15] More et al also reported significant increased levels of TC, TG, LDL-C and decreased levels of HDL-C in AMI patients as compared to controls, which are also similar to the findings of our study.[16] In contrast with our study, Khan et al showed significant decreased levels of total cholesterol and LDL-cholesterol in AMI patients.[7] Moreover, they added the reduced serum lipids in AMI patients were not clear and it could be related to dietary modifications or due to metabolic changes during acute crisis.[7] Lehto et al did not found any difference in mean serum levels of TC between the AMI patients and controls whereas mean HDL was significantly lower in the AMI subjects.[17] Elevated serum cholesterol has depended on elevated consumptions of fat and genetic basis.[15,18-19]

LDL carries the most of cholesterol in the plasma and increasing of LDL depends on increasing of total cholesterol level.[15-20] Nigam et al reported increasing trend of triglyceride after myocardial infarction with a significant increase on day three and predischarge as compared to day one.[21] However, Vetter et al showed that triglyceride levels diminished slowly from the second hour after myocardial infarction.[22] Ryder et al reported that there was no significant difference in triglyceride levels between cases and controls.[23]

There is a different mechanism about elevation of triglycerides after AMI. It has been reported that elevated triglyceride levels may depend on genetic basis[18] and nutritional habits.[24] The levels of triglyceride may also change because of inherited abnormality of very low density lipoprotein. It may happen because of elevated flux of fatty acids and impaired elimination of VLDL from the plasma.[21-25]

Epidemiologically high serum levels of HDL are associated with reduced risk for the development of atherosclerotic disease. HDL particles are believed to be antiatherogenic, secondary to their capacity to drive reverse cholesterol transport and antagonize pathways of inflammation, thrombosis, and oxidation.[26] Al Aqeel et al., observed that HDL appears to be the main lipid risk factor in patients of AMI, suggesting that primary prevention strategies should focus on treatment modalities that increase HDL.[27] There is an increased focus on targeting and treating low serum levels of HDL in an effort to further reduce risk for cardiovascular disease, including myocardial infarction.[26]

AMI is a multifactorial disease, in which inflammatory processes play a central role.[28] In this regard, CRP is considered to be the most important marker and it has been extensively studied in recent years.[29,30] In our study, we found significant increased mean level of CRP in AMI cases as compared to controls. This is in agreement with the study done by Sesani et al who also reported increased mean level of CRP in AMI patients as compared to controls.[30] Kausadikar et al showed elevated C-reactive protein in patients who present with an anterior wall myocardial infarction reflecting the greater amount of myocardial damage being associated with raised C-reactive protein levels.[31] An increased level of

CRP in our study seems to be due to the persistent and subclinical low grade chronic inflammation in acute myocardial infarction.

In the present study, we found strong significant positive correlation of serum CRP levels with total cholesterol, triglyceride, LDL and VLDL in AMI patients. In the other hand, we found significant negative correlation of serum CRP levels with HDL-cholesterol. Khan et al in their study found significant inverse correlation between hs-CRP and HDL (but not with other lipid parameters) in AMI patients.[7] The findings of our study support the fact that there occurs co-existence of inflammation and impaired lipid metabolism.

CONCLUSION

In present study, we found alterations in the lipid profile and inflammatory marker in AMI cases, which play significant role in incidence of AMI. Hence, all the people should undergo regular checkup including lipid profile evaluation and inflammatory marker such as CRP to decrease the incidence, morbidity and mortality from the disease.

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