International Journal of Research in Medical Sciences
Rathore A et al. Int J Res Med Sci. 2016 Apr;4(4):1051-1055
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20160782

Research Article

A study of high-sensitivity C- reactive protein and trop-T in patients with coronary artery disease

Anita Rathore*, Umesh Pareek, Avdhesh Sharma

Department of Biochemistry, Geetanjali Medical College and Hospital, Udaipur Rajasthan, India

Received: 05 January 2016
Accepted: 03 February 2016

***Correspondence:**
Dr. Anita Rathore,
E-mail: ani1234.rathore@gmail.com

ABSTRACT

Background: The diagnosis of Hs-CRP levels is helpful to correlate with the clinical severity of coronary artery disease and with coronary events in both acute and sub-acute phase of myocardial ischemia. Coronary heart disease (CHD) is the major cause of death in the developed world. The level of certain proteins in plasma increase during acute inflammatory state or secondary to certain types of tissue damage.
Methods: 100 subjects and 50 controls were taken from the outdoor and indoor department of medicine in Geetanjali Medical College & Hospital, Udaipur, Rajasthan, India. C-reactive protein was estimated by immuno-turbidometry method.
Results: We have also come across very high levels of Hs-CRP in the CAD patients and found that the value of Hs-CRP was 2.51±0.79 mg/L as compared to 0.76±0.34 mg/L in control and therefore the rise in Hs-CRP is highly significant because of p value (0.0001).
Conclusions: Hs-CRP is highly significant in CAD patients when compared to the control group. It is an independent and better predictor of CAD.

Keywords: CAD, HS-CRP, Troponin -T, Inflammation

INTRODUCTION

Coronary heart disease (CHD) is the major cause of death in the developed world.[1] The clinical manifestation usually occurs in the form of myocardial infarction (MI), stroke, angina, or sudden death between ages 50 and 60 years in men and between 60 and 70 years. Inflammation plays a vital role in accentuating the formation of atherosclerotic plaque, and thus the measurement of inflammatory markers provides a method of assessing cardiovascular risk.[2] Clinical and laboratory studies have shown that inflammation plays a major role in the initiation, progression, and destabilization of atheromas. C- reactive protein (CRP), an acute phase reactant that reflects low-grade systemic inflammation, in patients with established coronary disease, CRP has been shown to predict adverse clinical events[3]. The level of certain proteins in plasma increase during acute inflammatory state or secondary to certain types of tissue damage.[2,4] These proteins are called "acute phase proteins" and include CRP, CRP is thus named because it reacts with the C polysaccharide of *pneumo-cocci* (involved in promotion of immune system through activation of complement pathway and macrophages), it produced in liver and present in the circulation in minute concentration.[5] CV risk assessment requires a more sensitive assay, hsCRP, which can accurately detect very low levels of CRP in healthy individuals.[6] As a result of its high tissue specificity, cardiac TnT is cardio- specific, highly sensitive marker for myocardial damage.[2] Serum cardiac troponin T levels as an indicator of myocardial injury in ischemic and hemorrhagic stroke patients.[7] Troponin T (TnT) is a component of the contractile apparatus of the striated musculature. Although the

function of TnT is the same in all striated muscle, TnT originating exclusively from the myocardium.[7-9] Troponin T (TnT) is a component of the contractile apparatus of the striated musculature. Although the function of TnT is the same in all striated muscle, TnT originating exclusively from the myocardium (mol Wt. 39.7 kD) clearly differs from skeletal muscles TnT. As a result of its high tissue specificity, cardiac TnT is cardio-specific, highly sensitive marker for myocardial damage.[10-12] Due to lack of aforementioned literature we were decided to evaluate the status of hs-C-reactive protein and trop-t in patients with coronary artery disease.

METHODS

Study subjects 100 (mean age 31 to 70 years) were taken from the outdoor and indoor department of medicine and healthy control subjects (n=50) were selected from the institution (Geetanjali Medical College & Hospital, Udaipur, Rajasthan, India).

Exclusion criteria

Confounding factors which could interfere in the biochemical analysis of study subjects and alter the results are smoking, diabetes, active inflammatory diseases, nutritional deficiencies, estrogen therapy, and collagen disease arthritis. Patients with these diseases were excluded from the study. The same exclusion criteria utilized for cases were applied for control selection. All the above exclusion factors were confirmed from the patient's personal physician report and history.

Inclusion criteria

As mentioned above, all consecutive patients with ACS including unstable angina (UA), non-ST elevation myocardial infarction (NSTEMI) and ST elevation myocardial infarction (STEMI) were enrolled in to the study. UA was diagnosed if the patients had at least one of the following angina chest discomforts at rest lasting for ≥20 min, recent onset angina of sufficient severity or exertional angina with crescendo pattern, along with either ST segment depression ≥0.05 mm or T inversion ≥0.03 mV in any two contiguous leads.

Collection and analysis of sample

Blood samples were drawn from patients and controls. 5 ml blood was collected in plain vials and EDTA vials for estimation of serum high- sensitivity C-reactive protein and trop-T. As a measure of high sensitivity C-reactive protein was estimated by immunoturbidometry method (Otsuji et al). (The detection range for Hs-CRP is ≤5 mg/L). CRP causes agglutination of the latex particles coated with anti-human CRP. The agglutination of the latex particles is proportional to the CRP concentration and can be measured by turbidimetry. Trop-T estimation is based on the dual monoclonal antibody sandwich principle using a poly (streptavidin)-biotin capture system with a gold sol particles label.

RESULTS

The present study was conducted on total 150 subjects where 50 were controls and 100 were the patients. The present study was primarily planned to estimate Hs-CRP and Trop-T. In the present study there were 54 patients from the urban area and 46 patients from the rural area. The incidence of CAD in urban population is because of their life style and various risk factors such as smoking, chewing habits and alcoholic habits. General characteristics of the population -in subjects 30% of total cases were non vegetarian, while in control group it was 15%. In subjects 20% population have tobacco chewing habit, while in control group it was 2%. 4% of control population had presented with alcohol drinking status while in MI patients it was 24%. The mean BMI (body mass index) of control group was 22.812±3.71 kg/m^2; while that of study group was 26.07±3.38 kg/m^2. In control group 10% population were belong from rural area and 90% from urban area whereas in cases 43% belong from rural area while 57% from urban area. 5% of control population had presented with smoking status while in MI patients it was 30%.

In our study the mean BMI (body mass index) of control group was 22.812±3.71 kg/m^2. While that of study group was 26.07±3.38 kg/m^2. 4% of control population had presented with alcohol drinking status while in CAD patients it was 24%. 5% of control population had presented with smoking habit while in CAD patients it was 30%. In control group 21 were belong from rural area and 29 from urban area whereas in cases 46 belong from rural area while 54 from urban area. Whereas even in rural area the incidence of CAD is also more and perhaps this is because of transition of rural population to urban site and changes in their life style.

We have also come across very high levels of Hs-CRP in the CAD patients and found that the value of Hs-CRP was 2.51±0.79 mg/L as compared to 0.76±0.34 mg/L in control and therefore the rise in Hs-CRP is highly significant because of p value (0.0001). A significant difference has been observed regarding the value of Hs-CRP in CHD subjects as compared to normal.

Table 1: Smoking.

Smoking habit (100)	Subjects(n=100)	Controls(n=50)
Smokers	35%	7%
Non smokers	65%	93%

Table 1 showing the total number of subjects which includes 35 smokers and 65 non smokers in case group while 7 smokers and 93 non smokers in control group.

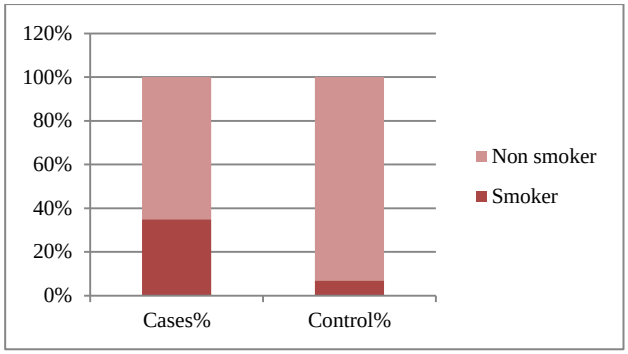

Figure 1: Smoking habits among subjects and control group.

As shown in Table 2 the total number of subjects which includes 24% alcoholic and 76% non alcoholic in case group while 4% alcoholic and 96% non alcoholic in control group.

Table 2: Alcoholic status.

Alcoholic status	Subjects (n=100)	Control (n=50)
Alcoholic	24%	4%
Non alcoholic	76%	96%

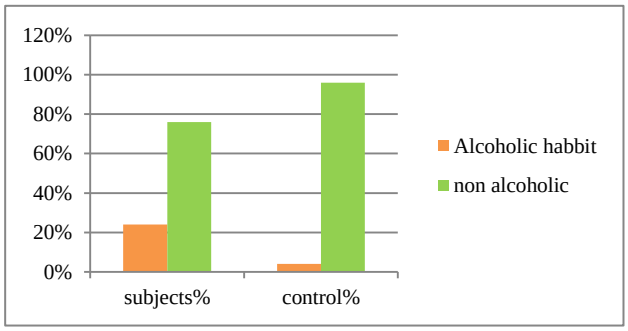

Figure 2: Alcoholic status among study group and control group.

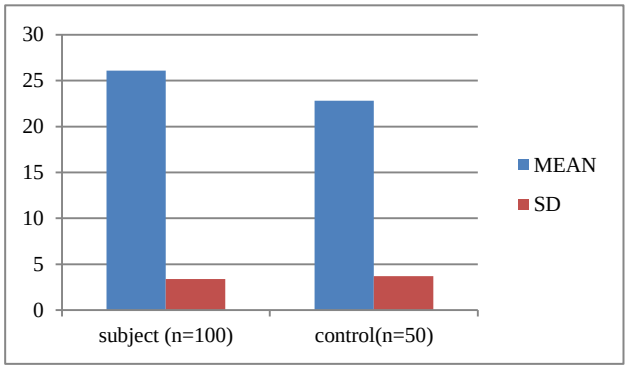

Figure 3: BMI status among subjects and control group.

Table 3 shows BMI status among subject and control group with mean 26.07, SD 3.38 in subject group whereas in control mean is 22.81 and SD 3.71.

Table 3: BMI.

BMI	Subject (N=100)	Control (n=50)
Mean	26.07	22.81
SD	3.38	3.71

Table 4 shows chewing habits among subjects with 20% chewing habits and 80% are none and in control group 2% has chewing habits and 98% are none.

Table 4: Chewing habits

Chewing habit	Subjects(n=100)	Control (n=50)
Chewer	20%	2%
None	80%	98%

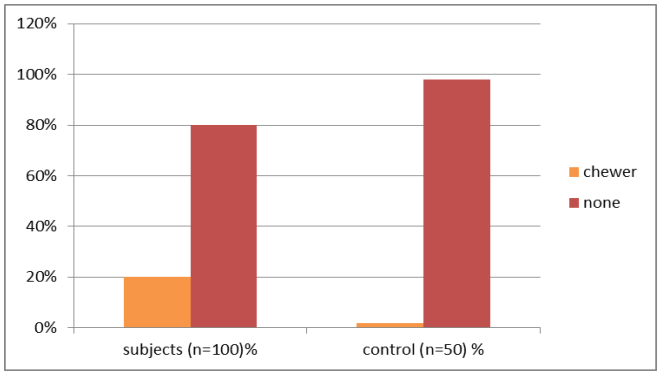

Figure 4: Chewing habit among subjects and control group.

Table 5: Risk factors.

Risk factors	Percentage (%)
Smoking	46%
Obesity	4%
Hypertension	10%
DM	8%
DM- hypertension	12%
No risk factors	20%

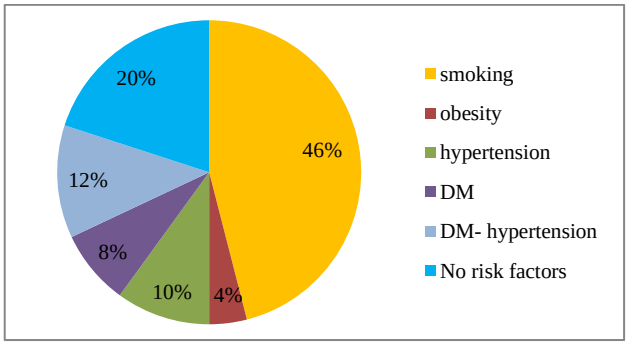

Figure 5: Risk factors among the study group.

Table 6 shows a comparison of Hs-CRP level in test and control group (mg/L). from the above result the mean concentration of Hs-CRP in CAD group is 2.51 mg/L

while that of control group is 0.76 mg/L ,That is very low as compared to test group and difference between two group is highly significant because of P value is (< 0.0001).

Table 6: Hs-CRP.

Groups	Number (n)	Mean±SD	P value	SEM
Test	100	2.51±0.79	0.0001	0.079
Control	50	0.76±0.34	-	0.04

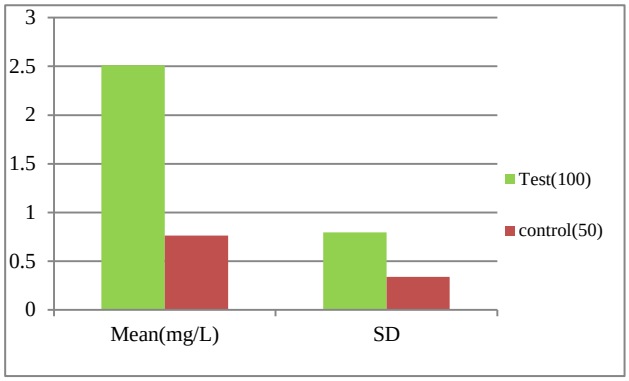

Figure 6: Hs-CRP(mg/L) level in test and control group.

Table 7 shows that CAD test group having Trop-T +ve in 100 patients while in control group were 50 have shown– ve.

Table 7: Trop-T.

Group	Number(n)	Result
Test	100	+ve
Control	50	-ve

DISCUSSION

Our results are in accordance with the previous result Pasupathi et al. These results are in close corroborating with these workers who also reported the similar observation. Liuzzo in 1994 showed that in 31 patients with severe unstable angina and no evidence of myocardial necrosis, as documented by the absence of increased cardiac troponin T, hs-CRP concentrations >3 mg/L at admission were associated with an increased incidence of recurrent angina, coronary revascularization, MI, and cardiovascular death.

We have also come across very high levels of Hs-CRP in the CAD patients and found that the value of Hs-CRP was 2.51±0.79 mg/L as compared to 0.76±0.34 mg/L in control and therefore the rise in Hs-CRP is highly significant because of p value (0.0001) similar results were found by Vahdat et al elevated Hs-CRP was significantly correlated with electrocardiogram defined coronary artery disease. A significant difference has been observed regarding the value of Hs-CRP in CHD subjects as compared to normal healthy control subjects in our study which reveals that there is an association between Hs-CRP and CHD.

CAD group having Trop-T +ve while in control group were –ve. The sensitivity of troponin-T test was found to be 100% in the studies done by Francois and Katus. While the specificities in those studies were 86% and 78% respectively, even the higher specificity of troponin-T test of 91.9% were reported by Apple. ECG diagnosis of CAD and myocardial infarction may not be evident during initials hours at all the times and it is always essential to diagnose myocardial infarction earlier for timely therapeutic intervention. CPK-MB appears in blood early but its sensitivity is lower than troponin-T test during early hours and troponin -T test is a better method to detect myocardial infarction earlier.

CONCLUSION

In a present study, Hs-CRP is highly significant in CAD patients when compared to the control group. It is an independent and better predictor of CAD.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Libby P, Ridker PM. Novel inflammatory markers of coronary risk: theory versus practice. Circulation 1999;100:1148-50.
2. Ross R. Atherosclerosis-an inflammatory disease. N Engl J Med. 1999;340:115-26.
3. Libby P. Molecular bases of the acute coronary syndromes. Circulation. 1995;91:2844-50.
4. Cybulsky MI, Gimbrone MA Jr. Endothelial expression of a mono- nuclear leukocyte adhesion molecule during atherogenesis. Science. 1991;251:788-91.
5. Ridker PM, Hennekens CH, Rifai N, Buring JE, Manson JE. Hormone replacement therapy and increased plasma concentration of C-reactive protein. Circulation. 1999;100:713-6.
6. Cushman M, Legault C, Barrett-Connor E, Stefanick ML, Kessler C, Judd HL, et al. Effect of postmenopausal hormones on inflammation-sensitive proteins: the Postmenopausal Estro-gen/Progestin Interventions (PEPI) Study. Circulation. 1999;100:717-22.
7. Kapyaho K, Welin MG, Tanner P, Karkkainen T, Weber T. Rapid determination of C-reactive protein by enzyme immunoassay using two monoclonal antibodies. Scand J Clin Lab Invest. 1989;49:389-93.
8. Kervinen H, Palosuo T, Maninnen V, Tenkanen L, Vaarala O, Mänttäri M. Joint effect or C-reactive protein and other risk factors on acute coronary syndrome. Am Heart J. 2001;141:580-5.

9. Katritis D, Korovesis S, Giazitizoglou E, Parissis J, Kalivas P, Webb-Peploe MM et al. C-Reactive protein concentrations and angiographic characteristics of coronary lesions. Clin Chem. 2000;47:882-6.
10. Patel VB, Robbins MA, Topol EJ. C-reactive protein: A golden marker' for inflammation and coronary artery disease. Cleveland Clinic J of Medicine. 2001;6(68):521-34.
11. Hull SK. How useful is high-sensitivity CRP as a risk factorfor coronary artery disease? J of Family Practice. 2005;54(3):268-9.
12. Koenig W, Sund M, Fröhlich M, Fischer H, Löwel H, Döring AA, et al. C-reactive protein, a sensitive marker of inflammation, predicts future risk of coronary heart disease in initially healthy middle-aged men. Circulation.1999;99:237-42.

Cite this article as: Rathore A, Pareek U, Sharma A. A study of high-sensitivity C- reactive protein and trop-T in patients with coronary artery disease. Int J Res Med Sci 2016;4:1051-5.