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Evaluation of the Acute Phase Reactants CRP in Unstable Angina Cases

Abstract

Unstable angina, a syndrome of symptoms caused by ischemia of the heart muscles, is both frightening and disabling and may herald acute myocardial infarction. The immediate precipitating events of the atherosclerotic plaque responsible for that critical degree of ischemia resulting in the syndrome of unstable angina are progression of atherosclerosis platelet aggregation, thrombosis and vasospasm. Acute phase reactants are proteins in the plasma whose levels increase during acute inflammatory states or secondary to certain types of tissue damage. A cross-sectional descriptive study was done in Safdarjung Hospital on 40 subjects and controls to establish the acute phase reactants CRP in unstable angina cases and their relationships. In our study, we found that CRP levels are increased in cases as compared to controls, thus showing a direct correlation.

Keywords: Acute phase reactants, CRP, Unstable angina.

Introduction

Unstable angina lies in the middle of the spectrum of coronary artery disease with silent ischemia at one end, and acute transmural infarction at the other end. It nearly always occurs in the setting of atherosclerotic disease. Unstable angina frequently progresses to myocardial infarction. The acute phase response is a non-specific early response that begins when injury or invading pathogens stimulate the release of cytokines and other factors. In response to injury, local inflammatory cells secrete a number of cytokines into the bloodstream. This mobilizes the body's immune reaction (including the liver responding) by producing a large number of acute-phase reactants. Each of these acute phase reactants plays an important role in the body's reaction to infection. The acute-phase response is triggered by the cytokines interleukin 6 (IL-6), IL-1 and tissue necrosis factor released by macrophages and other cells at the injury site. These factors induce fever and stimulate the liver to produce CRP and other acute-phase proteins. The acute-phase response mobilizes other immune and inflammatory responses as immune cells are recruited to the area and blood supply to site increases. This study is an attempt to probe into the pathogenesis of unstable angina by testing the hypothesis that a relationship exists between acute-phase reactants like CRP and unstable angina.

Method

A cross-sectional descriptive study on eighty subjects was done in Safdarjung Hospital, New Delhi, which included forty cases and forty controls. Detailed history, general physical examination and thorough systemic examination of every volunteer were done.

Inclusion

Patients with new onset angina (less than one month, worsening of previously stable angina, increase in severity or frequency, or decrease in effect tolerance), and angina at rest were included. Any of these criteria singly or in combination had to be satisfied along with both of the following:

1. Presence of ischemic ST and/ or T changes in ECG (ST segment depression-1 mm in one or more lead, with ST remaining horizontal or downsloping for more than 0.08 sec; symmetrical T inversion more than 5 mm).
2. Absence of baseline evidences of myocardial infarction. (new Q wave or persistent ST elevation in ECG, rise in levels of cardiac enzymes-SGOT, CPK, LDH).

Exclusion

Subjects having conditions known to be associated with raised acute phase proteins were a basis of exclusion, namely, liver disease, malignancy, major injury surgery within last one month, any acute infective condition, rheumatic fever within the last one month, collagen vascular diseases, osteoarthritis and myocardial infarction within the last one month. Also control group included age and sex-matched normal people. Subjects in this group had to satisfy the criteria: No episode of

any acute infection, major injury, surgery in last one month; no evidence of ischaemic heart disease as evident from history examination and resting ECG; not suffering from any chronic illness and pregnant women.

Limitations

Due to ethical, logistic and socio-economic constraints, coronary angiography could not be performed in all the subjects, hence the diagnosis of the unstable angina was only clinical, and the control group which was considered healthy may have had subjects with abnormal coronary vessels though no findings on history, examination, ECG and baseline investigations.

Table 1 shows that cases and controls are similar in age distribution. There is no statistically significant difference between the two groups ($p=0.614$).

Table 2 shows that males comprised 67.5 and 82.5% of controls and cases respectively. There was no statistical difference between cases and controls ($p=0.124$).

Table 1. Age Distribution of Subjects

Age groups (Years)	Controls		Cases	
	Number	% age	Number	% age
20-30	3	7.5	0	0
30-40	1	2.5	2	5
40-50	3	7.5	5	12.5
50-60	15	37.5	17	42.5
60-70	10	25	11	27.5
70-80	8	20	5	12.5
Total	40	100	40	100
Mean Age	58.50	-	57.10	-
Standard Deviation	14.01	-	10.48	-

$p=0.6140$

Table 2. Sex Distribution of Subjects

Sex	Controls		Cases	
	Number	% age	Number	% age
Males	27	67.5	33	82.5
Females	13	32.5	7	17.5
Total	40	100	40	100

$P=0.124$

Table 3. Distribution of N.C.D. Morbidities and Risk Factors in Subjects

	Controls		Cases	
	Number	% age	Number	% age
Diabetic	0	0	6	15
Non- diabetic	40	100	34	85
Total	40	100	40	100
Smokers	24	60	21	52.5
Non-smokers	16	40	19	47.5
Total	40	100	40	100

Table 3 shows that there is significant statistical difference in case and control group w.r.t. diabetes mellitus (p=0.01). Smokers comprised 60 and 52.5% of

controls and cases respectively. There was no statistically significant difference between controls and cases on the basis of smoking (p=0.505).

Table 4. Comparison of Lipid Profile in Subjects

Serum Lipid (mg/dL)	Controls	Cases	Significance
Total Cholesterol (mean ± S.D.)	171.55 ± 56.88	175.03 ± 53.65	0.7794
HDL	44.95 ± 12.91	40.45 ± 21.37	0.2577
LDL	100.17 ± 47.34	92.64 ± 57.76	0.5253
Triglycerides	126.70 ± 57.20	205.68 ± 127.91	0.0006

S.D.=Standard Deviation

Table 4 shows that among the two groups, controls and cases, lipid profile is not statistically significant except for triglycerides. For triglycerides, there is a statistically

significant difference between controls and cases, triglycerides being significantly higher in the case group (p=0.006).

Table 5. Comparison of CRP (C-Reactive Protein) in Controls and Cases

CRP (mg/L)	Controls	Cases
Mean	7.49	10.91
Standard Deviation	6.21	5.53

p=0.01098

Table 5 shows that there is a statistically significant difference between cases and controls with respect to the C-reactive protein. CRP is significantly higher in the case group.

Covariate analysis was performed adjusting for age, sex, smoking, fasting blood sugar, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, diabetes and hypertension. The results were as follows:-

Table 6. Comparison of the Acute Phase Reactants between Controls and Cases after Adjusting for Confounding Factors

Dependent Variable	Mean Square Effect	Mean Square Error	F (df1, 2) 1,68	p-level
TLC	113074E2	1857587	6.087121	0.016140
ESR	49	12	4.103050	0.046733
CRP	198.64	31.28	6.349518	0.014095

Table 6 shows that even after adjusting, ESR, CRP and TLC are significantly different in controls and cases.

patients with normal angiograms (group 2), and 37 control healthy subjects found higher level of CRP in patients with unstable angina and previous myocardial infarction than in patients with stable symptoms and group 2 and group 3.

Discussion and Conclusion

In our study, we attempted to test hypothesis that a relationship exists between acute-phase reactants and unstable angina, especially C-Reactive Protein.

The present study supports this. Besides the increased level of CRP in the case group as compared to the control group (p=0.01098), statistically significant difference was found between the case and control groups in TLC (p=0.0037) and ESR (p=0.0368), TLC and ESR being more in the case group as compared to the control group.

For this study, 80 subjects were enrolled who were divided into two groups: cases who were patients with unstable angina, and controls who were age and sex-matched normal people. Forty cases and 40 controls were taken. All the factors known to cause a rise in acute phase proteins were a basis for exclusion from the study group.

The statistically significant difference between the two groups for CRP, ESR and TLC exists even after adjustments for the other variables thus implicating that the difference in the acute phase reactants in the two groups is not merely due to confounding factors.

Various studies done in the past like a study by Berk et al.² found the CRP levels significantly elevated in unstable angina as compared to the control group with no ischemic illness. Another study by Abdelmoutaleb et al.³ in 142 patients with coronary disease (group 1), 37

Another finding of interest was a statistically significant difference of triglycerides in case and control group

($p=0.0006$), triglycerides being significantly higher in the case group. But no statistically significant difference was observed for total cholesterol, HDL-cholesterol or LDL-cholesterol.

Conflict of Interest: None

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