

Study of Creactive Protein (CRP) Levels and its Prognostic Significance in Acute Myocardial Infarction (MI)



Medical Science

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Dr. Pothula Ramarao

M.D., Assistant professor, Kurnool Medical College, Kurnool

Dr. Kunigiri Somappa

M.D., Assistant professor, Kurnool Medical College, Kurnool

ABSTRACT

CRP levels partially reflect the extent of myocardial necrosis and can be used to predict in hospital and long term outcome in patients with AMI. A prospective clinical Study consisting of 50 AMI patients was undertaken to evaluate the CRP levels on admission and at 48 hours in patients with Q Wave MI and Non-Q wave MI and its association with type of infarct and outcome

Introduction:

Acute Myocardial Infarction is overwhelmingly the most important form of IHD which continues to be the leading cause of death in the industrialized and developing countries like India, despite spectacular progress in their prevention, detection and treatment over the last three decades. AMI has rapidly emerged as the major contributor towards the increasing morbidity and mortality.¹

A large number of asymptomatic individuals are at serious risk of developing MI because of their genetic predisposition, smoking behavior and sedentary lifestyle. About one third of patients with evolving myocardial infarction die before they reach the hospital to receive any effective treatment. Thus, myocardial infarction remains an important health problem and merits continued attention from basic and clinical researchers, epidemiologists and practicing physicians.

A growing body of evidence supports the concept that local and systemic inflammation play a role in the initiation and progression of atherosclerosis and its complications.^{2, 3} Inherent to the inflammatory process is the occurrence of an acute phase response. This response is induced by pro-inflammatory cytokines (Interleukin 1 and 6) which are released from the inflamed tissue by inflammatory and / or parenchymal cells. These in turn stimulate the liver to synthesize a number of acute phase proteins. CRP is a hepatically derived classical acute phase reactant, the serum level of which has long been known to increase after myocardial infarction

Case study:

Materials and methods

50 cases of myocardial infarction were included in the study. The study sample comprised of patients admitted to intensive coronary care unit of Govt General Hospital, Kurnool. Patients were of unrestricted age and gender and gave informed consent to participate in our ethical committee, KMC, Kurnool.

Prior to admission to the study, evaluation was done by detailed history, meticulous clinical examination, electrocardiogram and relevant laboratory investigations such as CKMB isoenzyme. Myocardial Infarction was diagnosed using following diagnostic criteria.

1. Chest pain

2. ECG by diagnostic criteria with the presence of any one of the following in the setting of chest pain

a. New or presumably new Q waves (at least 30ms wide and 0.20mv deep) in at least 2 leads from any of the following: i. Leads II, III, aVF, ii. Leads V1 through V 6; or iii. Leads I and aVL.

b. New or presumably new ST-T segment elevation or depression (> 0.10 mV measured 0.02 s after the J point in two contiguous leads of the previously mentioned lead combination).

c. A complete left bundle branch block in the appropriate clinical setting.

3. CK-MB levels double the normal limits.

Based on the findings of the above, patients were categorized into:

1. Q wave acute myocardial infarction (Q-wave AMI)
2. Non- Q wave acute myocardial infarction (Non- Q-AMI)

Inclusion criteria:-

Acute myocardial infarction as diagnosed by above criteria, presenting within six hours of onset of chest pain.

Exclusion criteria:-

1. Recent infections
2. Immunologic disorder
3. Known or suspected neoplastic disease
4. Recent (less than 3 months) major trauma
5. Surgery
6. Burns
7. Osteoarthritis, Rheumatoid arthritis (Ostochondritis and all other inflammatory disease)
8. Re- infarction patient
9. Valvular heart disease, infarction with pericarditis.

In patients suffering from myocardial infarction, venous blood was collected

a) On admission.

b) At 48 hours after the onset of chest pain.

From these blood samples, concentration of C- reactive protein was estimated by latex agglutination slide test [commercially available kit].

The detection limit of this test is 6 mg/l. Therefore, value of < 6mg/l was taken as normal concentration of CRP. Values of more than 6mg/l were taken as elevated

CRP.

- 6mg/l [6-11mg/l]
- 12 mg/l [12-47mg/l]
- 24 mg/l [24-47 mg/l]
- 48 mg/l [48-95 mg/l]
- 96 mg/l [96-191 mg/l]

During the hospital stay, patients were observed for complications like cardiogenic shock, arrhythmias, post infarction angina

and left ventricular failure. Primary end point was death.

Results:

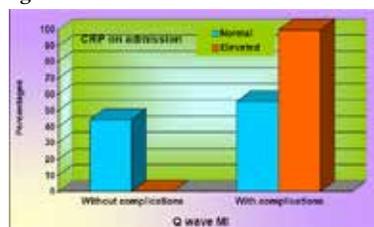
Association between CRP concentration on admission and complications during hospital stay in patients with Q-Wave MI

Table 1

CRP on Admission	Number of patients	Percentage	Without Complications		With Complications	
			Number	%	Number	%
Normal	27	71.05	12	44.44	15	55.55
Elevated	11	28.94	-	-	11	100.00
Total Q Wave MI	38	100.0	12	31.58	26	68.42

$\chi^2=7.145, P=0.008$

figure 1



Association between CRP concentration on admission and complications during hospital stay in patients with Q-Wave MI:

Among the total number of 38 patients in Q-wave MI, 27 patients (71.05%) had normal CRP and 11 (28.94%) had elevated CRP on admission.

Among the 11 patients who had elevated CRP, all the 11 (100%) developed complication during hospital stay.

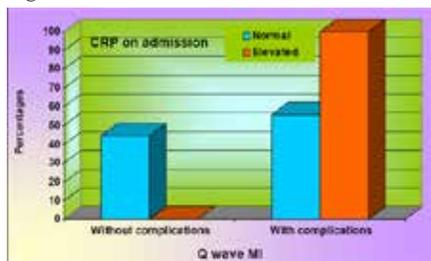
By applying chi square test the association between elevated CRP on admission and complication were statistically significant

Association between CRP concentration on admission and complications during hospital stay in patients with Non-Q wave MI

Table 2

CRP on Admission	Number of patients	Percentage	Without Complications		With Complications	
			Number	%	Number	%
Normal	11	91.67	10	90.90	1	9.09
Elevated	1	8.33	0	0	1	100
Total Non Q Wave MI	12	100.00	10	90.90	2	16.66

Figure 2



Association between CRP concentration on admission and complications during hospital stay in patients with Non-Qwave MI:

Among the total number of 12 patients, with NQWMI, 11 patients (91.67%) had normal CRP and 1 patient (8.33%) had elevated CRP on admission.

Among the 11 patients who had normal CRP, 10 patients (90.90%) did not develop any complication, and only one patient (9.09%) developed complication during the hospital stay, among the 1 patient who had elevated CRP, that 1 patient (100%) developed complication.

By applying chi square test the association between elevated CRP concentration and complication is statistically significant.

Discussion:

Acute tissue injury such as myocardial infarction rapidly leads to acceleration in the synthesis of C-reactive protein. The duration of synthesis is related to the extent of tissue injury.

In our study, 50 cases of acute myocardial infarction were included. Occurrence of acute myocardial infarction was high in males (84%) compared to females (16%), this is attributed to increased amount of stress and strain. It is also attributed to absence of protective influence of estrogen in premenopausal age which serves as cholesterol clearing factor. In our study maximum numbers of cases were in age group of 51-60 years. In Anzai.T et al and Bae H et al study, the maximum number of cases were in the age group of 61-70. In our study, the mean age (54.36±10 years) was a decade younger than the Western population. It is because the Asian Indians have underlying genetic susceptibility associated with a modest abnormality in lipid and lifestyle factors.¹

In our study CRP was elevated in 24% of patients at admission. At 48 hours after onset of chest pain, CRP was elevated in 76% patients of acute myocardial infarction. Their appeared to be some difference in this pattern, when Q-Wave MI and Non-Q Wave MI were compared. The CRP level were more often raised in both at admission and 48 hours in Q-Wave MI, whereas this CRP changes were less often seen in Non-Q Wave MI. However overall CRP was elevated in majority (80%) of the patients, at 48 hours after the onset of chest pain. Our results were similar to those observed by Lagrand et al, Nader eligharib et al. For plasma CRP to be elevated, some time lag is expected. Cytokines released as a result of tissue damage precede the synthesis and subsequent increase in CRP in plasma after the onset of myocardial damage

Out of 50 cases, 38 patients had Q-Wave MI and 12 had Non-Q-Wave MI.

In 38 patients with QWMI, 11 (28.94%) patients had elevated CRP on admission, 48 hours after the onset of chest pain 4 patients died. Hence only 34 patients were available for study. Out of them 28 patients (82.35%) had elevated CRP.

In 12 patients of NQWMI, only one patient had elevated CRP on admission but at 48 hours 7 (58.33%) had elevated CRP

In our study, anterior wall (56%) was the most common site of infarction, and inferior wall (26%) was the next common site. This was similar to Anzai et al study where anterior wall was (64%) the most common location of infarction. The next common location was inferior wall MI (34%).

CRP level and outcome:**Q Wave MI:**

27 out of 38 had normal CRP on admission and 11 had elevated CRP levels. During hospital stay, patients with elevated CRP developed complications, cardiac shock in 6 patients (54.54%) , LVF in 5 patients (27.27%) and ventricular fibrillation in 3 patients.

At 48 hours after the onset of chest pain, 28 patients had elevated CRP levels from 34 patients under study, out of them 21 patients (75%) developed complications. Among these 21 patients 11(39.3%) had cardiogenic shock, 7 (25.0%) had post MI angina, 7 (25.0%) developed LVF, 7(25%) patients had AV-Block.

Non-Q-Wave MI:

Out of 11 patients with normal CRP, only one (8.33%) developed complication and the one patient with elevated CRP had developed ventricular tachycardia during hospital stay.

At 48 hours after the onset of chest pain, 5 patients with normal CRP had no complication, whereas out of 7 patients with elevated CRP levels 2 patients developed complications. One had cardiac shock and other had ventricular tachycardia.

Overall, elevated CRP at admission was significantly associated with subsequent complications during hospital stay and during 7 days follow up. Early raise of CRP denotes extensive tissue damage and hence higher is the morbidity and mortality

Conclusion:

CRP is elevated in majority of patient with acute myocardial infarction (80%).

Association of elevated CRP levels and extensive anterior wall MI is significant statistically.

CRP is a potent predictor of prognosis in patients with acute myocardial infarction. Elevated CRP level at admission and at 48 hours after the onset of chest pain indicates poor prognosis in patients with acute myocardial infarction during hospital study.

It is useful to carry out this simple and inexpensive test routinely in all patients with acute myocardial infarction as it helps in prognosis, stratification and management of patients with myocardial infarction

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