ORIGINAL RESEARCH

A Study of C - Reactive Protein in Essential Hypertension

J. Prabhakar Patro[1], M. Nagamallika[2], Vinayak Shegokar[3]

ABSTRACT

Introduction: Markers of inflammation like C reactive protein (CRP), cytokines like interleukin – 6 (IL 6) and Tumour Necrosis Factor (TNF) can be measured in plasma and they provide a window on the inflammatory process at the level of arterial wall. Systemic hypertension also cause significant inflammation, which will result in raised C reactive protein (CRP) levels. Objective of the study wasto evaluate link between C-reactive protein and essential hypertension.

Material and method: The present study was Carried out at Government Medical College and Hospital Nagpur, Department of Medicine from January 2004 to June 2005.Patients who were getting treatment for primary hypertension were included in study.

Results: It was found that serum CRP was significantly higher in male and females in cases as compare to control group. There was positive correlation between CRP and Age in cases. Mean CRP was significantly higher in smokers. CRP was significantly higher in alcoholic and nonalcoholic in cases. Mean CRP was significantly higher in normal BMI and BMI $\geq$ 25 kg/m^2 in cases.

Conclusion: CRP estimation is an easy, cheap and cost effective method of monitoring in essential hypertension. CRP has role in the prediction, diagnosis and management of hypertension. CRP had significant correlation with staging of hypertension.

Keywords: Hypertension, CRP, target organ damage

How to cite this article: J. Prabhakar Patro, M. Nagamallika, Vinayak Shegokar. A study of C - reactive protein in essential hypertension. International Journal of Contemporary Medical Research 2015;2(4):1055-1058

[1]Professor, [2]Associate Professor, Department of General Medicine, Malla Reddy Institute of Medical Sciences, Hyderabad [3]Professor, Department of General Medicine, C. M. Medical College, Durg, Chhattisgarh, India

Corresponding author: Dr. J. Prabhakar Patro, Professor, Department of General Medicine, Malla Reddy Institute of Medical Sciences, Hyderabad, India

Source of Support: Nil

Conflict of Interest: None

INTRODUCTION

Hypertension is adequately controlled in fewer than 30% of all hypertensive in the United States. Patients noncompliance with medical therapy accounts, at least in part, for the failure to control high BP. It has been estimated that 50% of hypertensive patients fall to keep follow-up appointments and only 60% follow their prescribed medication regimens.[1]

Hypertension kills by producing the target organ damaged and its complications. In the 1950s Perera[2] observed that among 500 untreated patients 74% of these complications were cardiac in nature, 43% were renal, and 32% were retinal. More than half the subjectsdied of heart disease (Principally congestive heart failure), 10% to 15% died of cerebral accidents, and about 10% died of renal failure. Malignant hypertension occurred in fewer than 5% of these patients.

Markers of inflammation like C reactive protein (CRP),cytokines like interleukin – 6 (IL 6) and Tumour Necrosis Factor (TNF) can be measured in plasma and they provide a window on the inflammatory process at the level of arterial wall. Systemic hypertension also cause significant inflammation, which will result in raised C reactive protein (CRP) levels.[11]

On the basis of this, Present study is undertaken to evaluate link between C-reactive protein and essential hypertension.

MATERIAL AND METHODS

Study Setting

The present study "A Study of C-Reative Protein in Essential Hypertension" was Carried out at Government Medical College and Hospital Nagpur, Department of Medicine from January 2004 to June 2005.

Selection criteria

Patients who were getting treatment for primary hypertension from GMC Nagpur-Medicine OPD or cardiology clinic or who were admitted in medicine ward.

Exclusion criteria

Conditions known to be associated with raised CRP levels and the conditions which leads to secondary hy-

pertension excluded:

- Coronary artery disease
- Diabetes mellitus
- Stroke
- Collagen Vascular Disease
- Hepatic and renal diseases
- Trauma
- Post operative patients
- Any acute or chronic infections
- Malignancy
- Hyperlipidemias
- Drugs – oral contraceptives

Study Design

Present study is a case-control study. Patients were selected and evaluated thoroughly with details of clinical history, examination, routine investigation.

STATISTICAL ANALYSIS

Two groups were made as cases and controls. Hypertensive subjects taken as cases and healthy normotensive subjects taken as controls.Two groups will be compared for CRP levels according to risk factors and staging of hypertension in cases and controls.Continuous variables are presented as mean ±SD and categorical variables are presented in percentage. The data was analyzed using statistical software Stata version 7.0. the student 't' test used for comparison between means of two continuous variables, and Chi-Square test for comparing two groups of categorical variables. $P < 0.05$ were considered as statistical significant.

In control group 10 (13.3%) patients were smokers; 7 (9.3%) patients were alcoholics and 11 (14.7%) patients had BMI ≥ 25mg/m^2.

In cases 22 (29.3%) patients were smokers; 18 (24.3%) patients were alcoholics and 23 (30.7%) patients had BMI ≥ 25 Kg/m^2.

Smokers, BMI ≥25 Kg/m^2 and Alcoholics found to be independent risk factor for hypertension. ($p< 0.05$).

In control group the mean CRP was 0.12 ±0.25 mg/dl in male and 0.06 ± 0.81 mg/dl in females, while it was 0.95 ±0.76 mg/dl in males and 0.81 ± 0.7 mg/dl in females in cases.

It was found that serum CRP was significantly higher in male and females in cases as compare to control group. ($P < 0.001$).

In control group mean CRP was 0.09 ± 0.22 mg/dl in the mean age group of 50.05 ± 11.61 years where as it was 0.89 ± 0.74 mg/dl in the mean age group of 49.58 ± 11.80 years in cases.

It was found that there was positive correlation between CRP and Age in cases ($r = 0.3264$; $p < 0.005$).but it not significant in controls ($r=0.1303$; $p=0.2651$)

In control groups mean CRP was 0.32 ± 0.31 mg/dl in smokers and 0.04=0.15 mg/dl in nonsmokers whereas it was 1.28 ±0.75 mg/dl in smokers and it was 0.74 ± 0.68 mg/dl in nonsmokers in cases.

It was found that mean CRP was significantly higher in smokers and nonsmokers in cases ($p < 0.001$) as compared to controls:

In alcoholics mean CRP was 0.34±0.32 mg/dl in controls and 1.67±0.64 mg/dl in cases whereas in nonalcoholics it was 0.071±0.19 mg/dl in controls and 0.65±0.59mg/dl in cases.

It was found that mean CRP was significantly higher in alcoholic and nonalcoholic in cases as compared to controls. ($p<0.001$)

In controls in BMI ≥ 25 kg/m^2 mean CRP was 0.27 ± 0.31 mg/dl and. It was 0.06 ± 0.018mg/dl in BMI ≤ 25 mg/m^2. Incases mean CRP was 1.43±0.07mg/dl in BMI≥25kg/m^2 whereas it was 0.66±0.62 mg/dl in BMI ≤ 256 kg/m^2 in cases. It was found that mean CRP was significantly higher in normal BMI and BMI ≥ 25 kg/m^2 in cases as compared to controls ($p < 0.001$)

In controls CRP was 0.09 ±0.22 mg/dl mean SBP/DBP, whereas it was 0.89±0.74 mg/dl in mean SBP / DBP in cases.

It was found that there was statistically significant correlation between Systolic Blood Pressure and CRP and Diastolic Blood Pressure and CRP in cases ($p<0.001$) as compare to controls.

DISCUSSION

Mean CRP was low in younger patients i.e., 0.48 ± 0.63 mg/dl and it progressively increased in order subjects, being highest in 6.-69 yrs age group ($p < 0.005$) in the present study.

Mario D. Nopoli et al[4] made three groups of < 70.70 – 79, and ≥ 80 yrs found similar observation, that it was more among older person.

Howard D. Sesso et al[5] in their study of 20525 females US health professional aged 45 or older found similar observations that as the mean age increased level of CRP also increased.

22 patients out of 75 were smokers in hypertensive group while 15 patients were smoker out of 75 in controls group.

Mean CRP was higher in hypertensive group in both smokers and nonsmokers as compared to control group ($p < 0.001$). Mean CRP was higher in smokers than nonsmokers in hypertensive group.

Howard D. Sesso et al[5] and Jerme L. Abranom et al[6] in

Risk Factors	Controls (n=75)	Cases (n=75)	OR	95% CI	p-value
Smokers	10(13.3%)	22(29.3%)	2.69	1.10-6.92	0.0168
Alcoholics	7(9.3%)	18(24%)	3.06	1.11-9.26	0.0160
Body mass index $\geq$ 25 Kg/m^2	11(14.7%)	23(30.7%)	2.57	1.07-6.38	0.0193

Table-1: Risk Factors

Sex	Means CRP in Controls (n=75)	Mean CRP in Cases (n=75)	P value
Male (n=46) 61.3%	0.12$\pm$ 0.25	0.95 $\pm$ 0.76	P < 0.001
Females (n=29) 38.7%	0.06 $\pm$ 0.18	0.18 $\pm$ 0.70	P < 0.001

Table-2: Gender distribution of CRP in controls and cases

Group	Mean age (years)	Mean CRP (mg/dl)	R value	P value
Controls (n=75)	50.05$\pm$ 11.61	0.09$\pm$ 0.22	R=0.1303	P=0.2651 (NS)
Cases (n=75)	49.58$\pm$ 11.50	0.89$\pm$ 0.74	E=0.3264	P=0.0042 (HS)

Table-3: Correlation between CRP and age in controls and cases

Risk factors	No. of patients in		CRP (mg/dl)		P value
	Controls	Cases	Controls	Cases	
Smokers	10 (13.3%)	22(29.33%)	0.30$\pm$ 0.31	1.28$\pm$0.75	P < 0.001
Nonsmokers	65 (86.7%)	53 (70.66)	0.06$\pm$0.18	0.74$\pm$0.68	P < 0.001

Tables-4: Correlation of CRP with risk factors (Smoking)

Risk factors	No. of patients in		CRP (mg/dl)		P value
	Controls n=75	Cases n=75	Controls	Cases	
Alcoholic	7(9.03%)	18 (24%)	0.34$\pm$0.32	1.67$\pm$0.64	P < 0.001
Nonalcoholic	68(90.7%)	57(76%)	0.07$\pm$0.19	0.65$\pm$0.59	P < 0.001

Table-5: Correlation of CRP with risk factors like alcoholic among controls and cases

Risk factors	No. of patients in		CRP (mg/dl)		P value
	Controls n=75	Cases n=75	Controls	Cases	
BMI$\geq$25kg/m^2	11 (14.7%)	23(30.7)	0.27$\pm$ 0.31	1.43$\pm$0.71	P < 0.001
BMI$\leq$25kg/m^2 (Normal wt)	64(85.3%)	52(69.31)	0.06$\pm$0.18	0.66$\pm$0.62	P < 0.001

Table-6: Co-relation of CRP with BMI in controls of cases

Group	Mean BP (mmhg)	CRP (mg/dl)	E value	P value
Controls (n=75)	Mean SBP (114.045$\pm$5.12) Mean DBP (74.11$\pm$3.06)	0.09$\pm$ 0.22	r = 0.0895 r=0.0392	0.476 0.738
Cases (n=75)	Mean SBP (155$\pm$19.15) Mean DBP (94$\pm$9.19)	0.89$\pm$0.74	r=0.8026 r=0.8294	P<0.001 P<0.001

Table-7: CRP Distribution of mean SBP and mean DBP among cases and controls

their study concluded similar observations.

18 patients out 75 were alcoholics in hypertensive group while 12 subjects out of 75 were locoholics in control group.

In the present study mean CRP was higher in alcoholic and nonalcoholics in hypertensive group as compared to control group ($p < 0.001$) also, CRP was higher in alcoholics than non-alcoholic in hypertensive group.

Howard D. Sesso et al[5] concluded similar observations in hypertensive group. But Jerome L. Abroham et al[6] was found that participants with high CRP level had a lower frequency of alcohol consumption, lower HDL cholesterol and engaged in physical activity less frequently.

Mean CRP was more in subjects of BMI > 25kg/m^2 in hypertensive group as compared to normotensive group ($p < 0.001$) also mean CRP was more in subjects of BMI > 25 kg/m^2 than BMI< 25 kg/m^2 in hypertensive group ion the present study.

Schillacl et al[7] concluded similar observation during their study of 135 hypertensive subjects and 40 healthy metched nonhypertensive controls. CRP was

also directly associated with body mass index (r=0.25, p<0.01)

Marjoelin Visser et al[8] concluded similar opinion that higher BMI was associated with higher CRP concentration which suggest a state of low-grade systemic inflammation in overweight and obese persons.

Howard D. Sesso et al[5]; Baustista et al[9], KiChul Sung et al[10] were made similar observations.

In the present study mean CRP was higher in mean SBP and mean DBP in hypertensive group as compared to normotensive group (p< 0.001) and CBP had a significant direct association with systolic blood pressure and diastolic blood pressure in hypertensive group. (r=0.8026; p=0.0000; and r=0.8294; p=0.0000 resp.)

However there was no statistically significant between CRP and SBP and DBP in control group (r=0.0835, p=0.4762; r=0.0392, p=0.7386 resp.)

Schillaci et al[7] observed similar resulted. Plasma CRP concentration was greater in hypertensive individuals (1.85 mg/dl, interquartile range 0.74 – 3.64) than in control individuals (1.01 mg / dl, interquartile range 0.67 – 1.88; p=0.02). CRP had a significant direct associated with systolic blood pressure and pulse pressure but not with diastolic pressure.

Ki Chul Sung et al[10] observed similar results as that of present study. According to their study CRP value showed a significant degree of correlation with systolic and diastolic blood pressure.

CONCLUSION

CRP estimation is an easy, cheap and cost effective method of monitoring in essential hypertension.CRP has role in the prediction, diagnosis and management of hypertension.CRP had significant correlation with staging of hypertension.

REFERENCES

1. Rizzo Ja: Economics and patient noncompliance: Looking at the real world.Cardiol Rev 13:21 -26, 1996.
2. Phillips SJ, Whisnant JP:Hypertension and stroke. In Laragh JH, Brenner BM (eds): Hypertension: Pathophysiology, Diagnosis, and Management, 2nded. Raven Press, New York, 1995, pp 465-480.
3. C-Reactive Protein Test. A journal Report, Heart Centure 21/5/2005
4. Mario Di Napoli, Francesca Papa, et alAssociation between blood pressure and C-reactiveprotein levels in acute ischemic stroke.Hypertension. 2003;42:1117-1123.
5. Howard D, Sesso, Julie E, Buring, et alC-reactive protein and the risk of developing hypertension. JAMA 2003;290:2945-2951.
6. Jerome L, Abramson; William S, Weintraub; Viola VaccarinoAssociation between pulse pressure and C-reactive Protien among apparently healthy US adults. Hypertension 2002;39:197.
7. Marjoleinvisser, lex M. Bouter, et alElevated C-reactive protein levels in overweight and obese adults. JAMA. 1999;282:2131-2135.
8. Schillaci, Giuseppe; Pirro, Matteo, et alIncreased C-reactive protein concentrations in nevertreated hypertension: the role of systolic and pulse pressures. Journal of hypertension. Oct 2003;21:1841-1846.
9. Bautista, Leonelo E, Lopez-jaramillo, Patricio, et al. Is C-Reactive protein an independent risk factor for essential hypertension? Journal of Hypertension: May 2001 vol. 19: 857-861.
10. Ki chul Sung, Jung YulSuh, Bum Soo Kim, et al High sensitivity C-reactive protein as an independent risk factor for essential hypertension. AJH 2003;16:429-433.