

## Original Article

## C-Reactive Protein Estimation in the Coronary Care Unit

Reda Helal, Sameer Al-Shammari, Hasan Ali Khan, Adnan Al-Assousi, Aiad Al-Enzi, Soondal Koomar Surrin

Coronary Care Unit, Department of Medicine, Al-Jahra Hospital, Kuwait

Kuwait Medical Journal 2004, 36 (4):264-265

## ABSTRACT

**Background:** C-reactive protein (CRP) is an acute phase reactant used as a marker of both acute and chronic tissue injury. Recently, it has been shown that inflammation is an important pathogenic component of atherosclerosis in general and of acute coronary syndromes in particular. The use of ultra-sensitive immunoassay has shown that increased levels of CRP predict future cardiovascular events. We evaluated the clinical significance of plasma CRP measurement in the risk stratification and management of patients hospitalised with suspected acute coronary syndrome.

**Methods and Results:** We studied 259 patients admitted to the coronary care unit (CCU) with suspected ischemic chest pain. An ultra-sensitive immunoassay of CRP was done in all patients at the time of admission. A CRP value

of  $< 0.8$  mg/dl was considered as normal. Patients were subsequently divided into three groups according to the final diagnosis. Group 1: included 52 patients with acute myocardial infarction (MI). The CRP was raised in 22 patients (42%) and the mean CRP value was  $2.38 \pm 2.23$  mg/dl. Group 2: comprised of 50 patients with unstable angina. CRP was raised in 9 patients (18%) and mean CRP was  $1.54 \pm 3.82$  mg/dl. Group 3: A control group, composed of 157 patients with non-cardiac chest pain, CRP was raised in 25 patients (15%) and mean CRP was  $0.73 \pm 2.51$  mg/dl.

**Conclusion:** CRP measurement at the time of admission of patients with suspected acute coronary syndromes may be very helpful for risk stratification and management strategies

KEY WORDS: acute coronary syndrome, acute myocardial infarction, C-reactive protein

## INTRODUCTION

C-reactive protein (CRP), which is an acute phase reactant, has been used as a marker of tissue injury, infection and inflammation. There is increasing evidence that inflammation is an important determinant in the development of atherosclerosis. Recently, it has been shown that increased CRP values could predict future cardiovascular events<sup>[1,2]</sup>. CRP selectively binds to low density lipoprotein (LDL), particularly partly degraded, nonoxidised low-density lipoprotein found within atheromatous plaques and enhances complement activation<sup>[3,4]</sup>. Several studies have shown a relationship between CRP and coronary artery diseases (CAD)<sup>[1,2]</sup>. In our study, we evaluated the clinical significance of plasma CRP measurement in the risk stratification and management of patients hospitalised with suspected acute coronary syndrome.

## METHODS

In the period from August to November 2000 we evaluated 259 patients admitted to the coronary care unit (CCU) of Al-Jahra Hospital, Kuwait, with suspected ischemic chest pain. Patients were divided into three groups. Group 1: included 52 patients, mean age  $53 \pm 11$  yr, with acute MI;

defined as those presenting with typical cardiac chest pain, lasting for more than 20 minutes, with ECG showing ST changes of either elevation or depression with T wave changes and a rise of cardiac markers, including CKMB-mass and cardiac Troponin I. Group 2: comprised of 50 patients, mean ages  $49 \pm 10$  yr, with unstable angina, defined as having a typical chest pain with ST/T changes in the ECG and normal cardiac markers. Group 3: a control group that included 157 patients with non-cardiac chest pain. The mean age of patients was  $52 \pm 8$  yr. These were patients admitted with chest pain that proved to be of non-cardiac origin by serial ECG(s) and cardiac markers follow up, and who did not have any apparent infection, inflammation, malignancy, valvular heart diseases or left ventricular failure.

CRP was assayed by an ultra-sensitive immunoassay; in which antibody to human C-reactive protein is brought into contact with human CRP in serum samples. The CRP was measured by Beckman array system, which measures the rate of increase in light scattered from particles suspended in solution as a result of complexes formed during an antigen-antibody reaction, by rate nephelometry. The increase in light scatter resulting from the antigen-antibody reaction is converted to a peak

Address correspondence to:

Dr. Hassan A. Khan, PO Box 62267, 02153 Jahra, Al-Jahra Hospital, Kuwait. Tel/Fax 4895508, E-mail: drhajn@yahoo.com

**Table 1**

Demographic comparison between the three groups

Variables	Group 1 No. (% of total)	Group 2 No. (% of total)	Group 3 No. (% of total)
No. of patients	52 (20)	50 (19.2)	157 (60.4)
Mean age (yr) $\pm$ SD	53 $\pm$ 11	49 $\pm$ 10	52 $\pm$ 8
Male	32 (12.4)	34 (13.1)	96 (26.6)
Female	20 (7.7)	16 (6.2)	61 (23.3)
Smoker	35 (12)	30 (11.6)	100 (38.6)
Diabetic	40 (15.4)	26 (10)	96 (37)
Hypertensive	31 (11.9)	23 (8.8)	87 (33.6)
Dyslipidemic	29 (11.2)	25 (9.6)	51 (19.7)

rate signal, which is a function of the sample C-reactive protein concentration. For this array systems, a serum CRP value of  $< 0.8$  mg/dl is considered as normal.

## RESULTS

The total number of patients studied were 259 (160 M and 99 F) with a mean age of  $51.8 \pm 11$  yr (Tables 1 and 2). In Group 1, CRP was raised in 22 patients (42%), with a mean CRP value of  $2.38 \pm 2.23$  mg/dl. This rise was statistically significant when compared to the control group ( $p < 0.05$ ). Of these patients, 13 had anterior MI with a mean CRP of  $5.79 \pm 2.74$  mg/dl and nine patients had an inferior MI with a mean CRP of  $2.97 \pm 1.32$  mg/dl. In group 2, CRP was raised with a mean CRP of  $1.54 \pm 3.82$  mg/dl in nine patients (18%); four of them developed acute MI during their stay in the hospital. In Group 3, 25 patients (15%) had raised CRP with a mean value was  $0.76 \pm 2.51$  mg/dl. Patients with unstable angina had elevated CRP that was not statistically significant when compared to the control group. Patients with anterior MI had higher CRP than those with inferior MI.

## DISCUSSION

The clinical manifestations of acute coronary syndromes largely depend on the presence and severity of functional factors that transiently and acutely interfere with coronary blood flow, together with an extremely variable degree of coronary atherosclerosis<sup>[5]</sup>. Several studies have described an increase in CRP values in MI, which may reflect an important inflammatory component in this clinical situation<sup>[6]</sup>. Mach *et al* in 1997, confirmed the observation that among patients with acute ischemic heart diseases and no biological markers of myocardial necrosis, the plasma concentration of CRP at the time of admission was significantly higher in patients in whom an acute MI was ultimately diagnosed, while in patients with unstable angina the CRP value was low<sup>[7]</sup>. In our

**Table 2**

Results of CRP in the study groups

Groups	Patients with high CRP No.	%	Mean CRP $\pm$ SD	p value
1 (n = 52)	22	42	$2.38 \pm 2.23$	$<0.05$
2 (n = 50)	9	18	$1.54 \pm 3.82$	NS
3 (n = 157)	25	15	$0.73 \pm 2.51$	NS

NS: not significant

study we confirmed the association between high CRP values and acute MI. Patients who had anterior MI had CRP values higher than those with inferior or non-Q MI. This may be due to the size of the infarction. It is of interest that statins lower CRP values, suggesting that some of their protective effects may be mediated through suppression of inflammation or cytokines<sup>[8]</sup>. In agreement with previous studies, we found that CRP increased significantly in patients with proven acute MI, but insignificantly in patients with unstable angina.

## CONCLUSIONS

CRP measurement at the time of admission of patients with suspected acute coronary syndromes may be very helpful for risk stratification and management strategies

## REFERENCES

1. Pepys MB. The acute phase response and C-reactive protein. In: Weatherall D, Ledingham J, Warrel DA, Eds. Oxford textbook of medicine, 3<sup>rd</sup> Ed, Vol 2, Oxford University Press, 1995. p. 1527-1533.
2. Haverkate F, Thompson SG, Pyke SDM, Gallimore JR, Pepys MB. Production of C-reactive protein and risk of coronary events in stable and unstable angina. *Lancet* 1997; 349:462-466.
3. Alexander RW. Inflammation and coronary artery disease. *N Engl J Med* 1994; 331:468-469.
4. Bhakdi S, Toprzewski M, Klouche M, Hemmes M. Complement and atherogenesis. Binding of CRP to degraded, nonoxidised LDL enhance complement activation. *Arterioscler Thromb Vasc Biol* 1999; 19:2348-2354.
5. Fuster V, Bdomon L, Bdomna JJ, Chesebro JH. The pathogenesis of coronary artery disease and acute coronary syndrome. *N Engl J Med* 1992; 326: 242-250.
6. de Beer FC, Hind DR, Fox KM., Allan RM, Maseri A, Pepys MB. Measurement of serum C-reactive protein concentration in myocardial ischemia and myocardial infarction. *Br Heart J* 1982; 46:239-243.
7. Mach F, Loveis C, Gaspoz JM, Unger PF, Bouilli M, Urban P, *et al*. C-reactive protein as a marker for acute coronary syndromes. *Eur Hear J* 1997; 18:897-902
8. Ridker PM, Rifat N, Pfeffer MA, Sacks F, Braunwald E. Long term effect of pravastatin and plasma concentration of C-reactive protein. *Circulation* 1999; 100:230-235.