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Original Research Article

High Sensitivity C- Reactive Protein in the Male Patients with Angina with Normal Coronary Arteries

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ABSTRACT

Aims: Endothelial dysfunction has been reported in patients with Cardiac Syndrome X but little is known regarding low grade chronic inflammation as a pathogenic mechanism. We assessed whether markers of inflammation in the form of Hs CRP differ in CSX patients as compared to control subjects. As most of studies have taken women predominated groups, we sought to undertake our study among male patients.
Methods and Results: The mean age was 50.36 (±9.77) among study group and 51.64 (±7.48) among control group. 25 had sedentary life style, 26 (53%) were urban dwellers, 33(67.3%) were hypertensive's, 11(22.4%) were diabetics/IFG/IGTT, 37(75.5%) were smokers, 18 were dyslipidemics, 7 had family history of CAD and 17 were obese. All the patients underwent baseline investigations like- Haemogram, Chest X-ray, ECG, Serum chemistry, lipid profile, Echocardiography, and an exercise electrocardiography (TMT). Patients with any systemic inflammatory conditions, baseline ST-T wave changes, epicardial coronary spasm, hypertrophic or dilated Cardiomyopathy, acute MI and inadequate exercise test were excluded from the study. Coronary angiography was performed in all the patients with positive TMT and negative hyperventilation test. In patients having normal coronary arteries, serum samples were taken and stored frozen at -70^{o}C until further analysis. The level of HSCRP in the serum samples was estimated by a high sensitivity (with lowest detectable level =0.11mg/L) immunoturbidometric assay on Hitachi 912 clinical chemistry auto-analyzer. HSCRP levels were significantly higher in patients with CSX compared to controls (3.91±2.74mg/L VS 1.87±1.86mg/L, P =<0.001)
Conclusions: The study showed that HSCRP levels are higher in CSX patients as compared to control patients. The significant differences were maintained after adjustment for traditional risk factors. This study further confirmed the previous reports that HSCRP levels correspond to the duration of chest pain episodes and the frequency of these episodes. Though the cause and effect relationship of this abnormality deserves further investigation. This study also showed that the HSCRP levels are higher among smokers, HTNsives, diabetics, dyslipidemics, and obese as compared to subjects without these risk factors in both the groups.

Key words: Cardiac Syndrome X, Chest Pain and Normal CAG Microvascular angina, microvascular dysfunction and CRP, HSCRP and CSX, Chronic inflammation and CSX, CSX and risk factors, CSX in men, HSCRP and CPNCA.

INTRODUCTION

Patients with cardiac syndrome-X are characterized by the typical symptom of exertion/stress induced chest pain that is associated with ischaemic-like ST-segment changes or presence of reversible perfusion defects on stress testing despite a normal coronary arteriogram. This syndrome (to be distinguished from metabolic syndrome–X which is characterized by abdominal obesity, hypertriglyceridemia, low HDL cholesterol, insulin resistance, hyperinsulinemia, and hypertension), is an important clinical entity that should be differentiated from classic ischemic heart disease caused by Coronary Artery Disease

Systemic inflammation is associated with endothelial dysfunction, and inflammatory markers such as *high sensitive C-reactive protein* (HSCRP) have been previously shown to be powerful prognostic markers in a variety of population groups. Evidence suggests that a significantly higher level of CRP is found in patients with cardiac syndrome-X when compared to a normal control and this low grade inflammatory process is unrelated to an increased infectious pathogen burden (Lanza *et al*., 2004). Thus, systemic inflammation may play an important role in the pathogenesis of cardiac syndrome-X through endothelial dysfunction. There is limited data on the efficacy of anti-inflammatory drug such as non-steroid anti-inflammatory drug or steroid in patients with cardiac syndrome-X. However, statin and angiotensin converting enzyme (ACE) inhibitor do have a role in treating these patients. Their efficacy on treating these patients may be due to their anti-inflammatory effects.

Both very low (<0.5mg/L) and very high (>10mg/L) levels of HSCRP provide important prognostic information on cardiovascular risk. The recent standardization of HSCRP assay allows acceptable precision down to and below 0.3 mg/L and it is in their lower previously normal ranges that HSCRP seems to have predictive abilities for cardiac disease events.

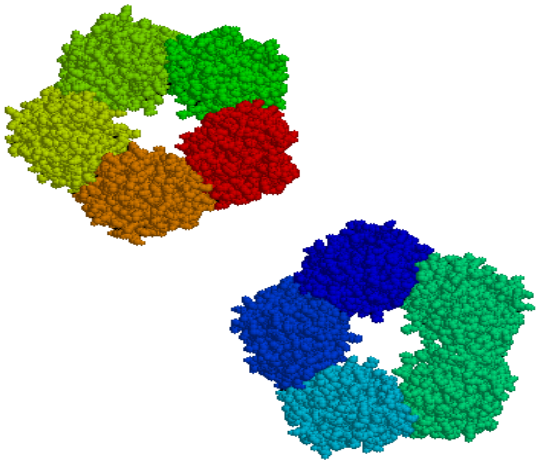

Fig 1: Pentameric structure of CRP.

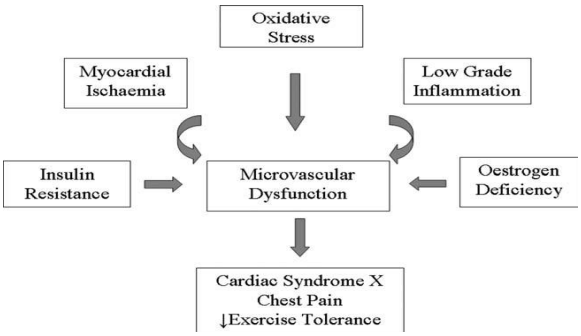

Fig 2: Scheme of the pathogenetic mechanisms that can lead to microvascular dysfunction in cardiac syndrome X patients with chest pain and reduced exercise tolerance (Tiong *et al*., 2009).

MATERIALS AND METHODS

All the patients who presented with chest pain typical enough to suggest of angina clinically and angiography revealing normal coronaries has been included in the

study. All the patients underwent the following baseline investigations:
Haemogram, Chest X-ray, ECG, Serum chemistry, lipid profile, Echocardiography, Exercise electrocardiography (Treadmill Test):

Positive TMT: Positive exercise test is defined as *The development of 0.10 mV (1 mm) or greater of J-point depression measured from the PQ-Junction, with a relatively flat ST segment slope (e.g., <0.7 to 1mV/sec), depressed 0.10 mV or more 80 msec after the J-point (ST 80) in three consecutive beats with a stable baseline is considered to be an abnormal response.*

Coronary angiography was performed in all the patients with positive TMT and negative hyperventilation test. Coronary angiography was performed in our Hospital's Cardiac Catheterization Lab., via femoral route using non-ionic contrast.

In patients having normal coronary arteries, peripheral blood sample was taken for HSCRP, stored to be analyzed at later time.

Specimen Collection:- Any time during day, about 2 ml of blood (0.5 ml serum), were taken, fasting was not required as there are no diurnal variations for HSCRP. Due to inter individual variation, two specimens were collected ideally two weeks apart.

Sera was separated from the blood of patient and control subjects by centrifugation at 3000 rpm, and stored frozen at -70^{o}C until further analysis. The level of HSCRP in the serum samples was estimated by a high sensitivity (with lowest detectable level =0.18mg/L) immunoturbidometric assay on Hitachi 912 clinical chemistry auto-analyzer, using HSCRP reagent kit from Audit Diagnostic Cork, Ireland. The CRP test is based upon the reaction between C-reactive protein in the sample and latex covalently bound antibodies against human CRP. Values were determined turbidometrically at 570 nm at 37 ^{o}C using fixed time measurement with sample blank correction. Standardization of the method was performed by using different dilution of the stock standard provided with the reagent kit, having a value of 144mg/L of CRP.

The HSCRP test is based upon the reaction between CRP in the sample and latex covalently bound antibodies against human CRP.

Exclusion criteria: All patients having rheumatoid arthritis, any other systemic inflammatory disease, baseline ST-T wave changes, epicardial coronary spasm, hypertrophic or dilated Cardiomyopathy, acute MI and inadequate exercise test were excluded from the study.

Statistical methods: Data has been shown as value ± SD. For comparing the data of two groups, based on ratio scale, a parameter test- independent samples T-test was used. And for comparing three groups, One way Analysis of Variance was used. And for intergroup comparison, a Post-Hoc test-LSD (Least Significant Difference) was used. P value of <0.05 was taken to be statistically significant. Multiple linear regression analysis was performed using natural CRP as a dependent variable and smoking status patterns, age, BMI, HTN, diabetes which were significantly related to CRP level in univariate analysis, as independent variables. All statistical analyses were performed using SPSS software (version 15.0).

OBSERVATIONS

A total of 50 male patients diagnosed as CSX and 50 male controls were taken. One patient of CSX was discarded as his HSCRP levels repeatedly came more than 10mg/L.

Table 1: Baseline Clinical Characteristics are:

S. No	Parameter		Study Group	Control Group
1	Number of subjects		49	50
2	Age (years)		50.36 (±9.77)	51.64 (±7.48)
3	Life style (Exersion)	Sedentary	25	18
		Moderate	24	27
		Heavy	0	5
4	Urban /Rural		26/23	30/20
5	HTN		33 (67.3%)	20 (40.0%)
6	Diabetes (with IFG)		11 (22.4%)	8 (16.0%)
7	Smoking	Total	37 (75.5%)	29 (58.0%)
		Current	25(51.0%)	18(36.0%)
		Former	11(22.4%)	11(22.0%)
		Never	1326.5%	21(42.0%)
8	Dyslipidemia	Number (n)	18 (36.7%)	13 (26.0%)
		Mean TG mg/dl (SD)	186.42 (±109)	164.48 (±76)
		Mean Cholesterol mg/dl (SD)	168.79 (±52)	152.50 (±42)
9	Obesity	Over weight	15	15
		Grade-I	2	3
10	BMI (mean in cms)		24.10 (±2.9)	23.74 (±3.36)
11	Waist (mean in cms)		85.36 (±6.5)	81.23 (±14.34)
12	Family H/O CAD		7	6
13	Alcohol History		4	0

Values are mean ± SD, numbers, or numbers with percentages.

Table 2: HS-CRP in CSX patients as compared to Normal controls.

	Group	N	Mean	Std. Deviation	Std. Error Mean	P-value
HSCRP	Study	49	3.9100	2.74432	0.39205	0.0001
	Control	50	1.8774	1.86451	0.26368	

HSCRP was significantly higher in study as compared to control subjects (p=0.0001)

Table 3: Chest pain duration and HSCRP.

Pain Duration Groups	No.	HSCRP(mg/L)		P-value
		Mean	Std. Deviation	
<5min	17	1.5053	1.14994	0.0001
5-10min	19	4.8526	2.43964	
>10min	12	5.9058	2.50932	
Variable	1	2.9300	.	
Total	49	3.9100	2.74432	

Values were significant at less than 0.0001.

HSCRP levels were significantly higher as the duration of pain episodes increased (p=0.0001).

Table 4: Chest Pain Episodes Per Week And HS CRP.

Episode / week	HSCRP(mg/L)						
	N	Mean	Std. Deviation	95% Confidence Interval for Mean		Min.	Max.
				Lower bound	Upper bound		
1-3/wk	30	2.8383	2.33958	1.9647	3.7119	.24	9.56
4-6/wk	11	5.6073	2.73779	3.7680	7.4465	1.98	9.30
>6/wk	8	5.5950	2.35908	3.6228	7.5672	2.48	8.80
Total	49	3.9100	2.74432	3.1217	4.6983	.24	9.56

P-value 0.002.

Table 5a: Multiple Comparisons (Dependent Variable: HSCRP).

(I) Episodes Per Week	(J) Episodes Per Week	Mean Difference (I-J)	Std. Error	Significance	95% Confidence Interval	
					Lower bound	Upper bound
1-3/wk	4-6/wk	-2.76894(*)	.85814	.002	-4.4963	-1.0416
	>6/wk	-2.75667(*)	.96874	.007	-4.7066	-.8067
4-6/wk	1-3/wk	2.76894(*)	.85814	.002	1.0416	4.4963
	>6/wk	.01227	1.13124	.991	-2.2648	2.2893
>6/wk	1-3/wk	2.75667(*)	.96874	.007	.8067	4.7066
	4-6/wk	-.01227	1.13124	.991	-2.2893	2.2648

(*)The mean difference is significant at the .05 level.

HSCRP levels were significantly higher in subgroups, 4-6/wks and >6/wks as compared to 1-3/wk subgroup (p=0.002 and p=0.007).

HSCRP levels were not significantly different between 4-6/wk and >6/wk subgroups.

Table 5b: Standardized regression coefficient by multiple regression analysis predicting logarithm-transformed CRP: Coefficients(a)

	Standardized Coefficients	Sig.
(Constant)		.206
Group	-.268	.002
BMI	.203	.146
Obesity	-.037	.775
Smoking	.191	.140
SmokePY	.056	.659
HTN	-.241	.177
HTNstage	.097	.556
Dyslipidemia	-.345	.000
DMIFGIGTT	.010	.908

a Dependent Variable: HSCRP.

Table 6: HS-CRP and ST depression in TMT.

	ST DEPRESSION	N	Mean	Std. Deviation	Std. Error Mean	P-value
HSCRP	<2	40	3.8470	2.68251	.42414	0.586
	≥2	9	4.1900	3.16225	1.05408	

P-value was insignificant between subgroups based on ST-depression on TMT.

Table 7:Metabolic Syndrome-X and HS-CRP among study group.

Metabolic Syndrome-X	HSCRP(mg/L)				P-value
	N	Mean	Std. Deviation	Std. Error Mean	
Present	11	6.3945	2.86733	.86453	0.185
Absent	38	3.1908	2.27636	.36928	

P-value was not found to be significant.

Table 8: Comparison of HSCRP among study and control groups regarding hypertension.

Parameter	HSCRP(mg/L)						P-value
	Study			Control			
	Number	Mean	SD	Number	Mean	SD	
Hypertensive	33	4.31	2.84	20	3.22	2.14	0.143
Normotensive	16	3.08	2.42	30	0.98	0.89	0.000
P-value	0.143			0.001			X

HSCRP levels were significantly higher in hypertensives as compared to normotensives in control group. But the same was insignificant in study group.

HSCRP levels were significantly higher in normotensives in study group as compared to normotensives in control group. While as among hypertensives the same relation was found insignificant.

Table 8a: HS-CRP levels with respect to hypertension stage(study).

Parameter		study group	HSCRP(mg/L)	
			HSCRP level (mg/L)	SD
HTN	Over all	33	4.31	2.84
	preHTN	2	5.65	2.42
	Stage 1	17	3.13	2.43
	Stage 2	14	5.54	2.53
Normotensive		16	3.08	2.42

Table 8b: Multiple comparisons between different HTN stages with HSCRP as dependent variable:

(I) HTN stage	(J) HTN stage	Mean Difference (I-J)	Significance
preHTN	Stage 1	2.51	0.198
	Stage 2	0.10	0.956
	normotensive	2.56	0.189
Stage 1	preHTN	-2.51	0.198
	Stage 2	-2.40*	0.013
	normotensive	0.05	0.950
Stage 2	preHTN	-.10	0.956
	Stage 1	2.40*	0.013
	normotensive	2.46*	0.012

*-The mean difference is significant at the 0.05 level.

HSCRP levels were significantly higher in Stage-2 as compared to Normotensive (P-value = 0.012) and stage-1 (p-value = 0.013).

Table 8c: HSCRP with respect to HTN years (in study group).

HTNsive years	No of patients	HSCRP(mg/L)	
		Mean	SD
<1	9	3.78	2.97
1-5	11	4.72	2.49
5-10	9	4.07	3.25
>10	4	4.89	3.33
Normotensives	16	3.08	2.42

Comparing HSCRP between different HTNsive year groups and normotensives, comparison between various HTN year groups amongst HTNsives in study group was found insignificant by ANOVA. Though same comparison among all subjects revealed only comparison between overall HTNsives and normotensives as significant (not shown in the table).

Table 9: HSCRP and Obesity in study group as compared to control group.

		HSCRP(mg/L)						P-Value
		Study Group			Control Group			
		N	Mean	SD	N	Mean	SD	
Obese	Over weight	15	5.45	3.25	15	2.23	1.49	0.002
	Grade 1	2	5.53	1.45	3	4.15	3.05	0.605
	Grade 2	0			0			
Non obese		32	3.08	2.18	32	1.49	1.78	0.002

HSCRP levels were significantly higher in study group among overall obese and non-obese as compared to same subgroups among control group ($p = 0.002$).
HSCRP levels were not significantly different between grade-1 obesity among study and control groups.

Table 10a: HSCRP in study group with respect to Obesity.

Obesity status	No of patients	HSCRP(mg/L)		P-value
		Mean	SD	
Non-obese	32	3.0834	2.18	0.009
Obese	17	5.4659	3.06	

In study group, HSCRP levels were significantly higher in overall obese as compared to non-obese ($p = 0.009$).

Table 10b: HSCRP in all subjects (study as well as controls) with respect to obesity.

Obesity status	No of patients	HSCRP(mg/L)		P-value
		Mean	SD	
Non-obese	64	2.29	2.13	0.011
Obese	30	3.84	2.98	

In all subjects (study as well as controls), HSCRP levels were significantly higher in overall obese as compared to non-obese ($p = 0.011$).

Table 11: HSCRP in relation with DM/IFG/IGTT, between study and control groups.

DM/IFG/IGTT	HSCRP (mg/L)		P-value
	Study group	Control group	
Yes	3.488 (±2.29)	2.796(±1.73)	0.46
No	4.032(±2.87)	1.702(±1.91)	0.0001

Comparing HSCRP in relation with DM/IFG/IGTT, between study and control groups, p-value was insignificant among DM/IFG/IGTT, while as it was significant among non-DM/IFG/IGTT.

Table 12: HSCRP and Alcohol history among study group.

	Alcohol History						P-value
	Yes			No			
	N	Mean	SD	N	Mean	SD	
HSCRP (mg/L)	4	4.03	1.86	45	3.89	2.82	0.061

HSCRP levels were not significantly different between alcohol users and non-users in study group.

Table 13: HSCRP in relation with dyslipidemia.

Dyslipidemia	HSCRP (mg/L)		P-value
	Study group	Control group	
Yes	5.39 (±2.98)	3.61(±2.13)	0.078
No	3.04(±2.21)	1.26(±1.31)	0.0001

HSCRP levels were significantly higher in non-dyslipidemia among study group as compared to those among control group ($p = 0.001$). While as same was insignificant among dyslipidemics.
Comparing dyslipidemic with non-dyslipidemics, HSCRP levels were significantly higher in dyslipidemic than non-dyslipidemics($p<0.05$).

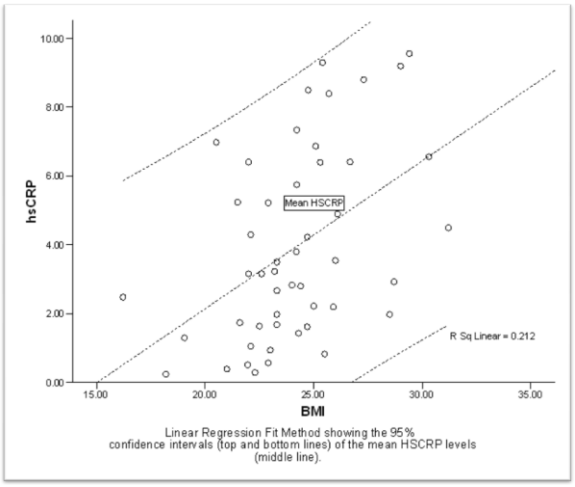

Figure 3: HSCRP and BMI depicted as a scatter plot._

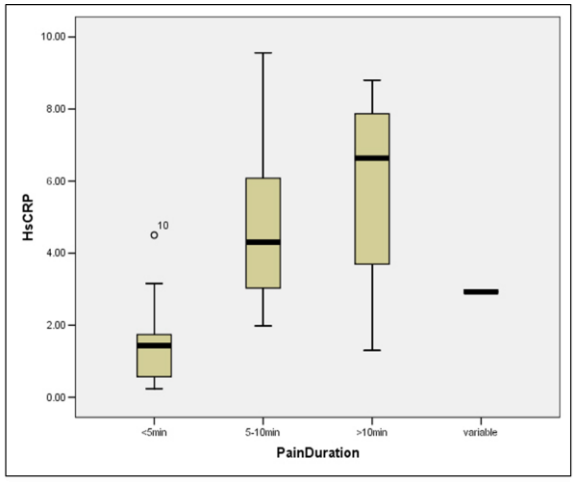

Figure 4. HSCRP and chest pain duration depicted in a box plot diagram.

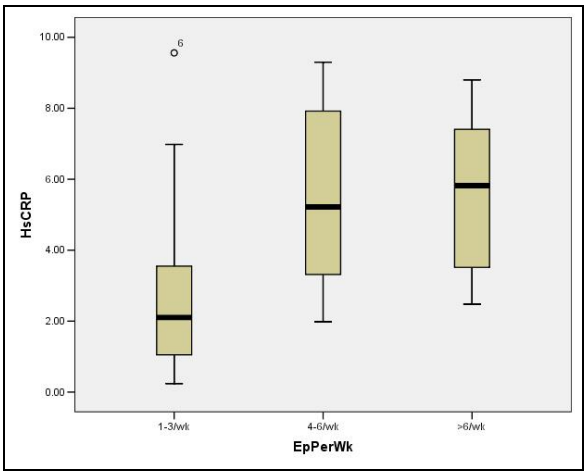

Fig 5: HSCRP and episodes of chest pain per week, depicted in an box plot diagram.

DISCUSSION

This study was carried out in Department of Cardiology, of our institute in coordination with Department of Biochemistry. Our study included 49 male patients diagnosed as CSX and 50 control male subjects. The mean age was 50.36 (±9.77) among study group and 51.64 (±7.48) among control group. 25 had sedentary life style, 26 (53%) were urban dwellers, 33(67.3%) were hypertensives, 11(22.4%) were diabetics/IFG/IGTT, 37(75.5%) were smokers, 18 were dyslipidemics, 7 had family history of CAD and 17 were obese. Details are given in table-1.

HSCRP levels were significantly higher in CSX patients as compared to control subjects (3.9100±2.74mg/L and 1.8774±1.86mg/L; p-value <0.0001 as depicted in table-2). Table 5b shows the results of multiple linear regression analysis using CRP as a dependent variable and using smoking status, HTN, diabetes, dyslipidemia, obesity as independent variables. The significant differences were maintained after adjustment for traditional risk factors.

Mechanisms involved in the microvascular abnormalities in patients with Cardiac syndrome- X are likely to be multiple and heterogeneous and may result in variable combination of impaired vasodilator function and increased vasoconstrictor activity, vasodilator abnormalities variably involving endothelium dependent mechanisms (i.e. in response to pacing & acetylcholine); and endothelium independent mechanisms (i.e. in response to dipyridamole & papaverine). True myocardial ischemia, reflected in the production of lactate by the myocardium during exercise or pacing, is present in some of these patients. Intravascular ultrasonographic studies (IVUS) have

demonstrated anatomical and physiological heterogeneity of syndrome-X, a spectrum ranging from normal coronary arteries to vessels with intimal thickening atheromatous plaque but without critical obstructions.

It has been suggested that in syndrome-X, angina is caused by myocardial ischemia determined by a dysfunction of small coronary vessels (<500 micrometers) not visible at coronary angiography, a condition defined as "microvascular angina" (Cannon *et al*., 1988).

Our study demonstrated that patients with prolonged anginal episodes (>10 minutes) had significantly higher HSCRP levels (5.90±2.50mg/L) than patients with moderate (5-10minutes) duration of pain (4.85±2.43mg/L) and than those with shorter duration of pain (1.50±1.14mg/L); p<0.0001. This is depicted in table-3 and figure 4.

Similarly patients with greater number of chest pain episodes per week (>6/wk) had significantly higher level of HSCRP (5.59±2.35mg/L) than patients with fewer number (1-3/week) of episodes (2.83±2.33mg/L);p=0.007.Similarly patients with >6 episodes/week had significantly higher level of HSCRP (5.60±2.73mg/L) than patients with fewer number (1-3/week) of episodes (2.83±2.33mg/L) ;p=0.002. But HSCRP levels were not significantly different between 4-6/wk and >6/wk subgroups. These results are depicted in table-5.

Comparison of HSCRP among study and control groups regarding hypertension revealed that HSCRP levels were significantly higher in hypertensives (3.22±2.14mg/L) as compared to normotensives (0.98±0.89mg/L) in control group (p=0.001). But the same was insignificant in study group (table 8). HSCRP levels were significantly higher in normotensives in study group as compared to normotensives in control group (p=0.0001). While as among hypertensives the same relation was found insignificant (table-8). Group comparisons among various HTNsive stages and normotensives revealed a significantly higher HSCRP levels in higher stages as compared to lower stages and normotensives (p<0.03). HSCRP levels were significantly higher in preHTN as compared to Normotensive (P-value = 0.0001) (table 2). Comparing HSCRP between different HTNsive year groups and normotensives, only comparison between overall HTNsives and normotensives was found significant (p<0.01); and comparison between various HTN year groups amongst HTNsives was insignificant.

Comparing overall smokers of two groups (study and control), HSCRP was significantly higher in study group (4.2033±2.94 mg/L) as compared to control group(3.09±1.96mg/L) (table 9). Comparing current smokers of two groups, HSCRP was significantly (p=0.003) higher in study group as compared to control group. Comparing overall smokers and never smokers in study group, P-value was insignificant at p-value = 0.141, while as comparing overall smokers and never smokers in control group, P-value was significant at p-value = 0.023. With regards to smoking exposure, HSCRP levels were significantly higher in subjects with exposure of >20 years as compared to never smokers (p=0.011); higher in subjects with exposure of 10-20 years as compared to those with 1-5 years exposure(p=0.039) and never smokers (p=0.001). These findings were consistent to most of the findings which Ohsawa *et al.* (2005)

HSCRP and Obesity in study group as compared to control group revealed that HSCRP levels were significantly higher in study group among overall obese and non-obese as compared to same subgroups

among control group (p = 0.002). In study group, HSCRP levels were significantly higher in overall obese as compared to non-obese (p = 0.009). In all subjects (study as well as controls), HSCRP levels were significantly higher in overall obese (3.84±2.98mg/L) as compared to non-obese (2.29±2.13mg/L) (p = 0.011). Comparing HSCRP with BMI, HSCRP shows a linear trend (Figure-3).

These findings confirmed the previous reports like that of Yudkin *et al.* (1999), who found that CRP levels were >1.35µg/ml in BMI group with BMI of 27.56±4.5 while as they were <1. 35 µg/ml in those with BMI of 24.36±4.0.

In relation with diabetes/IFG/IGTT, HSCRP levels were significantly higher in subjects with diabetic/IFG/IGTT (2.796(±1.73)) in control group as compared to subjects without diabetic/IFG/IGTT (1.702(±1.91)) the same group (p < 0.05). Comparing HSCRP in relation with DM/IFG/IGTT, between study and control groups, p-value was insignificant among DM/IFG/IGTT, while as it was significant among non-DM/IFG/IGTT (4.032(±2.87) and 1.702 (±1.91); p=0.0001). This has been depicted in table 11.

HSCRP in relation with dyslipidemia revealed that HSCRP levels were significantly higher in non-dyslipidemia among study group as compared to those among control group (p = 0.001). While as same was insignificant among dyslipidemics. Comparing dyslipidemics with non-dyslipidemics, HSCRP levels were significantly higher in dyslipidemic than non-dyslipidemics(p<0.05).

HSCRP levels were not significantly different between alcohol users and non-users in study group (table 12). Mendall *et al*. (2000) also recorded that HSCRP levels did not differ significantly between alcohol users and others. Doggen *et al*. (2000) found that Alcohol use was associated with C-reactive protein levels in patients, with never users having higher levels compared with regular users. However, no difference in levels was found amongst control subjects.

CONCLUSION

The study showed that HSCRP levels are higher in CSX patients as compared to control patients. The significant differences were maintained after adjustment for traditional risk factors. This study further confirmed the previous reports that HSCRP levels correspond to the duration of chest pain episodes and the frequency of these episodes. Though the cause and effect relationship of this abnormality deserves further investigation. This study also showed that the HSCRP levels are higher among smokers, HTNsives, diabetics, dyslipidemics, and obese as compared to subjects without these risk factors in both the groups.

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ABBREVIATIONS

AMI : Acute myocardial infarction.
ANOVA :Analysis of variance.
BMI :Body mass index.
CAD :Coronary artery disease.
CRP : C-reactive protein
CSX :Cardiac syndrome-X.
CPNCA :Chest Pain and Normal Coronary Artery.
DM :Diabetes mellitus.
ECG :Electrocardiogram.
ELISA :Immunosorbent Assay.
HTN :Hypertension.
HSCRP :High sensitivity C-reactive protein.
HDL :High density Lipoprotein.
IFG :Impaired Fasting Glucose.
IGTT :Impaired Glucose tolerance test.
IL-6 :Interleukin-6.
IVUS :Intravascular Ultrasonography.
kD :kilo Dalton
L :Litre
mg :milligram
MIBG :Metaiodobenzylguanine.
mmHg :millimeters of mercury
TMT :Treadmill Test.
TG :Triglyceride

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