



“STUDY OF C-REACTIVE PROTEIN, SUPEROXIDE DISMUTASE LEVEL AND BIOMARKERS IN RECENTLY DIAGNOSED HYPERTENSION”

Sonia Kukreti¹, Neha Rawat²

^{1,2}Assistant Professor Faculty of Physiotherapy and Diagnostic, Jayoti Vidyapeeth Women's University, Jaipur, Rajasthan, India.

Article Info: Received 05 January 2019; Accepted 30 January. 2019

Cite this article as: Kukreti, S., & Rawat, N. (2019). “STUDY OF C-REACTIVE PROTEIN, SUPEROXIDE DISMUTASE LEVEL AND BIOMARKERS IN RECENTLY DIAGNOSED HYPERTENSION”. *International Journal of Medical and Biomedical Studies*, 3(2).

DOI: <https://doi.org/10.32553/ijmbs.v3i2.85>

Address for Correspondence: Sonia Kukreti, Assistant Professor Faculty of Physiotherapy and Diagnostic, Jayoti Vidyapeeth Women's University, Jaipur, Rajasthan, India.

Conflict of interest: No conflict of interest.

Abstract

Acute myocardial infarction (AMI) is a significant cause of morbidity and mortality worldwide, which results from occlusion of coronary artery. C-reactive protein (CRP) is an acute phase protein, synthesized by hepatocytes in response to cytokines released into circulation by activated leukocytes. It is a sensitive marker of coronary inflammation as well as the extent of myocardial necrosis. We incorporated all the ambulant patients (over 18 years old) of both sex having hypertension and normotension going to in the general outpatient office (OPD) for one month. CRP measurement has many advantages in detection and monitoring the acute phase response. This study showed that mean serum CRP levels were increased in the study group. Among two subgroups, mean CRP level increased significantly in the group with risk factors when compared with another group. Most of the male subjects were in 51 – 60 years old and the female were in 41 - 50 years. Among 75 of male subjects, 64% were hypertensive, and among 35 females 61% were hypertensive. Mean CRP level in normotensive gathering was 1.072 mg/L (\pm 0.320) and in hypertensive gathering was 2.824 mg/L (\pm 0.258). The individuals who created LVH (n=75, 51.02%), their gauge CRP was 2.767 mg/L (\pm 0.318).

Key words: Acute myocardial infarction, C-reactive protein, Hypertension, superoxide dismutase.

Introduction:

C-reactive protein (CRP) is an acute phase protein, synthesized by hepatocytes in response to cytokines released into circulation by activated leukocytes. It is a sensitive marker of coronary inflammation as well as the extent of myocardial necrosis. CRP measurement has many advantages in detection and monitoring the acute phase response. As cardiovascular sicknesses are presently considered as an outcome of incessant provocative process, CRP is

generally used to foresee the danger of creating complications.^{8,9} As cardiovascular intricacy is regular in numerous people without having ordinary hazard factors (e.g. corpulence, family ancestry, hyperlipidemia), job of CRP is progressively assessed for screening these individuals.^{10,11} Hypertension is one of the cardiovascular ailments in charge of much bleakness and mortality all through the world¹⁰⁻¹³ and has been turned out to be an outcome of interminable aggravation of the blood vessel wall.^{11,13} So it very well may be accepted that

CRP has a relationship with hypertension, however it is as yet disputable whether hypertension is the aftereffect of raised CRP.^{4-6,9,10}

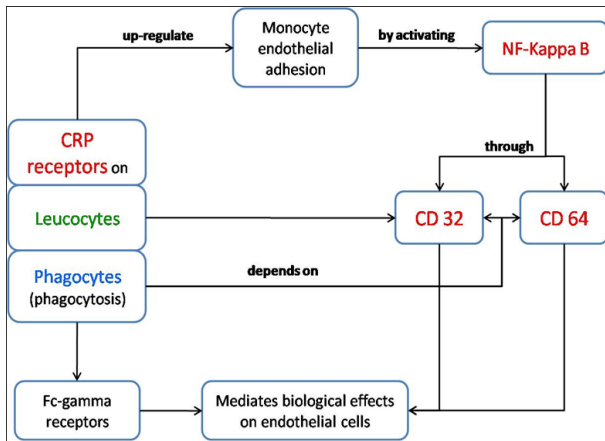


Figure 1: Role of CRP receptors

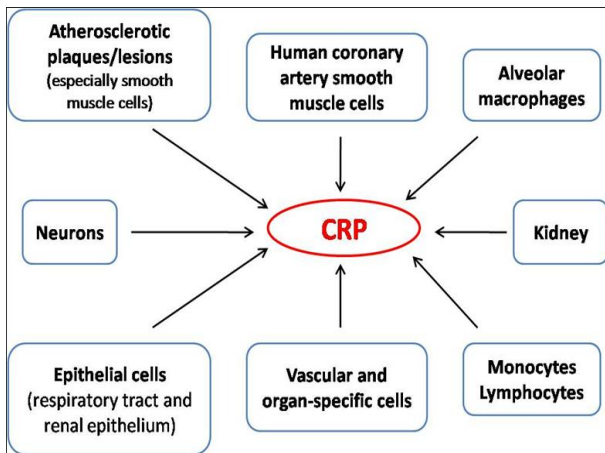


Figure 2: Extrahepatic sites of CRP production

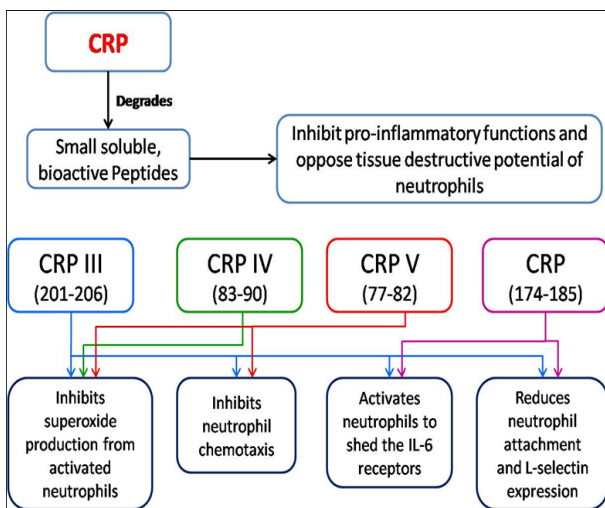


Figure 3: Degradation products of CRP and their functions

2. MATERIALS AND METHODS

This imminent observational investigation was completed in a tertiary dimension medical clinic on outpatient with the essential target of finding any connection among CRP and hypertension, and auxiliary goal of finding prescient estimation of CRP in hypertensive entanglements. We incorporated all the ambulant patients (over 18 years old) of both sex having hypertension and normotension going to in the general outpatient office (OPD) for one month. We exclude known instances of ischemic coronary illness (IHD), analyzed instance of any irresistible and incendiary condition (e.g. fever because of any reason, urinary contamination, symptomatic joint pain, and so on), mental patients, the individuals who had uncontrolled diabetes mellitus (DM) (HbA1C > 7.0%) and the individuals who were reluctant to partake in the examination. The target of the investigation was talked about in subtleties with the patients or their orderlies previously their choice to select themselves into the examination and composed assent was taken. Subject's age, sexual orientation, therapeutic and clinical history was gathered before conduction of study. Hypertension was analyzed by physically estimating circulatory strain with aneroid sphygmomanometer on the spot alongside gathering information of past three months from their therapeutic record books. Hypertension was viewed as when either systolic circulatory strain (SBP) was more than 120 mmHg or diastolic pulse (DBP) was more than 80 mmHg, or both. Clinical examination was done and pattern CRP was sent. CRP was estimated in BN ProSpec by Nephelometry and was communicated as mg/L. Ordinary range is under 6 mg/L. Information was gathered in a pre-planned organized information accumulation sheets. Both hypertensive and normotensive gatherings were followed up following a half year. By this period, we appropriately offered administration to the hypertensive patients. Following a half year, ECG was done in each patient of the two gatherings to distinguish left ventricular hypertrophy (LVH)

and ischemic coronary illness (IHD). Explicit examinations (chest X-beam, troponin-I, echocardiogram) were done just in the individuals who grumbled about angina or highlights of heart disappointment (dyspnea or hack on lying). All the applicable gathered information were accumulated on an ace outline first and then sorted out by utilizing logical determined and standard factual recipes, rate was determined to discover the extent of the discoveries. Information section and examination were finished utilizing SPSS for windows variant 22.0. Yield of information and graphical portrayal was finished utilizing Microsoft Office outline and Microsoft-Word. P-esteem was considered as noteworthy when it was <0.05 . The outcomes were displayed in tables, figures, outlines and so on.

3. RESULT AND DISCUSSION

Our complete examination populace was 110, among them 70% (n=75) were male and 32.0% (n=35) were female. Most of the male subjects were in 51 – 60 years old and the female were in 41 - 50 years. Among 75 of male subjects, 64% were hypertensive, and among 35 females 61% were hypertensive. Mean CRP level in normotensive gathering was 1.072 mg/L (± 0.320) and in hypertensive gathering was 2.824 mg/L (± 0.258). The individuals who created LVH (n=75, 51.02%), their gauge CRP was 2.767 mg/L (± 0.318). The individuals who created heart disappointment (HF) (n=2, 2.78%), their mean CRP was 3.108 mg/L (± 0.121). The individuals who created angina pectoris (n=14, 20.01%), their mean CRP was 3.062 mg/L (± 0.238). Nonetheless, no noteworthy distinction in CRP level [2.892 mg/L (± 0.234)] was seen in those hypertensive patients (n=21, 30.01%) who did not build up any type of confusion. We didn't get any relationship between age or sex and dimension of CRP.

In our investigation we got two noteworthy discoveries: First, hypertension is related with higher CRP level. Second, raised CRP level isn't related with hypertensive difficulties. We

additionally have some auxiliary discoveries: Frequency of hypertension is more in male than in female, yet female created hypertension sooner than male. It is as of now demonstrated in numerous epidemiological examinations that atherosclerosis is a fiery procedure and may prompt hypertension and other cardiovascular diseases.³⁻¹¹ So there was presumption that hypertensive people ought to have higher CRP. Like other studies^{10,14-16} we likewise got similar discoveries: Hypertensive subjects have higher CRP than those of normotensive subjects. There are numerous examinations completed to discover the connection whether more elevated amount of CRP may prompt hypertensive entanglements, yet regardless it stays questionable. High CRP was observed to be related especially with IHD.^{3,16-18} However, an ongoing report (n = 476) demonstrated that high CRP is related with safe hypertension and hence has more regrettable cardiovascular prognosis.¹⁹ We have not discovered any noteworthy relationship between high CRP with hypertensive confusions. Numerous hypertensive subjects in our investigation have not built up any hypertensive entanglements however they have high CRP like the individuals who created inconveniences. Male has higher recurrence of hypertension than female.^{20,21} But, those huge epidemiological investigations demonstrated that most female has created hypertension in around fifth and 6th decade. We found that most female has hypertension in their fourth decade. This is likely a direct result of our single focus based investigation with referral inclination. We have a few impediments in our investigation. To begin with, this is a solitary focus based observational investigation which has much referral biasness. Second, IHD stays quiet in numerous old diabetic patients, and this can without much of a stretch raise dimension of CRP. Usually numerous patients have attendant hypertension, DM and IHD. Furthermore, CRP can be raised increasingly because of DM and IHD.¹⁴ As we barred IHD just by side effect and ECG discoveries, there was high possibility of missing many genuine instances of IHD. All things

considered, our examination has a few qualities. In the first place, this is an a lot bigger examination in our nation contrasting CRP in both hypertensive and normotensive subjects. Second, we pursued the hypertensive gathering for somewhere around a half year. Third, we prohibited every diabetic patient whose HbA1C is over 7.0% to limit the mistake in estimating CRP.

In any case, because of the critical restrictions referenced above, a lot bigger populace based investigations are expected to distinguish whether dimension of CRP is really connected with hypertensive complexities, and along these lines we can foresee ahead of time to keep those grimness and mortality.

Table 1: (Mean±SD), P Value among cases and controls

Parameter	Cases (Mean±SD) n = 75	Controls (Mean±SD) n =75	P
CRP in male population	2.824 mg/L (± 0.258).	1.072 mg/L (± 0.320)	<0.01

Table: 2 (Mean±SD), P Value among cases and controls

Parameter	Cases (Mean±SD) n = 35	Controls (Mean±SD) n =35	P
CRP in female population	2.767 mg/L (±0.318).	1.058 mg/L (± 0.330)	<0.01

Table3: (Mean±SD), P Value among subgroup (cases with risk factor and without risk factor)

Parameter	(Mean±SD) n = 75 with risk factor	(Mean±SD) n =75 without risk factor	P
CRP in male population	12.62 mg/L (± 3.10)	5.58 mg/L ± 1.28)	<0.02

Table 4: (Mean±SD), P Value among subgroup (cases with risk factor and without risk factor)

Parameter	(Mean±SD) n = 35 with risk factor	(Mean±SD) n =35 without risk factor	P
CRP in female population	11.42 mg/L (± 2.20)	6.23 mg/L ± 1.35)	<0.02

4. CONCLUSION:

CRP is a valuable hypertensive biomarker in various clinical conditions. However, being non-specific, its use is limited. In view of these,

guidelines are necessary to interpret the CRP levels in a clinical context. Standardization of measurement techniques and reporting should improve the utility of CRP in regular clinical

practice. Hypertension is related with higher CRP level. Second, raised CRP level isn't related with hypertensive difficulties. Hypertensive subjects have higher CRP than those of normotensive subjects. There are numerous examinations completed to discover the connection whether more elevated amount of CRP may prompt hypertensive entanglements, yet regardless it stays questionable. High CRP was observed to be related especially with IHD.

REFERENCES

1. Bray C, Bell LN, Liang H, Haykal R, Kaiksow F, Mazza JJ et al. Erythrocyte Sedimentation Rate and C - reactive protein Measurements and Their Relevance Clinical Medicine. *WMJ* 2016;115(6):317-21
2. Lloyd-Jones DM, Liu K, Tian L, Greenland P. Assessment of C - reactive protein in Risk Prediction for Cardiovascular Disease. *Annals of Internal Medicine* 2006;145:1 35-42.
3. Ingle PV, Patel DM. C-reactive protein in various disease condition an overview. *Asian J Pharm Clin Res* 2011;4(1):9-13.
4. Pasceri V, Cheng JS, Willerson JT, Yeh ET, Chang J. Modulation of C-reactive protein mediated monocyte chemoattractant protein-1 induction in human endothelial cells by antiatherosclerosis drugs. *Circulation* 2001;103:2531-4.
5. Pasceri V, Willerson JT, Yeh ET. Direct proinflammatory effect of C-reactive protein on human endothelial cells. *Circulation* 2000;102:2165-8.
6. Torzewski M, Rist C, Mortensen RF, Zwaka TP, Bienek M, Waltenberger J et al. C-reactive protein in the arterial intima: role of C-reactive protein receptor-dependent monocyte recruitment in atherogenesis. *Arterioscler Thromb Vasc Biol* 2000;20 :2094
7. Mackiewicz A, Speroff T, Ganapathi MK, Kushner I. Effects of cytokine combinations on acute phase protein production in two human hepatoma cell lines. *J Immunol* 1991;146 :3032-7.
8. Adukauskiene D, Ciginskiene A, Adukauskaite A, Pentiaginiene D, Slapikas R, Ceponiene R. Clinical relevance of high sensitivity C-reactive protein in cardiology. *Medicina* 2016;52: 1-10.
9. Cozlea DL, Farcas M, Nagy A, Keresztesi AA, Tifrea R, Cozlea L et al. The Impact of C Reactive Protein on Global Cardiovascular Risk on Patients with Coronary Artery Disease. *Curr Health Sci J* 2013;39(4):225-31.
10. Clearfield MB. C - reactive protein: A New Risk Assessment Tool for Cardiovascular Disease. *J Am Osteopath Assoc* 2005;105(9):409-16.
11. Victor RG. Systemic Hypertension: Mechanisms and Diagnosis. In: Mann DL, Zipes DP, Libby P, Bonow RO, editors. *Braunwald's Heart Disease*. 10th ed. Philadelphia: Elsevier Saunders; 2015. p.939.
12. Newby DE, Grubb NR, Bradbury A. Cardiovascular disease. In: Walker BR, Colledge NR, Ralston SH, Penman ID, editors. *Davidson's Principles & Practice of Medicine*. 22nd ed. Edinburgh:Churchill Livingstone; 2014. P. 607-13.
13. Ridker PM: Moving beyond JUPITER: Will inhibiting inflammation reduces vascular event rates? *Curr Atheroscler Rep* 2013; 15:295.
14. Lima LM, Carvalho MDG, Soares AL, Sabino AD, Fernandes AP, Novelli BA et al. High-Sensitivity C-Reactive Protein in Subjects with Type 2 Diabetes Mellitus and/or High Blood Pressure. *Arq Bras Endocrinol Metab* 2007;51(6):956-60.
15. Blake GJ, Ridker PM. High sensitivity C-reactive protein for predicting cardiovascular disease: an inflammatory hypothesis. *European Heart Journal* 2001; 22: 349-52.
16. Blake GJ, Rifai N, Buring JE, Ridker PM. Blood Pressure, C-Reactive Protein, and Risk of Future Cardiovascular Events. *Circulation* 2003;108:2993-9.
17. Fonseca FAH, Izar MCO. High-Sensitivity C - reactive protein and Cardiovascular Disease Across Countries and Ethnicities. *Clinics* 2016;71(4):235-42. 5 *Bangladesh Crit Care J* March 2018; 6 (1): 3-6

18. Li Y, Zhong X, Cheng G, Zhao C, Zhang L, Hong Y et al. Hs-CRP and all-cause, cardiovascular, and cancer mortality risk: A meta-analysis. *Atherosclerosis* 2017;259:75-82.
19. Cortez AF, Muxfeldt ES, Cardoso CRL, Salles GF. Prognostic Value of C - reactive protein in Resistant Hypertension. *Am J Hypertens* 2016;29(8):992-1000.
20. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults. *JAMA* 2002;288:1723-7.
21. Stamler J, Stamler R, Riedlinger WF, Algera G, Roberts RH: Hypertension screening of 1 million Americans. *Community Hypertension Evaluation Clinic*.