Study The Level Of Hs-Crp With Risk Factors Like Smoking, Hypertension And Diabetes Mellitus

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Abstract: Background: With the turn of the century, CVDs have become the leading cause of mortality in India. Objectives: To correlate the level of Hs-crp with risk factors smoking, hypertension and diabetes mellitus. Material & Methods: This cross sectional study was conducted on patients admitted in the Intensive cardiac Care Unit of Karnataka Institute of Medical Sciences, Hubli between November 2015 – January 2017. A total number of 110 patients were recruited for the study. Results: Out of the total 110 cases 78(70.9%) were male patients and 32(29.1%) female patients. Most of patients were in age group of 51-60 yrs (40%). In the study 18.2% had HTN, 22.7% had DM, 84.5% were smoking and consuming tobacco, 80% were consuming tobacco and 35.5% were alcoholics. In the study there was no significant association between hsCRP and Age, HTN, DM, Smoking, Alcohol and BMI. Hence the above mentioned factors were not affecting HSCRP. Conclusion: HSCRP can be included as one of the risk factor for CVD assessment and it can be used as screening in high risk individual for CVD assessment, which helps in primary prevention. [Rathod V Natl J Integr Res Med, 2020; 11(4):05-09]

Key Words: Hs-CRP, Smoking, Hypertension, Diabetes Mellitus, CVD

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Introduction: Ischemic heart disease (IHD) and stroke constitute the majority of CVD mortality in India (83%), with IHD being predominant[1]. The years of life lost attributable to CVD in India increased by 59% from 1990 to 2010 (23.2 million to 37 million)[1].The prevalence of IHD in 1960 in urban India was 2%, and increased 7-fold to ≈14% by 2013[2,3,4]. Similarly, it more than quadrupled in rural areas, from 1.7% to 7.4% between 1970 and 2013[4,5]. The Macroeconomic Commission for Health estimated that the absolute number of IHD patients in India will increase from 36 million in 2005 to 62 million in 2015 (a ≈70% increase)[6].

Conventional risk factors in the Framingham risk score (FRS), such as age, male sex, hypercho-lesterolemia, hypertension, and smoking, account for most of the risk of CHD and have been the bedrock of risk assessment for decades, However, approximately one-third of individuals with 0 or 1 risk factor develop CHD[7,8] and up to 40% of individuals with cholesterol levels below the population average die from CHD[9].

We know atherosclerosis is most important cause of CHD, and Inflammation is the key mechanism in the pathogenesis of the different stages of atherosclerosis, from onset, progression of atheroma, plaque instability and rupture and restenosis following angioplasty[10,11,12].

Inflammatory biomarkers provide useful information on the inflammatory process of atherosclerosis; they act as a window into the process of cell activation, recruitment of inflammatory cells and proliferation[13]. In recent decades, many studies have shown that CPR is associated with cardiovascular risk. This molecule has characteristics that make it a particularly attractive subject of study:

As a positive acute phase protein it is a marker of systemic inflammation that increases in response to various types of injury, particularly bacterial infections that function as inflammatory stimuli[14].

Its production in the liver is induced mainly by interleukin- 6 (IL-6) and, unlike other acute phase markers, its levels are relatively stable, with no significant diurnal variation, and can thus be accurately measured[15].

During the 1990s high-sensitivity techniques were developed to detect lower serum CRP levels than by previous laboratory methods (down to 0.3 mg/l), known as high-sensitivity CRP (hs-CRP), and these techniques should be used when assessing the cardiovascular risk associated withthe chronic vascular inflammation of atherosclerosis.

There is growing evidence that CRP is not merely a marker of inflammation, but also plays an active role in atherogenesis[16,17].

This study was conducted with the objective: To correlate the level of Hs-crp with risk factors smoking hypertension and diabetes mellitus.

Material & Methods: This cross sectional study was conducted on patients admitted in the Intensive cardiac Care Unit of Karnataka Institute of Medical Sciences, Hubli between November 2015 – January 2017. A total number of 110 patients were recruited for the study. Approval for the study was obtained from the KIMS, Hubli Ethics Committee. Informed consent was taken prior to inclusion in the study.

Inclusion Criteria: All patients with ACS 1. STEMI 2. NSTEMI 3. Unstable angina

Sample Size: A total of 110 cases of ACS were included in the study during the period extending between December 2015 to January 2017. Sample size is calculated to be 110, considering the prevalence of ACS as 7.5% with confident interval of 95% and precession of 5%.

Exclusion Criteria: Patient with past CABG, Patient with PTCA, Patient with valvular heart disease. Patient with hepatic dysfunction, Patient with renal dysfunction creatinine > 1.5 mg / dL, Patient with collagen vascular disease, All patients with recent or ongoing infection, fever or inflammatory disorder & Recent trauma.

Method Of Collection Of Data: Patients presen-with h/o chest pain, shortness of breath, palpitation were taking thorough clinical history for previous h /o DM, HTN, IHD & measurement of blood pressure, & BMI , ECG and 2Decho was done, patients with clinical features ,ECG & 2Decho features suggestive of STEMI,NST-ACS, were selected for blood investigation which included Lipid profile, hsCRP, urea ,creatinine ,complete blood count and LFT,RA factor,and coronary angiography. patients with raised total count and abnormal LFT & Positive RA factor a features suggestive of inflammation were excluded from study . Informed written concent was taken from every patient.

Statistical Analysis: Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Continuous data was represented as mean and standard deviation. Independent t test or was used as test of significance to identify the mean difference between two quantitative variables. ANOVA (Analysis of Variance) was the test of significance to identify the mean difference between more than two groups for quantitative data. Pearson correlation or Spearman's correlation was done to find the correlation between two quantitative variables and qualitative variables respectively.

Results: 110 patients presenting with ACS who satisfied the inclusion criteria were enrolled in the study. The data both clinical as well as laboratory values were collected and then analysed accordingly. Out of the total 110 cases 78(70.9%) were male patients and 32(29.1%) female patients. 25(22.7%) patients were diabetic and 20(18.2%) patients were hypertensive on medication, 93 (84.5%) were smokers. By means of ECG out of 110 cases 81(73.6%) patients diagnosed as STEMI, 29(26.4%) were NST-ACS. On coronary angiography 56(52.8%) has SVD, 12(11.3%) has DVD, 20(18.9%) has TVD & 18(17.0%) has normal coronaries.

In the study majority of subjects were in the age group 51 to 60 years (40%), 20% were in the age group 41 to 50 years, 19.1% were in the age group 61 to 70 years, 16.4% were in the age group <40 years and 4.5% were in the age group >70 years. In the study 29.1% were females and 70.9% were males. Table 1

Table 1: Age Distribution Of Subjects In The Study

		Count	%
Age	<40 Years	18	16.4%
	41 to 50 Years	22	20.0%
	51 to 60 Years	44	40.0%
	61 to 70 Years	21	19.1%
	>70 Years	5	4.5%

In the study 18.2% had HTN, 22.7% had DM, 84.5% were smoking and consuming tobacco, 80% were consuming tobacco and 35.5% were alcoholics. Table 2

Table 2: Risk Factors Among Subjects

	Yes		No	
	Count	%	Count	%
HTN	20	18.2%	90	81.8%
DM	25	22.7%	85	77.3%
Smoking and Tobacco	93	84.5%	17	15.5%
Alcohol	39	35.5%	71	64.5%

In the study 18.2% had HTN, 22.7% had DM, 84.5% were smoking and consuming tobacco, 80% were consuming tobacco and 35.5% were alcoholics.

In the study there was no significant association between hsCRP and Age, HTN, DM, Smoking, Alcohol and BMI. Hence the above mentioned factors were not affecting HSCRP. Table 3

Table 3: Association Between Hscrp And Age, HTN, DM, Smoking, Alcohol And BMI

		hsCRP						P value
		<1 mg%		1 to 3 mg%		>3 mg%		
		Count	%	Count	%	Count	%	
Age	<40 Years	8	16.7%	9	23.1%	1	4.3%	0.372
	41 to 50 Years	13	27.1%	6	15.4%	3	13.0%	
	51 to 60 Years	16	33.3%	16	41.0%	12	52.2%	
	61 to 70 Years	10	20.8%	6	15.4%	5	21.7%	
	>70 Years	1	2.1%	2	5.1%	2	8.7%	
HTN	Yes	10	20.8%	7	17.9%	3	13.0%	0.727
	No	38	79.2%	32	82.1%	20	87.0%	
DM	Yes	11	22.9%	10	25.6%	4	17.4%	0.755
	No	37	77.1%	29	74.4%	19	82.6%	
Smoking	Yes	39	81.2%	35	89.7%	19	82.6%	0.530
	No	9	18.8%	4	10.3%	4	17.4%	
Alcohol	Yes	18	37.5%	12	30.8%	9	39.1%	0.742
	No	30	62.5%	27	69.2%	14	60.9%	

In the study there was no significant difference in mean hsCRP levels with respect to risk factors HTN, DM, Smoking, Tobacco and Alcohol. Table 4

Table 4: Comparison Of Hscrp With Respect To Risk Factors

		hsCRP			P value
		Mean	SD	Median	
HTN	No	2.2	2.5	1.1	0.823
	Yes	2.3	2.4	1.2	
DM	No	2.2	2.5	1.2	0.745
	Yes	2.4	2.4	1.2	
Smoking	No	2.3	2.4	1.2	0.887
	Yes	2.2	2.9	.8	
Alcohol	No	2.3	2.2	1.2	0.908
	Yes	2.3	2.6	1.2	

Independent t test

Discussion: Several modifiable and non-modifiable factors such as HT, DM, smoking etc are recognized as major risk factors for CVD and aggressive correction of these play vital role in CVD prevention, because not all adverse CV events can be predicted or explained by these conventional risk factors, which limits our ability to accurately identify the individuals who are at "high risk" of developing CVD.

hsCRP And Age: Syed tanveer etal[18] study found that patients with STEMI detected higher hs-CRP values (≥0.5 mg/dl) in older patients than young, study by Guruprasad etal[19] had higher hsCRP in old age (1.8 ± 24mg/l) when compared to young patient of (1.5 ± 1.1mg/l) ,Ramon Arroyo-Espliguero etal[20] study also support this where they concluded that hsCRP concentrations raise with increasing age.

In our study hsCRP increased with age, where mean hscrp was highest among those aged more than 70 yrs (mean hscrp 3.9) and lowest aged less than 40 yrs (mean hscrp 1.5) which is concordant with other studies.

hsCRP And Sex: Studies by Sharad Gupta etal[21], McConnell JP etal[22], have shown that the levels of hs-CRP are usually higher in women than men. In our study, men (mean hsCRP 2.4± 2.5mg/l) were found to have higher hsCRP levels as compared to women (mean hscrp 2.3 ± 2.4mg/l) which is not in concordant with other studies.

hsCRP And HTN: Study done by Bautista LE etal[23],i,e Independent association between inflammatory markers (C-reactive protein, interleukin-6, and TNF-alfa) and essential hypertension and concluded TNF-alfa and IL-6 could be independent risk factors for HTN in apparently healthy subjects and CRP was not significantly associated with hypertension. Study done by sharad gupta etal[21] also shows no significiant correlation with hscrp and hypertension p-value 0.035. In our study we did not find significant association between elevated hsCRP levels and HTN p-value 0.727, which in concordant with other studies.

hsCRP And Diabetes Mellitus: Study conducted bt sharad etal[21],p-value 0.08 (showed no stastical significance between hsCRP and Diabetes mellitus. In our study there was no stastical significance between non diabetic (mean hscrp 2.48mg/l) and diabetes (2.4mg/l)with p-value 0.745 which is in concordance with other studies.

hsCRP And Smoking: Studies by Yanbaeva etal[24], sharad gupta etal[21] and MA Mendall[25] which showed significant correlation between hscrp and smoking. In our study, there was no significiant correlation of hsCRP levels and smoking which is not in concordance with other studies.

Conclusion: It was concluded that hsCRP can be included as one of the risk factor for CVD assessment and it can be used as screening in high risk individual for CVD assement,which helps in primary prevention.

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