

## Evaluation of serum C-reactive protein and lipid profile in patients with myocardial infarction

Received: 8/3/2017

Accepted: 3/7/2017

Leweza B. Abbass \*

### Abstract

**Background and objective:** Myocardial infarction is the irreversible death (necrosis) of heart muscle secondary to prolonged lack of oxygen supply (ischemia), when the blood supply to a part of the heart is interrupted, most commonly due to rupture of a vulnerable plaque. The resulting ischemia or oxygen shortage causes damage and potential death of heart tissue. It is a medical emergency, and the leading cause of death worldwide. In this study, we investigated the association of serum C-reactive protein and serum lipid profile with myocardial infarction.

**Methods:** A cross-sectional study was carried out from January to May 2016 in College of Medicine, Hawler Medical University. The study involved 108 patients with myocardial infarction (70 men and 38 women) in comparison with 50 healthy individuals as the control group (29 men and 21 women).

**Results:** The levels of serum C-reactive protein were significantly higher ( $P < 0.05$ ) in patients with myocardial infarction in both genders when compared with the control group. There was no statistically significant difference in serum total cholesterol, serum triglyceride and serum LDL-C in patients with myocardial infarction and control group. Regarding serum high-density lipoprotein (HDL-C), there was a statistically significant difference ( $P < 0.01$ ) in patients with myocardial infarction when compared with the control group.

**Conclusion:** C-reactive protein may play an important role in providing clinicians and biochemists with valuable information regarding diagnosis and following up the patient with suspected myocardial infarction.

**Keywords:** Myocardial infarction; C-reactive protein; Lipid profile.

### Introduction

Myocardial infarction is a life-threatening condition that occurs when blood flow to the heart is abruptly cut off causing tissue damage. This is usually the result of a blockage in one or more of the coronary arteries.<sup>1</sup> An association between sustained high values of C-reactive protein (CRP) following myocardial infarction and adverse outcomes was first reported in 1982. Subsequent large studies have shown that increased peak and post-infarct CRP concentrations are significantly associated with increased incidence of cardiac complications including heart failure and cardiac death, apparently independently of other predictors. Tissue

necrosis is a potent acute phase stimulus, and following myocardial infarction, there is a major CRP response.<sup>2,3</sup> Despite great progress in the field of pharmacological therapy chronic conditions like coronary artery diseases, diabetes mellitus, obesity, and metabolic syndrome remain the major cause of mortality and morbidity in the modern world.<sup>4</sup> Inflammation plays a key role in the initiation and progression of these conditions. CRP is a highly conserved plasma protein that participates in the systemic response to inflammation. CRP is a marker of inflammation and a potential independent predictor of cardiovascular disease as it may play a role in the development of atherosclerosis;

\* Department of Clinical Biochemistry, College of Medicine, Hawler Medical University, Erbil, Iraq.

additionally, it also adversely affects mortality.<sup>5</sup> Many studies showed that cardiovascular diseases, metabolic syndrome and Type 2 diabetes mellitus are associated with high CRP levels as there is low-grade systemic inflammation in these conditions.<sup>3,6</sup> Also, several studies have conducted and accepted that increased levels of low-density lipoprotein (LDL-C), triglycerides (TG), total cholesterol (TC) and decreased levels of high-density lipoprotein (HDL-C) are associated with atherosclerosis.<sup>3</sup> Oxidized LDL-C is one hypothetical risk factor for atherosclerotic inflammation. It is believed that oxidation of LDL-C may be modulated by nitric oxide (NO) and its products. LDL-C component of lipid profile is known to activate a cascade of local inflammation which can lead to the formation of atherosclerotic plaques.<sup>7</sup> This study aimed to evaluate the role of serum CRP levels and serum lipid profiles on myocardial infarction.

## Methods

### Collection of samples

Blood samples (5ml) of our present cross-sectional study of were withdrawn from patients diagnosed with myocardial infarction who attended Hawler and Rizgari Teaching Hospitals in Erbil City during the first six hours of admission and also from healthy subjects as the control group. Blood samples were collected into vacutainer tubes and were allowed to clot, and the serum was recovered by centrifugation at 3000 rpm for 15 minutes for removal of any suspended cells. The samples were either used immediately for estimation of serum C-RP, TC, TG, HDL-C, and LDL-C or kept at about -20 °C until further analysis. Serum TC, TG, LDL-C, HDL-C were estimated by using Cobas diagnostic kit (Roche/COBAS 311 IN-TEGRA), with a fully automated chemical analyzer. Serum C-RP protein CRP was analyzed using automatic chemical analyzer (Cobas biochemical analyzer).

### Study Population

The present study was carried out at the

College of Medicine, Hawler Medical University, Erbil, Kurdistan from January to May 2016. A total of 158 participants were enrolled in this study, 108 patients with myocardial infarction with no previous antimicrobial drug intake and 50 normal healthy individuals as a Control group.

### Statistical analysis

The data were analyzed using the statistical package for the social sciences. Data were expressed as mean  $\pm$  SE. The significance of differences was evaluated by independent t-test. The levels of serum CRP, total cholesterol, triglyceride, HDL-C and LDL-C of the control group were compared with patients with myocardial infarction concerning the *P* value. Statistical significance was set at *P* < 0.05.

### Ethical consideration

Ethics approval was granted by the Research ethics committee of the College of Medicine, Hawler Medical University. The cohort consisted of 120 individuals. The patient's blood was used as samples.

## Results

A total of 108 patients with myocardial infarction with age variant ranging between 36 and 80 years and a mean  $\pm$  SE age of 58.62  $\pm$  1.06. For the male, the range of age was 36-80 years, and the mean  $\pm$  SE was 59.26  $\pm$  1.30 years. For the female, the range of age was 38-76 years, and the mean  $\pm$  SE was 57.45  $\pm$  1.85 years. The 50 control or healthy adults were with age range variant between 34 and 76 years a mean  $\pm$  SE age of 56.78  $\pm$  1.44. The general characteristics of the study population are shown in Table 1. There were no statistically significant differences in both ages of gender factors between patients with myocardial infarction and control group (*P* > 0.05).

### Biochemical analysis

#### Serum C-reactive protein

The mean  $\pm$  S.E values of serum CRP were significantly higher (*P* < 0.05) in male patients with myocardial infarction with a mean value of 36.93  $\pm$  5 compared with control group (4.05  $\pm$  0.7). The mean serum

CRP was significantly higher in female patients with myocardial infarction (32.46±4.2) when compared with control group (4.19±0.7) as shown in Table 2.

**Serum Lipid profile**

The serum lipid profile of control and patients are shown in Table 3 and 4.

**Serum Total Cholesterol (TC)**

There was no statistically significant difference ( $P > 0.05$ ) between the mean serum TC in myocardial infarction group in female (194.02±7.8) when compared with the control group with (180.92±7.0) as shown in Table 3. The mean serum TC was significantly higher ( $P < 0.05$ ) in

myocardial infarction in male (200.8±4.1) when compared with control group (182.5±7.01) as shown in Table 4

**Serum Triglyceride (TG)**

The difference between the mean serum TG in the myocardial infarction group in female (166.5±11.2) and the control group (133.3±13.5) was not significant ( $P > 0.05$ ) as shown in Table 3. The mean TG was significantly higher ( $P < 0.05$ ) in myocardial infarction male group (184.3±6.3) compared with the control group (129.6±10.4) as shown in Table 4.

**Table 1:** The baseline characteristics of patients with myocardial infarction and control group.

Gender	Patients with myocardial infarction			Control group			P value
	Age Mean± SE	Range	No	Age Mean± SE	Range	No	
Males	59.26±1.3	36-80	70	57.41±1.9	35-77	29	0.069
Females	57.45±1.8	38-76	38	55.90±2.2	50-80	21	0.072
Total	58.62±1.0	36-80	108	56.78±1.4	34-76	50	0.078

**Table 2:** Mean ± SE of CRP in control group and myocardial infarction group.

Parameter	Control group Mean± SE	Myocardial infarction group Mean± SE	P value
CRP (mg/L) Male	4.05±0.7	36.93±5	0.001
CRP (mg/L) Female	4.19±0.7	32.46±4.2	0.001

**Serum High-density lipoprotein (HDL-C)**

There was a highly statistically significant difference ( $P < 0.05$ ) in mean HDL-C for patients with myocardial infarction when compared with the control group in both genders as shown in Table 3 and Table 4.

**Serum Low-density lipoprotein (LDL-C)**

The mean±S.E LDL-C was significantly

higher ( $P < 0.05$ ) in patients with myocardial infarction in female compared with the control group as shown in Table 4. There was no statistically significant difference in male patients with myocardial infarction compared with the control group as shown in Table 3 and Table 4.

**Table 3:** Lipid profile test levels in the female with myocardial infarction compared with the control group.

Parameters mg/dl	Control group Mean± SE	Myocardial infarction group Mean± SE	P value
TC	180.9±7.0	194.0±7.8	0.272
TG	133.3±13.5	166.5±11.2	0.073
HDL-C	49.4±2.4	37.1±1.8	0.0015
LDL-C	92.2±4.3	108.6±5.5	0.022

**Table 4:** Lipid profile test levels in the male with myocardial infarction compared with the control group.

Parameters (mg/dl)	Control group Mean± SE	Myocardial infarction group Mean± SE	P value
TC	182.5±7.0	200.8±4.1	0.021
TG	129.6±10.4	184.3±6.3	0.0014
HDL-C	51.2±2.4	36.8±1.0	0.0018
LDL-C	104.1±4.7	13.07±16.0	0.266

## Discussion

Results of the present study indicated that CRP levels were significantly elevated in patients with myocardial infarction when compared to the control group. This is in agreement with other studies.<sup>7</sup> In this regard CRP is considered to be the most important marker and have been extensively studied in recent years CRP might not only mirror an inflammatory stimulus, but also have direct effects promoting atherosclerotic propagation and destabilizing plaque. Our finding suggests that these substantially increased serum CRP levels in the clinical setting of myocardial infarction are due to the results of myocardial damage. Lipid abnormality regarded as one of the important risk factors for ischemic heart disease.<sup>8</sup> Mean serum TC and TG levels in patients with myocardial infarction in female were found to be higher than those of control, but not statistically significant. In contrast to female Mean serum TC, TG levels in the male with myocardial infarction were found to be higher than the control, and statistically significant. The values of serum HDL-C were significantly lower in patients with myocardial infarction compared with control in both genders. High serum level of high-density lipoprotein HDL-C, on the other hand, is associated with reduced risk for the development of atherosclerotic disease. The HDL-C particle is believed to be anti-atherogenic and antagonized pathways of inflammation, thrombosis, and oxidation.<sup>9-13</sup> Unlike to male with myocardial infarction the mean serum levels of LDL-C were significantly higher ( $P < 0.05$ ) in the female with myocardial infarction compared to the control. Studies have shown that the levels of LDL-C were associated with atherosclerosis. Oxidized LDL-C is known to enhance the expression of pro-inflammatory genes, leading to monocyte recruitment into the vessel wall and dysfunction of vascular endothelial cells.<sup>14-16</sup> It is cytotoxic to endothelial cells via generation of free radicals and impairs nitric oxide synthase gene expression and

its activity.<sup>17,18</sup>

## Conclusion

We concluded from the current study that high levels of CRP were detected in the serum of patients with myocardial infarction compared with healthy control group. Although many factors are involved in the development of myocardial infarction, measurements of CRP may be valuable as an indicator of inflammation during management and following up the patient with myocardial infarction. Serum CRP levels showed an inverse correlation with serum HDL-C levels in both genders. Serum CRP levels correlated with TC, TG, and LDL-C levels.

## Competing interests

The author declares no competing interests.

## References

1. Isoaito Y, Sato S, Kitamura A, Okamura T, Komach Y. Serum triglycerides and risk of coronary heart disease among Japanese men and women. *AJP* 2011; 153(5):490–9.
2. Paoletti R, Gotto A, Hajjar D. Inflammation in atherosclerosis and implications for therapy. *Circ* 2004; 109(23):111–20.
3. Choudhry A, Prabhu K, Kase. Association of high sensitive C-reactive protein, lipid profile parameters in South Indian population. *IJSRP* 2010; 37(2):35–43.
4. Iciar M, Cristina S, Amparo S, Francisco J. Type 2 diabetes and cardiovascular disease: Have all risk factors the same strength? *World J Diabetes* 2014; 5(4):444–70.
5. Behzad H. C-reactive protein and other markers of inflammation in hemodialysis patients *Caspian J Intern Med* 2013; 4(1):611–6.
6. Rodolfo P, Chiara B, Andrea P, Andrea C. Metabolic syndrome, inflammation and atherosclerosis. *Vasc Health Risk Manag* 2006; 2(2):145–52.
7. Ilya C, Sudhakar V, Utpal S, Suresh CT. Atherogenesis: hyperhomocysteinemia interactions with LDL, macrophage function, paraoxonase 1, and exercise *Ann N Y Acad Sci* 2016; 1363(1):138–54.
8. Khan HA, Alhomida AS, Sobki SH. Lipid profile of patients with acute myocardial infarction and its correlation with systemic inflammation. *Biomark Insights* 2013; 8:1–7.
9. Mehta L, Petters M. Oxidized LDL-C: A critical factor in atherogenesis. *Cardiovasc Res* 2005; 68(3):353–4.

10. Calabro P, Goliaand TT. Role of C-reactive protein in acute dial infarction and stroke: possible therapeutic approaches. *Curr Pharm Biotechnol* 2012; 13(1):4–16.
11. Chaudhur J, Mridula K, Umamahesh M, Swathi A, Balaraju B. High sensitivity C-reactive protein levels in acute ischemic stroke and subtypes: A study from a tertiary care center. *Iran J Child Neurol* 2013; 12(3):92–100.
12. Shalia K, Savant S, Haldankar V, Nandu T, Pawar P. Study of C-reactive protein and myocardial infarction in the Indian population. *Indian J ClinBiochem* 2012;27(1):74–82.
13. Shrivastava A, Singh H, Raizada A, Singh S. Serial measurement of lipid profile and inflammatory markers in patients with acute myocardial infarction. *EXCLI J* 2015; 14:510–7.
14. Janabi M, Yamashita S, Hirano K, Sakai N, Hiraoka H, Matsumoto K. Oxidized LDL–induced NF-κB activation and subsequent expression of proinflammatory genes are defective in monocyte-derived macrophages from CD36-deficient patients. *Arteriosclerosis, Arterioscler Thromb Vasc Biol* 2000; 20(8):1953–60.
15. Li D, Mehta JL. Antisense to endothelial ox-LDL receptor LOX-1 inhibits ox-LDL-mediated upregulation of MCP-1 expression and monocyte adhesion to human coronary artery endothelial cells. *Circulation* 2000; 101:2889–96.
16. Cominacini L, Pasini AF, Garbin U, Davoli A, Tosetti ML, Campagnola M, et al. Oxidized low density lipoprotein (ox-LDL) binding to ox-LDL receptor-1 in endothelial cells induces the activation of NF-kappaB through an increased production of intracellular reactive oxygen species. *J BiolChem* 2000; 28(275):12633–8.
17. Elena G, Klaus L. Immune and Inflammatory Mechanisms of Atherosclerosis. *Annu Rev Immunol* 2009; 27:165–97.
18. Li D, Mehta JL. Upregulation of endothelial receptor for oxidized LDL (LOX-1) by oxidized LDL and implications in apoptosis of human coronary artery endothelial cells: evidence from use of antisense LOX-1 mRNA and chemical inhibitors. *Arterioscler Thromb Vasc Biol* 2000; 20:1116–22.