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Original Research Article

A Comparative Study of the Level of High Sensitive C-Reactive Protein in People with and without Hypertension

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Abstract:

Background: High sensitivity C-reactive protein (hs-CRP), one of the hepatic acute phase reactants, has been linked to reduced endothelium-dependent relaxation, which may be a risk factor for hypertension. The purpose of this study is to determine whether circulating CRP levels are connected with hypertension.

Materials and Methods: The study included 50 patients with essential hypertension taking antihypertensive drugs, and 50 normotensive volunteers between the ages of 40 and 60 years, both sexes The blood pressure was measured with a Mercury sphygmomanometer and the hs-CRP was tested using the CRP HS ELISA.

Results: The patient group had significantly higher hs-CRP values (8.26±2.76) compared to the control group (3.28±1.42).

Conclusion: Our study found that hypertensive individuals had higher levels of hs-CRP, indicating the role of inflammation in the pathophysiology of hypertension.

Keywords: high-sensitivity C-reactive protein, hypertension, inflammation.

Introduction

Because of its prevalence and the associated risks of cardiovascular and renal disease, hypertension is a significant global public-health concern.[1,2] Hypertension is estimated to be the fourth leading cause of early death in affluent countries and the seventh in underdeveloped countries.[3] In India, hypertension causes 57% of stroke deaths and 24% of coronary heart disease deaths.[4] Because of its high mortality and morbidity, early diagnosis and effective prevention are essential. C-reactive protein (CRP) is an acute phase reactant generated by the liver when interleukin-6 is stimulated. It is commonly used to assess a variety of inflammatory conditions.[5.6]

C-Reactive Proteins (CRP) are found in trace amounts in healthy people's plasma, but their concentration rises 100 times in response to injury, infection, or inflammation. CRP is named because its capacity to precipitate the somatic C-polysaccharides of Streptococcus pneumoniae and is the first acute phase protein to be described.[7,8] CRP is largely produced by the liver in response to IL-6 and IL-1ß. It is a suitable biological marker since it is stable, has a half-life of 19 hours, and has little fluctuation between fresh and frozen forms.[9.10]

Chronic vascular inflammation plays a role in the beginning and progression of essential hypertension, either as a primary or secondary cause. Inflammatory mediators such CRP, IL-1β, IL-6, TNF-α, and reactive oxygen species have been linked to essential hypertension through many mechanisms, including increased arterial stiffness and endothelial dysfunction [11]. hs-CRP is implicated in vascular inflammation and plays an important role in the progression and development of atherosclerosis. As a result, it is important to investigate the link between serum high sensitive C-reactive protein (hs-CRP) and hypertension.

Materials and Methods:

A cross-sectional study was undertaken at the Department of Physiology, Government Medical College, Mancherial,Telangana for a duration of six months. The study involved a sample of one hundred participants. Fifty hypertensive subjects and fifty normal subjects aged 40 to 60 years who satisfied the inclusion and exclusion criteria were matched in age and BMI.

The subjects were equally divided into two groups. Group 1 (n = 50) comprised hypertensive individuals while subjects in Group 2 (n = 50) were Healthy normal. Informed consent was collected from all the subjects and ethical clearance was obtained from the Institutional Ethics Committee.

Inclusion Criteria

1. Participants aged 40 to 60 years old.
2. Both males and females.
3. Case subjects having hypertension.
4. Individuals with normal blood pressure (defined as systolic blood pressure <120 mmHg and diastolic blood pressure <80 mmHg) as control.
5. Availability of highly sensitive C-reactive protein (hsCRP) data.
6. Participants who have provided informed consent to participate in the study.

Exclusion Criteria

1. Participants with a history of diagnosed hypertension or antihypertensive medication use, history of cardiovascular diseases (e.g., myocardial infarction, stroke, congestive heart failure).
2. Participants with a history of diabetes mellitus, chronic kidney disease, Pregnant women or women who are breastfeeding, known inflammatory conditions (e.g., rheumatoid arthritis, lupus), liver diseases (e.g., cirrhosis, hepatitis), known endocrine disorders (e.g., thyroid dysfunction, Cushing's syndrome), malignancies.

Height

During the recording, the subject stood barefoot on a flat surface. In order to estimate height to the nearest 0.1 cm, we measured the uppermost point on the head that produces enough pressure on the hair to bandage it to the the bottom of the feet

Weight

The weights were recorded using an ISI-accredited weighing machine to the nearest 100 grams. The weighing was performed while barefoot and in light attire, with both feet equally distributed, while standing upright in the center of the machine.

Body Mass Index

(Quetelet's index) Calculated as wt (kg)/ht2 (mt2)

Blood Pressure:

After five minutes of rest, seated participants' right arm blood pressure was measured using sphygmomanometers. To the closest 2 mmHg, the 1st and 5th Korotkoff sounds were utilized for recording. The first Korotkoff sound's arrival was used to calculate SBP. The 5th Korotkoff Sound served as the basis for calculating DBP. The mean of the previous two blood pressure readings was used for analysis.

Following a 12-hour fast the previous night, five milliliters of blood were drawn from the antecubital vein using disposable needles and vacutainers after all safety procedures were followed. The samples were collected and then left undisturbed for 30 minutes to allow the clots to fully develop. The serum was isolated from the clot after centrifugation. After centrifugation, the serum was kept in Eppendorf tubes at -200 C until the analysis and then CRP was measured by CRP HS ELISA (enzyme immunoassay for quantitative determination of CRP in human serum). In this method, the normal range of hs-CRP was 0.068-8.2 mg/L

Statistical Analysis

Data was analysed using SPSS 23. In continuous measurements, Mean ± SD (Min - Max) was used and the data was compared among the t test. A p-value of 0.05 was considered to be significant.

Results

A total of 100 patients were selected in this study. Fifty participants with hypertension and 50 patients with normal subjects. The 50 hypertensive patients in the case group were comprised of 30% males and 70% females at a mean age of 57.3 years. The control group comprised 28% males and 72% females at a mean age of 56.2 years. The body mass index in the case group was 23.9kg/m^2 versus 23.5kg/m^2 in the control group. In the case group, the mean systolic and diastolic blood pressures were 148.6 mm Hg and 91.6 mm Hg, respectively. These means in the control group were 106 and 72 mm Hg, respectively (Table 1).

Table 1: Physiological parameters in Hypertensive and control groups

	Hypertension (N=50)	Control (N=50)	p value
Age (yrs)	57.3±4.84	56.2±4.92	0.114
BMI (kg/m^2)	23.9±3.64	23.5±3.12	0.03*
SBP(mm Hg)	148.6±7.54	91.6±7.23	0.002*
DBP(mm Hg)	106±6.45	72±6.12	0.01*

*significant

The mean levels of hs-CRP in the case and control groups were 5.43 $mg/\ l$ and 2.32 $mg/\ l$, respectively., there was a significant analytical difference between the two groups (Table 2) (p value <0.001).

Table 2: Serum hs-CRP in Hypertensive and control groups

	Hypertension	control	p value
Hs-CRP (mg/L)	8.26± 2.76	3.28± 1.42	0.002*

*significant

Discussion

Serum hs-CRP levels were considerably higher in hypertensives compared to controls. CRP raises blood pressure through many ways. CRP decreases the production of nitric oxide by endothelial cells, which promotes vasoconstriction, leukocyte adhesion, platelet activation, oxidation, and thrombosis [12]. High CRP levels stimulate endothelin-1 expression [13], as well as endothelial cell production of plasminogen activator inhibitor-1, promoting vasoconstriction, platelet activation, and thrombosis [14]. CRP has been demonstrated to upregulate angiotensin receptors, which enhances the angiotensin-II-induced rise in blood pressure [15]. Angiotensin-II causes vascular inflammation by increasing oxidative stress and up-regulating pro-inflammatory transcription factors, including NF-κB. These, in turn, influence the production of inflammatory mediators, which contribute to endothelial dysfunction and vascular damage. Patients with hypertension have elevated inflammatory markers (e.g., C-reactive protein, chemokines, and adhesion molecules), which predict the development of cardiovascular disease.

Ki Chul Sung et al. observed hs-CRP to be an independent risk factor for developing hypertension in the Korean population [16]. In 2001, Bautista et al. conducted the first cross-sectional investigation to quantify CRP in hypertension and discovered CRP to be an independent risk factor for the development of hypertension [17]. Bautista et al. (2003) found no connection between hs-CRP and hypertension. They attributed this to their study's tiny sample size [18].

Sesso et al. found a link between higher CRP levels and an increased risk of developing hypertension. Sesso et al. suggested that greater hs-CRP levels were associated with an increased risk of developing hypertension [19]. In the Strong Heart Study (2006), an aberrant lipid profile (reduction in HDL cholesterol from baseline) was observed to predict the development of hypertension in the American Indian community over an 8-year period [13].

In the CARDIA trial of 5115 black and white young adults, the development of incident hypertension was linked with starting systolic blood pressure, triglyceride, and HDL-cholesterol levels over a decade [11].In a 2009 study (the Strong Heart Study data), Marco et al observed that pre-hypertensives who developed hypertension had greater levels of inflammatory markers, higher triglycerides, and lower HDL cholesterol [20].

Conclusion

Our study found that hypertension patients had higher hs-CRP levels, indicating the role of inflammation in the disease pathophysiology.

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