International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

IJMRP

Original Research Article

Assessment of C-Reactive Proteins in Patients with Ischemic Heart Disease

Meenakshi Puri

Senior Resident, Department of Biochemistry, PIMS, Jalandhar, Punjab, India.

ABSTRACT

Background: C-reactive protein (CRP) is one of the systemic markers of inflammation. Among young and older women, Ischemic heart disease (IHD) is one of the leading causes of death. Out of every 3 American women, one dies due to IHD. For the psychological support, little attention is thus paid to psychological support of patients. Myocardial infarction (MI) and acute angina pectoris remarkably affect quality of life of patients and their families and depict a major crisis in their life. Hence; we planned the present study to assess the relationship of CRP in patients with ischemic heart disease.

Materials & Methods: The present study included assessment of all the patients who reported with the chief complaint of sudden numbness or weakness of the face, arm or leg, sudden confusion and seizures, trouble speaking or understanding speech, sudden trouble seeing in one or both eyes, sudden trouble walking, loss of balance or coordination, sudden severe headache without any known cause. In all the patients, there was evidence of ischemic stroke which was diagnosed on imaging of brain i.e. CT scan, MRI. Venous blood sample with a disposable syringe was taken for the assessment of the levels of CRP and was sent to the laboratory for assessment. All the results were analyzed by SPSS software.

Results: Among males, 65.2 percent of the subjects had raised value of CRP while 34.8 percent of the male subjects had normal values of CRP. Among females, 49.4 percent of the subjects had raised values of CRP while among remaining females, CRP values were normal. Significant results were obtained while comparing the CRP values between males and distribution. Significant results were obtained wile comparing CRP values with various scores of MRR.

Conclusion: Significant association of the raised values of CRP does exist with the severity of ischemic stroke.

Key words: C- reactive proteins, Ischemic, Risk.

***Correspondence to:**

Dr. Meenakshi Puri,
Senior Resident,
Department of Biochemistry,
PIMS, Jalandhar, Punjab, India.

Article History:

Received: 03-01-2017, **Revised:** 17-01-2017, **Accepted:** 05-02-2017

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2017.3.2.016	

INTRODUCTION

One of the systemic dynamic marker of inflammation is C-reactive protein (CRP) which is a plasma protein synthesised by the liver. During acute responses to serious infection or major tissue damage, its concentration in the circulation can increase by up to 10 000-folds during acute responses to serious infection or major tissue damage.[1] In the absence of such spikes, however, the year-to-year within person variations in CRP concentration are similar to those in total cholesterol concentration and systolic blood pressure. Various studies of CRP have been assisted by the stability of this protein during long-term frozen blood storage and availability of standardised assays.[2,3]

Aside from whether measurement of CRP is useful in assessment of vascular risk, studies are needed to help find out if CRP is a mediator of vascular disease. CRP is present in atherosclerotic plaques in the bound form and so it has been proposed that CRP may have a causal role in coronary heart disease. Among young and older women, Ischemic heart disease (IHD) is one of the leading causes of death. Out of every 3 American women, one dies due to IHD.[4] It is among life-threatening diseases in women that presents as angina pectoris, myocardial infarction (MI), or sudden death. Admission of the MI patients is generally done in the critical care unit (CCU). Their health care mostly focuses on physiotherapy, pharmacology, and their biological response. For the psychological support, little attention is thus paid to psychological support of patients. MI and acute angina pectoris remarkably affect quality of life of patients and their families and depict a major crisis in their life.[5] Hence; we planned the present study to assess the relationship of CRP in patients with ischemic heart disease.

MATERIALS & METHODS

The present study was conducted in the department of cardiac medicine of the medical institute and included assessment of all the patients who reported with the chief complaint of sudden

numbness or weakness of the face, arm or leg, sudden confusion and seizures, trouble speaking or understanding speech, sudden trouble seeing in one or both eyes, sudden trouble walking, loss of balance or coordination, sudden severe headache without any known cause. All the patients reported from June 2014 to July 2016 were included in the present study. In all the patients, there was evidence of ischemic stroke which was diagnosed on imaging of brain i.e. CT scan, MRI. Ethical approval was taken from the institutional ethical committee and written consent was obtained after explaining in detail the entire research protocol. Complete detailed history of all the patients was taken on their admission. Special emphasis was made on the assessment of risk factors such as hyperlipidemia, hypertension, and diabetes, smoking history etc. After the fulfilment of all the management protocol in all the patients, venous blood sample with a disposable syringe was taken for the assessment of the levels of CRP and was sent to the laboratory for assessment. Any value of CRP more than 6 mg/L was considered under raised category. For the measurement of the degree of disability due to stroke, modified Rankin scale (MRR) was used. In the patients with ischemic stroke, degree of disability and value of CRP was noted. All the results were analyzed by SPSS software. Chi-square test and student t test were used for the assessment of level of significance. P-value of less than 0.05 was taken as significant.

Table 1: CRP in relation to the gender

CRP values	Gender (% of subjects)		Total	P-value
	Male	Female		
Raised	65.2	49.4	62.7	0.04*
Normal	34.8	50.6	37.3	
Total	100	100	100	

*Significant

Graph 1: CRP in relation to the gender

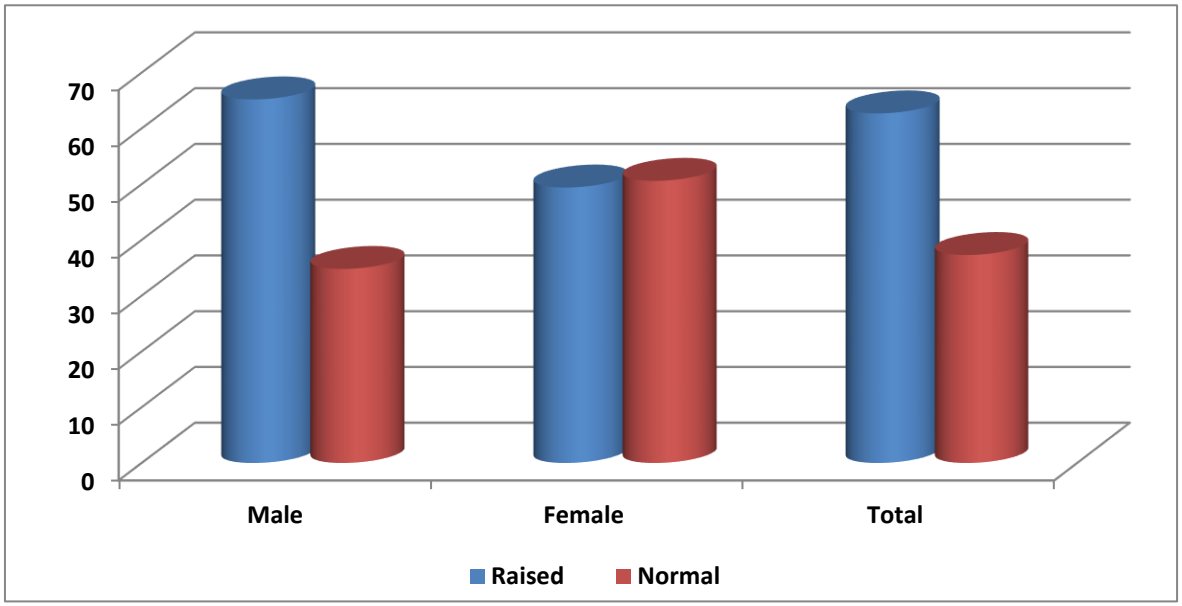
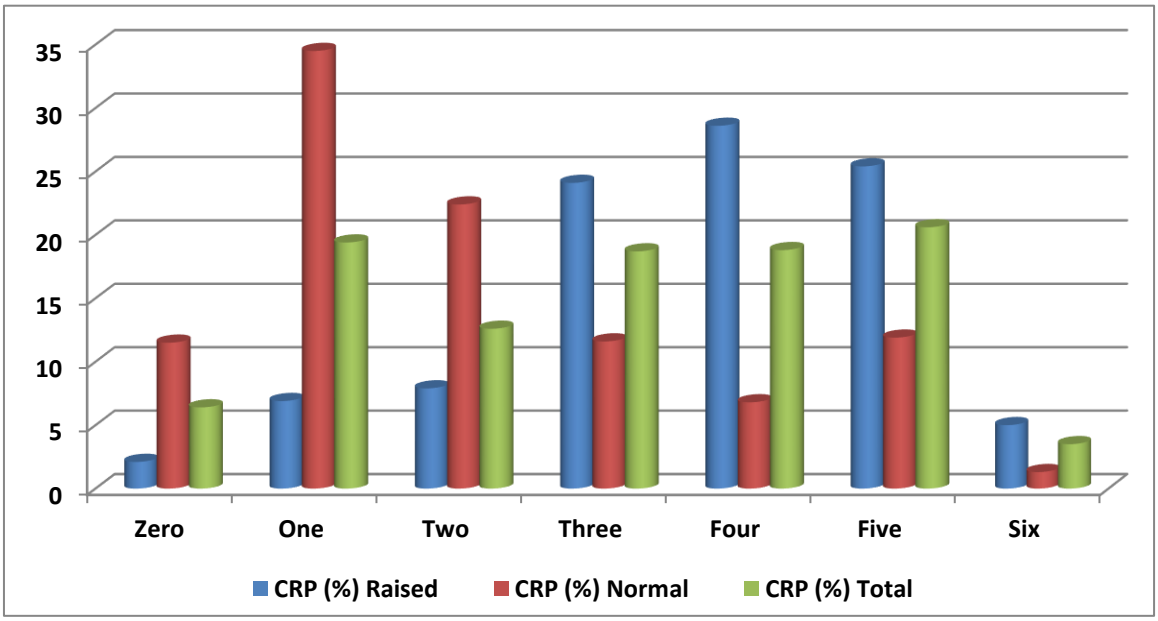


Table 2: CRP in relation to MRR

MRR score	CRP (%)			P-value
	Raised	Normal	Total	0.04*
Zero	2.1	11.5	6.4	
One	6.9	34.5	19.4	
Two	7.9	22.4	12.6	
Three	24.1	11.6	18.7	
Four	28.6	6.8	18.8	
Five	25.4	11.9	20.6	
Six	5	1.3	3.5	

*Significant

Graph 2: CRP in relation to MRR



RESULTS

Value of CRP in relation the gender is shown in Table 1 and Graph 1. Among males, 65.2 percent of the subjects had raised value of CRP while 34.8 percent of the male subjects had normal values of CRP. Among females, 49.4 percent of the subjects had raised values of CRP while among remaining females, CRP values were normal. Significant results were obtained while comparing the CRP values between males and distribution. Table 2 and Graph 2 shows the CRP values in relation to MRR. At zero MRR score, 2.1 percent of the subjects had raised CRP values while at four MRR score, 28.6 percent of the subjects had raised CRP values.

DISCUSSION

C-reactive protein (CRP) is an acute phase reactant synthesized mainly by hepatocytes in response to cytokines such as IL-6, IL1β and TNFα. Elevation of CRP is an essential component of the acute phase response to a variety of cellular insults such as infection, inflammation, tissue trauma and malignancies.[6,7] On the long arm of the chromosome 1, the genes coding for CRP have been mapped.[3,8] Basal levels of CRP are independently influenced by two polymorphisms at the CRP locus, namely CRP 2 and CRP 4 alleles. CRP binds to polysaccharides of micro-organisms and plays a role in the activation of the classical complement pathway, as well as clearance of apoptotic cells.[9] Hence; we planned the present study to assess the relationship of CRP in patients with ischemic heart disease.

In the present study, we observed that significantly high score of MRR was present with raised CRP values. Casas et al reviewed the data available on C-reactive protein and its association with coronary heart disease. Modestly elevated baseline concentrations of C-reactive protein (CRP), the classical acute phase protein, are associated with the long-term risk of coronary heart disease in general populations, whilst the major acute phase response of CRP following myocardial infarction is associated with death and cardiac complications. The pathogenic and clinical significance of these associations is controversial. Here we critically review the evidence and describe large-scale epidemiological studies, novel experiments and possible specific therapies which will rigorously inform the debate. They distinguish between the potential pathogenicity of high acute phase circulating CRP concentrations in individuals with substantial tissue damage and modest but persistent increases in baseline values in generally healthy subjects.[10,11]

Eldrup et al measured baseline plasma CRP and matrix metalloproteinase-9 in 1090 patients with stable coronary heart disease and as the primary composite endpoint detected incident unstable angina, myocardial infarction and any death during 15 years of follow-up. CRP above versus below the median of 3.0 mg/L was associated with an increased cumulative incidence of unstable angina, myocardial infarction and any death combined. Elevated CRP, but not elevated matrix metalloproteinase-9, associates with increased risk of unstable angina, myocardial infarction and death in patients with stable coronary heart disease.[12] Haim et al assessed the association between CRP and subsequent coronary risk in patients with chronic coronary heart disease (CHD). Patients with chronic CHD (n = 3122) were recruited to a secondary prevention study that assessed the efficacy of bezafibrate versus placebo. C-reactive protein was measured in plasma samples collected at prerandomization and after 2 years of follow-up. Mean follow-up time was 6.2 years. Primary end point was fatal and nonfatal myocardial infarction and sudden cardiac death. Increased baseline CRP levels were associated with increased risk of myocardial infarction, the primary end point, total death and cardiac death. After 2 years, CRP levels increased by 3.0% in the bezafibrate group and by 3.7% in the placebo group. C-reactive protein levels after 2 years were associated with increased subsequent cardiovascular risk. Baseline CRP and 2-year CRP levels were associated with subsequent risk of myocardial infarction and death in patients with chronic CHD. Bezafibrate did not reduce CRP levels as compared with placebo.[13]

Sabatine et al measured hs-CRP in 3771 patients with stable coronary artery disease from the Prevention of Events With Angiotensin-Converting Enzyme Inhibition (PEACE) trial, a randomized placebo-controlled trial of the angiotensin-converting

enzyme inhibitor trandolapril. Patients were followed up for a median of 4.8 years for cardiovascular death, myocardial infarction, or stroke, as well as new heart failure and diabetes. There were no significant interactions between hs-CRP levels and the effects of trandolapril on any of the above outcomes. In stable coronary artery disease, an elevated hs-CRP level, even >1 mg/L, is a significant predictor of adverse cardiovascular events independently of baseline characteristics and treatments. An elevated hs-CRP does not appear to identify patients with stable coronary artery disease and preserved ejection fraction who derive particular benefit from angiotensin-converting enzyme inhibition.[14]

CONCLUSION

From the above results, the authors conclude that significant association of the raised values of CRP does exist with the severity of ischemic stroke. However, future studies are recommended.

REFERENCES

1. Scirica BM, Morrow DA. Is C-reactive protein an innocent bystander or proatherogenic culprit? The verdict is still out. Circulation. 2006; 113:2128–2134.
2. Verma S, Devaraj S, Jialal I. Is C-reactive protein an innocent bystander or proatherogenic culprit? C-reactive protein promotes atherothrombosis. Circulation. 2006; 113:2135–2150.
3. de Beer FC, Soutar AK, Baltz ML, Trayner IM, Feinstein A, Pepys MB. Low density lipoprotein and very low density lipoprotein are selectively bound by aggregated C-reactive protein. J Exp Med. 1982; 156:230–242.
4. Pepys MB, Rowe IF, Baltz ML. C-reactive protein: binding to lipids and lipoproteins. Int Rev Exp Pathol. 1985; 27:83–111.
5. Zhang YX, Cliff WJ, Schoefl GI, Higgins G. Coronary C-reactive protein distribution: its relation to development of atherosclerosis. Atherosclerosis. 1999; 145:375–379.
6. Gladman D, Ginzler E, Goldsmith C, Fortin P, Liang M, Urowitz M, et al. The development and initial validation of the systemic lupus international collaborating clinics / American College of Rheumatology damage index for systemic lupus erythematosus. Arthritis Rheum. 1996; 39:363–369.
7. Jarva H, Jokiranta TS, Hellwage J, Zipfe PF, Meri S. Regulation of complement activation by C-reactive protein: targeting the complement inhibitory activity of Factor H by an interaction with short consensus repeat domains 7 and 8-11. J Immunol. 1999; 163:3957–62.
8. Vigo C. Effect of C-reactive protein on platelet-activating factor-induced platelet aggregation and membrane stabilization. J Biol Chem. 1985; 260:3418–22.
9. Bos MJ, Schipper CM, Koudstaal PJ, Witteman JC, Hofman A, Breteler MM. High serum C-reactive protein level is not an independent predictor for stroke: the Rotterdam Study. Circulation. 2006; 114:1591–1598.
10. Casas JP, Shah T, Hingorani AD, Danesh J, Pepys MB. C-reactive protein and coronary heart disease: a critical review. J Intern Med. 2008 Oct; 264(4):295-314.
11. The Emerging Risk Factors Collaboration. C-Reactive Protein, Fibrinogen, and Cardiovascular Disease Prediction. The New England journal of medicine. 2012; 367 (14): 1310-1320. doi:10.1056/NEJMoa1107477.
12. Eldrup N, Kragelund C, Steffensen R, Nordestgaard BG. Prognosis by C-reactive protein and matrix metalloproteinase-9 levels in stable coronary heart disease during 15 years of follow-up. Nutr Metab Cardiovasc Dis. 2012 Aug; 22(8):677-83.
13. Haim M, Benderly M, Tanne D, Matas Z, Boyko V, Fisman EZ C-reactive protein, bezafibrate, and recurrent coronary events in patients with chronic coronary heart disease. Am Heart J. 2007 Dec; 154(6):1095-101. Epub 2007 Sep 27.
14. Sabatine MS, Morrow DA, Jablonski KA, Rice MM, Warnica JW, Domanski MJ et al. Prognostic significance of the Centers for Disease Control/American Heart Association high-sensitivity C-reactive protein cut points for cardiovascular and other outcomes in patients with stable coronary artery disease. Circulation. 2007 Mar 27; 115(12):1528-36. Epub 2007 Mar 19.

Source of Support: Nil.

Conflict of Interest: None Declared.

Cite this article as: Meenakshi Puri. Assessment of C-Reactive Proteins in Patients with Ischemic Heart Disease. Int J Med Res Prof. 2017; 3(2):84-87. DOI:10.21276/ijmrp.2017.3.2.016